

# UNIT 5

## Cardiovascular and Circulatory Function

### Case Study

#### USING TECHNOLOGY TO PREVENT MEDICATION ERRORS

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**A**n 85-year-old male presents to the emergency department with complaints of substernal chest pressure rated 9 on a 0–10 pain scale with pain radiating to his left arm. Other signs and symptoms include nausea, dizziness, shortness of breath (SOB), diaphoresis, and a feeling of “something bad is happening to me.” He is emergently sent for cardiac catheterization; a 90% distal and 85% proximal stenosis of the right coronary artery was detected. He subsequently underwent a percutaneous coronary intervention with balloon angioplasty and placement of stents. Once recovered from anesthesia he is admitted to the cardiac step-down unit where you work. When receiving report you notice in the electronic health record (EHR) that the patient should have received the first dose of two medications in the cardiac catheterization lab following the procedure. However, these medications were not administered as prescribed and you notify the interventional cardiologist about this error.

### **QSEN Competency Focus: Informatics**

The complexities inherent in today’s health care system challenge nurses to demonstrate integration of specific interdisciplinary core competencies. These competencies are aimed at ensuring the delivery of safe, quality patient care (Institute of Medicine, 2003). The Quality and Safety Education for Nurses project (Cronenwett, Sherwood, Barnsteiner, et al., 2007; QSEN, 2020) provides a framework for the knowledge, skills, and attitudes (KSAs) required for nurses to demonstrate competency in these key areas, which include *patient-centered care, interdisciplinary teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics*.

**Informatics Definition:** Use information and technology to communicate, manage knowledge, mitigate error, and support decision-making.

SELECT PRE-LICENSURE KSAs	APPLICATION AND REFLECTION
Knowledge	
Explain why information and technology skills are essential for safe patient care	Describe how the use of the EHR and other technology can relay information to the nurse to prevent medication errors.
Skills	
Apply technology and information management tools to support safe processes of care	Describe the type of training needed to navigate the EHR efficiently.
	Identify how you can utilize technology to ensure effective communication, manage medication administration, and prevent errors.
Attitudes	
Value technologies that support clinical decision-making, error prevention, and care coordination	Reflect on how you value technologies that support clinical decision-making, error prevention, and care coordination. Do these values have the potential to create barriers to effective use of technology to prevent errors in your workplace?

Cronenwett, L., Sherwood, G., Barnsteiner, J., et al. (2007). Quality and safety education for nurses. *Nursing Outlook*, 55(3), 122–131; Institute of Medicine. (2003). *Health professions education: A bridge to quality*. Washington, DC: National Academies Press; QSEN Institute. (2020). *QSEN competencies: Definitions and pre-licensure KSAs; Informatics*. Retrieved on 8/15/2020 at: [qsen.org/competencies/pre-licensure-ksas/#informatics](https://qsen.org/competencies/pre-licensure-ksas/#informatics)

# 21 Assessment of Cardiovascular Function

## LEARNING OUTCOMES

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

1. Describe the structure and function of the cardiovascular system as well as associated cardiac risk factors.
2. Explain and demonstrate the proper techniques to perform a comprehensive cardiovascular assessment.
3. Discriminate between normal and abnormal assessment findings identified by inspection, palpation, percussion, and auscultation of the cardiovascular system.
4. Recognize and evaluate the major manifestations of cardiovascular dysfunction by applying information from the patient's health history and physical assessment findings.
5. Identify diagnostic tests and methods of hemodynamic monitoring (e.g., central venous pressure, pulmonary artery pressure, arterial pressure monitoring) of the cardiovascular system and related nursing implications.

## NURSING CONCEPTS

Perfusion

## **GLOSSARY**

**acute coronary syndrome:** a constellation of signs and symptoms due to the rupture of atherosclerotic plaque and resultant partial or complete thrombosis within a diseased coronary artery

**afterload:** the amount of resistance to ejection of blood from the ventricle

**apical impulse:** impulse normally palpated at the fifth intercostal space, left midclavicular line; caused by contraction of the left ventricle  
(synonym: point of maximal impulse)

**atrioventricular (AV) node:** secondary pacemaker of the heart, located in the right atrial wall near the tricuspid valve

**baroreceptors:** nerve fibers located in the aortic arch and carotid arteries that are responsible for control of the blood pressure

**cardiac catheterization:** an invasive procedure used to measure cardiac chamber pressures and assess patency of the coronary arteries

**cardiac conduction system:** specialized heart cells strategically located throughout the heart that are responsible for methodically generating and coordinating the transmission of electrical impulses to the myocardial cells

**cardiac output:** amount of blood pumped by each ventricle in liters per minute

**cardiac stress test:** a test used to evaluate the functioning of the heart during a period of increased oxygen demand; test may be initiated by exercise or medications

**contractility:** ability of the cardiac muscle to shorten in response to an electrical impulse

**depolarization:** electrical activation of a cell caused by the influx of sodium into the cell while potassium exits the cell

**diastole:** period of ventricular relaxation resulting in ventricular filling

**ejection fraction:** percentage of the end-diastolic blood volume ejected from the ventricle with each heartbeat

**hemodynamic monitoring:** the use of pressure monitoring devices to directly measure cardiovascular function

**hypertension:** blood pressure that is persistently greater than 130/80 mm Hg

**hypotension:** a decrease in blood pressure to less than 90/60 mm Hg that compromises systemic perfusion

**murmurs:** sounds created by abnormal, turbulent flow of blood in the heart

**myocardial ischemia:** condition in which heart muscle cells receive less oxygen than needed

**myocardium:** muscle layer of the heart responsible for the pumping action of the heart

**normal heart sounds:** sounds produced when the valves close; normal heart sounds are S<sub>1</sub> (atrioventricular valves) and S<sub>2</sub> (semilunar valves)

**opening snaps:** abnormal diastolic sounds generated during opening of rigid atrioventricular valve leaflets

**orthostatic hypotension:** a significant drop in blood pressure (20 mm Hg systolic or more or 10 mm Hg diastolic or more) after an upright posture is assumed

**preload:** degree of stretch of the cardiac muscle fibers at the end of diastole

**pulmonary vascular resistance:** resistance to blood flow out of the right ventricle created by the pulmonary circulatory system

**pulse deficit:** the difference between the apical and radial pulse rates

**radioisotopes:** unstable atoms that give off small amounts of energy in the form of gamma rays as they decay; used in cardiac nuclear medicine studies

**repolarization:** return of the cell to resting state, caused by reentry of potassium into the cell while sodium exits the cell

**S<sub>1</sub>:** the first heart sound produced by closure of the atrioventricular (mitral and tricuspid) valves

**S<sub>2</sub>:** the second heart sound produced by closure of the semilunar (aortic and pulmonic) valves

**S<sub>3</sub>:** an abnormal heart sound detected early in diastole as resistance is met to blood entering either ventricle; most often due to volume overload associated with heart failure

**S<sub>4</sub>:** an abnormal heart sound detected late in diastole as resistance is met to blood entering either ventricle during atrial contraction; most often caused by hypertrophy of the ventricle

**sinoatrial (SA) node:** primary pacemaker of the heart, located in the right atrium

**stroke volume:** amount of blood ejected from one of the ventricles per heartbeat

**summation gallop:** abnormal sounds created by the presence of an S<sub>3</sub> and S<sub>4</sub> during periods of tachycardia

**systemic vascular resistance:** resistance to blood flow out of the left ventricle created by the systemic circulatory system

**systole:** period of ventricular contraction resulting in ejection of blood from the ventricles into the pulmonary artery and aorta

**systolic click:** abnormal systolic sound created by the opening of a calcified aortic or pulmonic valve during ventricular contraction

**telemetry:** the process of continuous electrocardiographic monitoring by the transmission of radio waves from a battery-operated transmitter worn by the patient

Nearly half, or 121.5 million, of American adults have one or more types of cardiovascular disease (CVD), including hypertension, coronary artery disease (CAD), heart failure (HF), and stroke (American Heart Association [AHA], 2019). Because of the increased prevalence of CVD, nurses practicing in any setting across the continuum of care, whether in the home, office, hospital, long-term care facility, or rehabilitation facility, must be able to assess the cardiovascular system. Key components of assessment include a health history, physical assessment, and monitoring of a variety of laboratory and diagnostic test results. This assessment provides the information necessary to identify nursing diagnoses, formulate an individualized plan of care, evaluate the response of the patient to the care provided, and revise the plan as needed.

## Anatomic and Physiologic Overview

An understanding of the structure and function of the heart in health and in disease is essential to develop cardiovascular assessment skills.

### Anatomy of the Heart

The heart is a hollow, muscular organ located in the center of the thorax, where it occupies the space between the lungs (mediastinum) and rests on the diaphragm. It weighs approximately 300 g (10.6 oz); the weight and size of the heart are influenced by age, gender, body weight, extent of physical exercise and conditioning, and heart disease. The heart pumps blood to the tissues, supplying them with oxygen and other nutrients.

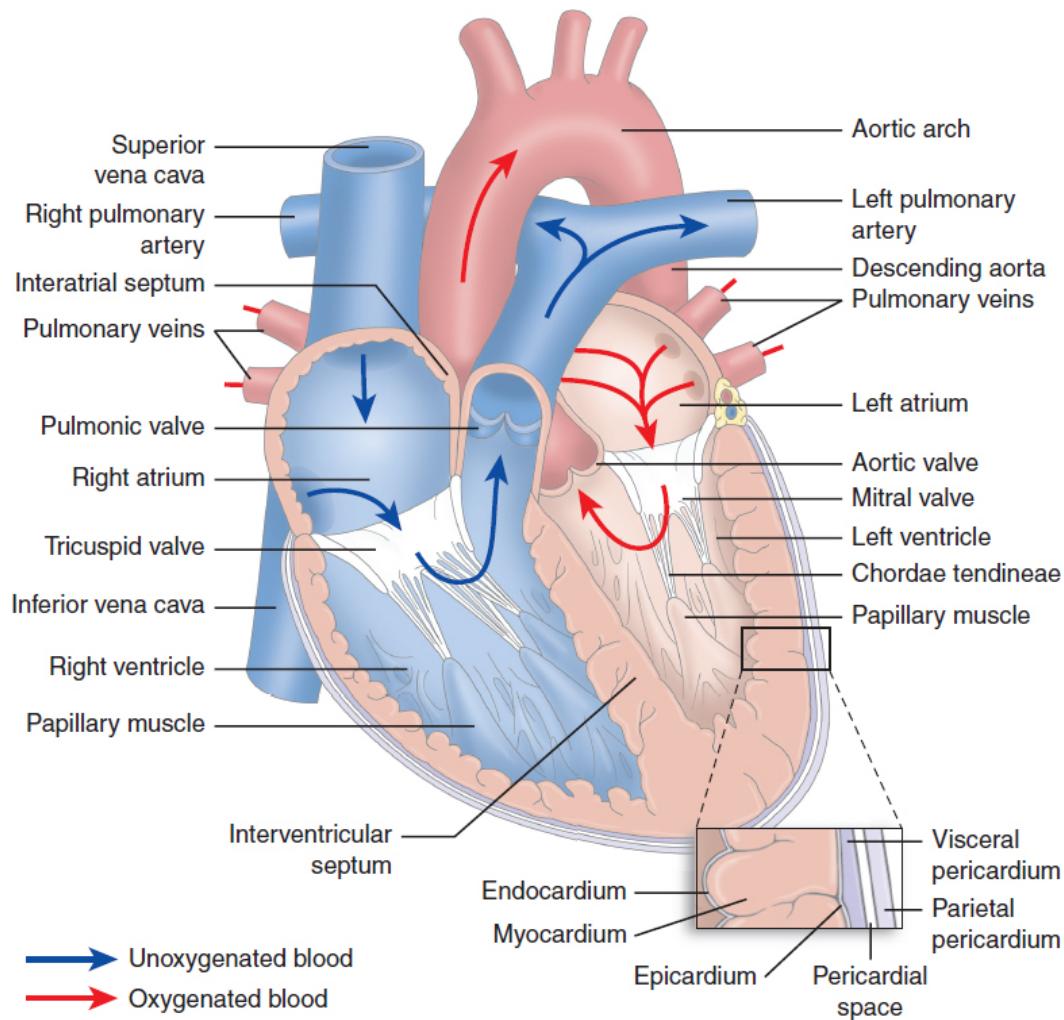
The heart is composed of three layers (Fig. 21-1). The inner layer, or endocardium, consists of endothelial tissue and lines the inside of the heart and valves. The middle layer, or **myocardium**, is made up of muscle fibers and is responsible for the pumping action. The exterior layer of the heart is called the **epicardium**.

The heart is encased in a thin, fibrous sac called the *pericardium*, which is composed of two layers. Adhering to the epicardium is the visceral pericardium. Enveloping the visceral pericardium is the parietal pericardium, a tough fibrous tissue that attaches to the great vessels, diaphragm, sternum, and vertebral column and supports the heart in the mediastinum. The space between these two layers (pericardial space) is normally filled with about 20 mL of fluid, which lubricates the surface of the heart and reduces friction during systole.

## Heart Chambers

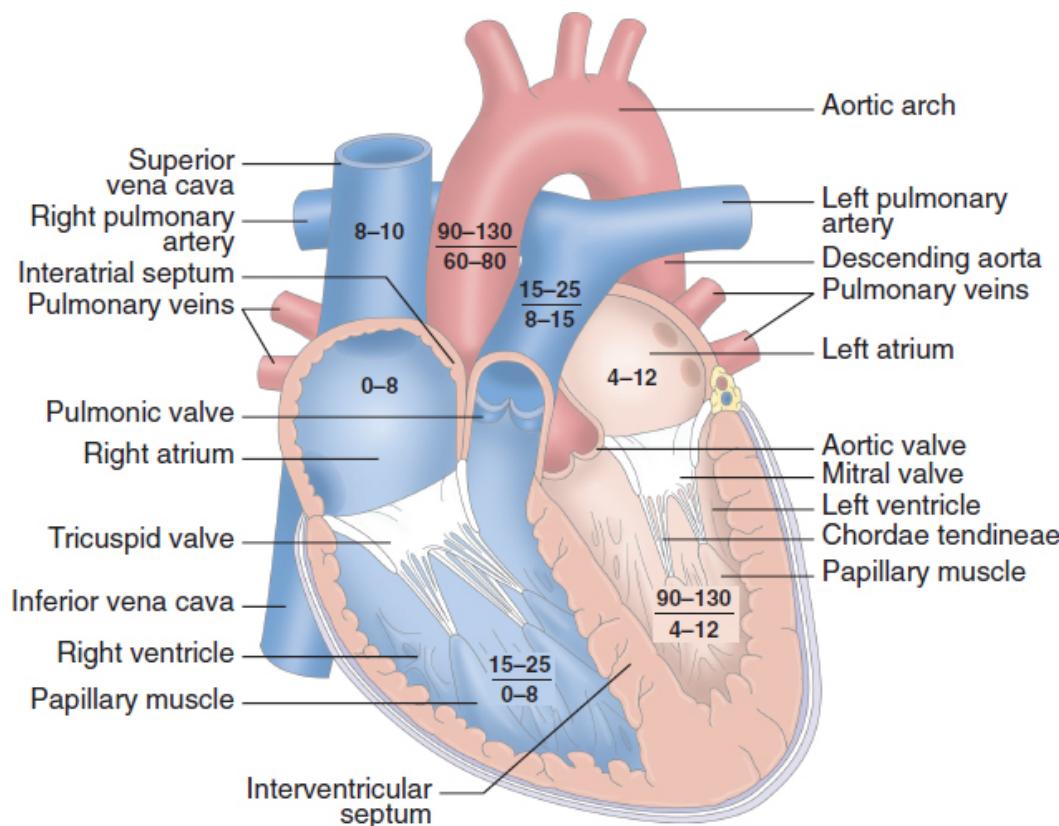
The pumping action of the heart is accomplished by the rhythmic relaxation and contraction of the muscular walls of its two top chambers (atria) and two bottom chambers (ventricles). During the relaxation phase, called **diastole**, all four chambers relax simultaneously, which allows the ventricles to fill in preparation for contraction. Diastole is commonly referred to as the period of ventricular filling. **Systole** refers to the events in the heart during contraction of the atria and the ventricles. Unlike diastole, atrial and ventricular systoles are not simultaneous events. Atrial systole occurs first, just at the end of diastole, followed by ventricular systole. This synchronization allows the ventricles to fill completely prior to ejection of blood from these chambers.

The right side of the heart, made up of the right atrium and right ventricle, distributes venous blood (deoxygenated blood) to the lungs via the pulmonary artery (pulmonary circulation) for oxygenation. The pulmonary artery is the only artery in the body that carries deoxygenated blood. The right atrium receives venous blood returning to the heart from the superior vena cava (head, neck, and upper extremities), inferior vena cava (trunk and lower extremities), and coronary sinus (coronary circulation). The left side of the heart, composed of the left atrium and left ventricle, distributes oxygenated blood to the remainder of the body via the aorta (systemic circulation). The left atrium receives oxygenated blood from the pulmonary circulation via four pulmonary veins. The flow of blood through the four heart chambers is shown in [Figure 21-1](#).



**Figure 21-1 • Structure of the heart. Arrows show course of blood flow through the heart chambers.**

The varying thicknesses of the atrial and ventricular walls are due to the workload required by each chamber. The myocardial layer of both atria is much thinner than that of the ventricles because there is little resistance as blood flows out of the atria and into the ventricles during diastole. In contrast, the ventricular walls are much thicker than the atrial walls. During ventricular systole, the right and left ventricles must overcome resistance to blood flow from the pulmonary and systemic circulatory systems, respectively. The left ventricle is two to three times more muscular than the right ventricle. It must overcome high aortic and arterial pressures, whereas the right ventricle contracts against a low-pressure system within the pulmonary arteries and capillaries (Pappano & Weir, 2019). [Figure 21-2](#) identifies the pressures in each of these areas.



**Figure 21-2 •** Great vessel and chamber pressures. Pressures are identified in millimeters of mercury (mm Hg) as mean pressure or systolic over diastolic pressure.

The heart lies in a rotated position within the chest cavity. The right ventricle lies anteriorly (just beneath the sternum), and the left ventricle is situated posteriorly. As a result of this close proximity to the chest wall, the pulsation created during normal ventricular contraction, called the **apical impulse** (also called the *point of maximal impulse [PMI]*), is easily detected. In the normal heart, the PMI is located at the intersection of the midclavicular line of the left chest wall and the fifth intercostal space (Bickley, 2017).

## Heart Valves

The four valves in the heart permit blood to flow in only one direction. The valves, which are composed of thin leaflets of fibrous tissue, open and close in response to the movement of blood and pressure changes within the chambers. There are two types of valves: atrioventricular (AV) and semilunar.

### Atrioventricular Valves

The AV valves separate the atria from the ventricles. The tricuspid valve, so named because it is composed of three cusps or leaflets, separates the right

atrium from the right ventricle. The mitral or bicuspid (two cusps) valve lies between the left atrium and the left ventricle (see Fig. 21-1).

During diastole, the tricuspid and mitral valves are open, allowing the blood in the atria to flow freely into the relaxed ventricles. As ventricular systole begins, the ventricles contract and blood flows upward into the cusps of the tricuspid and mitral valves, causing them to close. As the pressure against these valves increases, two additional structures, the papillary muscles and the chordae tendineae, maintain valve closure. The papillary muscles, located on the sides of the ventricular walls, are connected to the valve leaflets by the chordae tendineae, which are thin fibrous bands. During ventricular systole, contraction of the papillary muscles causes the chordae tendineae to become taut, keeping the valve leaflets approximated and closed. This action prevents backflow of blood into the atria (regurgitation) as blood is ejected out into the pulmonary artery and aorta.

### Semilunar Valves

The two semilunar valves are composed of three leaflets, which are shaped like half-moons. The valve between the right ventricle and the pulmonary artery is called the *pulmonic valve*. The valve between the left ventricle and the aorta is called the *aortic valve*. The semilunar valves are closed during diastole. At this point, the pressure in the pulmonary artery and aorta decreases, causing blood to flow back toward the semilunar valves. This action fills the cusps with blood and closes the valves. The semilunar valves are forced open during ventricular systole as blood is ejected from the right and left ventricles into the pulmonary artery and aorta, respectively.

### Coronary Arteries

The left and right coronary arteries and their branches supply arterial blood to the heart. These arteries originate from the aorta just above the aortic valve leaflets. Unlike other arteries, the coronary arteries are perfused during diastole. With a normal heart rate of 60 to 80 bpm, there is ample time during diastole for myocardial perfusion. However, as heart rate increases, diastolic time is shortened, which may not allow adequate time for myocardial perfusion. As a result, patients are at risk for **myocardial ischemia** (inadequate oxygen supply) during tachycardia (heart rate greater than 100 bpm), especially patients with CAD.

The left coronary artery has three branches. The artery from the point of origin to the first major branch is called the *left main coronary artery*. Two branches arise from the left main coronary artery: the left anterior descending artery, which courses down the anterior wall of the heart, and the circumflex artery, which circles around to the lateral left wall of the heart.

The right side of the heart is supplied by the *right coronary artery*, which travels to the inferior wall of the heart. The posterior wall of the heart receives its blood supply by an additional branch from the right coronary artery called the *posterior descending artery* (see [Chapter 23, Fig. 23-2](#)).

Superficial to the coronary arteries are the coronary veins. Venous blood from these veins returns to the heart primarily through the coronary sinus, which is located posteriorly in the right atrium.

## Myocardium

The myocardium is the middle, muscular layer of the atrial and ventricular walls. It is composed of specialized cells called *myocytes*, which form an interconnected network of muscle fibers. These fibers encircle the heart in a figure-of-eight pattern, forming a spiral from the base (top) of the heart to the apex (bottom). During contraction, this muscular configuration facilitates a twisting and compressive movement of the heart that begins in the atria and moves to the ventricles. The sequential and rhythmic pattern of contraction, followed by relaxation of the muscle fibers, maximizes the volume of blood ejected with each contraction. This cyclical pattern of myocardial contraction is controlled by the conduction system.

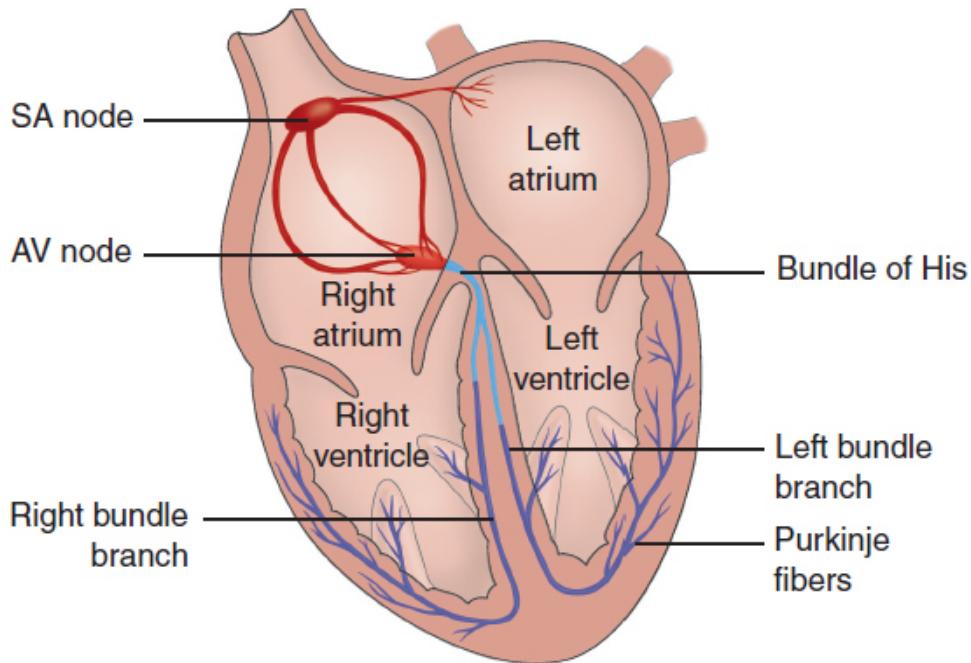
## Function of the Heart

### Cardiac Electrophysiology

The **cardiac conduction system** generates and transmits electrical impulses that stimulate contraction of the myocardium. Under normal circumstances, the conduction system first stimulates contraction of the atria and then the ventricles. The synchronization of the atrial and ventricular events allows the ventricles to fill completely before ventricular ejection, thereby maximizing cardiac output. Three physiologic characteristics of two types of specialized electrical cells, the nodal cells and the Purkinje cells, provide this synchronization:

- *Automaticity*: ability to initiate an electrical impulse
- *Excitability*: ability to respond to an electrical impulse
- *Conductivity*: ability to transmit an electrical impulse from one cell to another

Both the **sinoatrial (SA) node** (the primary pacemaker of the heart) and the **atrioventricular (AV) node** (the secondary pacemaker of the heart) are composed of nodal cells. The SA node is located at the junction of the superior vena cava and the right atrium ([Fig. 21-3](#)). The SA node in a normal resting adult heart has an inherent firing rate of 60 to 100 impulses per minute; however, the rate changes in response to the metabolic demands of the body (Weber & Kelley, 2018).



**Figure 21-3 •** Cardiac conduction system. AV, atrioventricular; SA, sinoatrial.

The electrical impulses initiated by the SA node are conducted along the myocardial cells of the atria via specialized tracts called *internodal pathways*. The impulses cause electrical stimulation and subsequent contraction of the atria. The impulses are then conducted to the AV node, which is located in the right atrial wall near the tricuspid valve (see Fig. 21-3). The AV node coordinates the incoming electrical impulses from the atria and after a slight delay (allowing the atria time to contract and complete ventricular filling) relays the impulse to the ventricles.

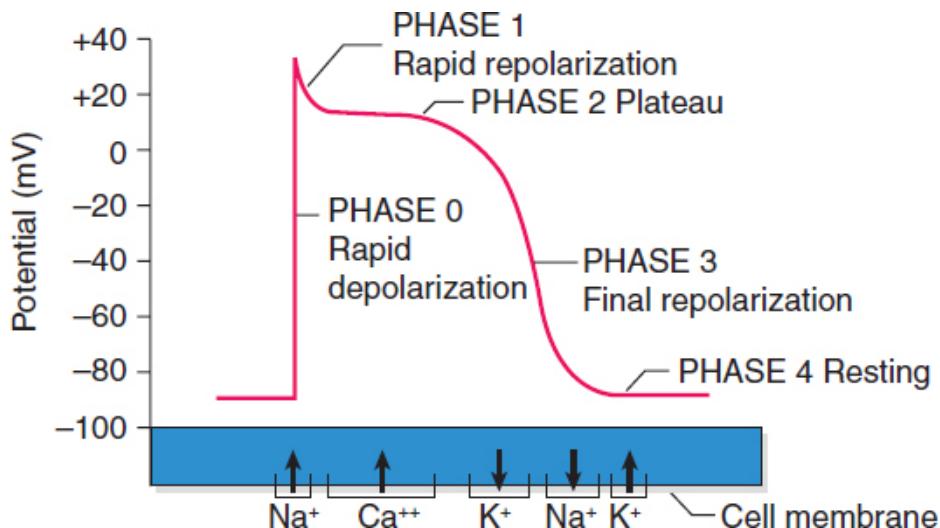
Initially, the impulse is conducted through a bundle of specialized conducting tissue, referred to as the bundle of His, which then divides into the right bundle branch (conducting impulses to the right ventricle) and the left bundle branch (conducting impulses to the left ventricle). To transmit impulses to the left ventricle—the largest chamber of the heart—the left bundle branch divides into the left anterior and left posterior bundle branches. Impulses travel through the bundle branches to reach the terminal point in the conduction system, called the *Purkinje fibers*. These fibers are composed of Purkinje cells that rapidly conduct impulses throughout the thick walls of the ventricles. This action stimulates the ventricular myocardial cells to contract (Weber & Kelley, 2018).

The heart rate is determined by the myocardial cells with the fastest inherent firing rate. Under normal circumstances, the SA node has the highest inherent rate (60 to 100 impulses per minute), the AV node has the second-highest inherent rate (40 to 60 impulses per minute), and the ventricular

pacemaker sites have the lowest inherent rate (30 to 40 impulses per minute) (Wesley, 2017). If the SA node malfunctions, the AV node generally takes over the pacemaker function of the heart at its inherently lower rate. Should both the SA and the AV nodes fail in their pacemaker function, a pacemaker site in the ventricle will fire at its inherent bradycardic rate of 30 to 40 impulses per minute.

### Cardiac Action Potential

The nodal and Purkinje cells (electrical cells) generate and transmit impulses across the heart, stimulating the cardiac myocytes (working cells) to contract. Stimulation of the myocytes occurs due to the exchange of electrically charged particles, called *ions*, across channels located in the cell membrane. The channels regulate the movement and speed of specific ions—namely, sodium, potassium, and calcium—as they enter and exit the cell. Sodium rapidly enters into the cell through sodium-fast channels, in contrast to calcium, which enters the cell through calcium-slow channels. In the resting or polarized state, sodium is the primary extracellular ion, whereas potassium is the primary intracellular ion. This difference in ion concentration means that the inside of the cell has a negative charge compared with the positive charge on the outside. The relationship changes during cellular stimulation, when sodium or calcium crosses the cell membrane into the cell and potassium ions exit into the extracellular space. This exchange of ions creates a positively charged intracellular space and a negatively charged extracellular space that characterizes the period known as **depolarization**. Once depolarization is complete, the exchange of ions reverts to its resting state; this period is known as **repolarization**. The repeated cycle of depolarization and repolarization is called the *cardiac action potential*.



**Figure 21-4 •** Cardiac action potential of a fast-response Purkinje fiber. The arrows indicate the approximate time and direction of movement of each ion influencing membrane potential.  $\text{Ca}^{++}$  movement out of the cell is not well defined but is thought to occur during phase 4.

As shown in [Figure 21-4](#), the cardiac action potential has five phases:

- *Phase 0:* Cellular depolarization is initiated as positive ions influx into the cell. During this phase, the atrial and ventricular myocytes rapidly depolarize as sodium moves into the cells through sodium-fast channels. The myocytes have a fast-response action potential. In contrast, the cells of the SA and AV node depolarize when calcium enters these cells through calcium-slow channels. These cells have a slow-response action potential.
- *Phase 1:* Early cellular repolarization begins during this phase as potassium exits the intracellular space.
- *Phase 2:* This phase is called the *plateau phase* because the rate of repolarization slows. Calcium ions enter the intracellular space.
- *Phase 3:* This phase marks the completion of repolarization and return of the cell to its resting state.
- *Phase 4:* This phase is considered the resting phase before the next depolarization.

### Refractory Periods

Myocardial cells must completely repolarize before they can depolarize again. During the repolarization process, the cells are in a refractory period. There are two phases of the refractory period: the effective (or absolute) refractory period and the relative refractory period. During the effective refractory period, the cell is completely unresponsive to any electrical stimulus; it is

incapable of initiating an early depolarization. The effective refractory period corresponds with the time in phase 0 to the middle of phase 3 of the action potential. The relative refractory period corresponds with the short time at the end of phase 3. During the relative refractory period, if an electrical stimulus is stronger than normal, the cell may depolarize prematurely. Early depolarizations of the atrium or ventricle cause premature contractions, placing the patient at risk for arrhythmias. Premature ventricular contractions in certain situations, such as the presence of myocardial ischemia, are of concern because these early ventricular depolarizations can trigger life-threatening arrhythmias, including ventricular tachycardia or ventricular fibrillation. Several circumstances make the heart more susceptible to early depolarization during the relative refractory period, thus increasing the risk for serious arrhythmias. (These arrhythmias and others are discussed in detail in [Chapter 22](#).)

## Cardiac Hemodynamics

An important determinant of blood flow in the cardiovascular system is the principle that fluid flows from a region of higher pressure to one of lower pressure (see [Fig. 21-2](#)). The pressures responsible for blood flow in the normal circulation are generated during systole and diastole.

### Cardiac Cycle

The cardiac cycle refers to the events that occur in the heart from the beginning of one heartbeat to the next. The number of cardiac cycles completed in a minute depends on the heart rate. Each cardiac cycle has three major sequential events: diastole, atrial systole, and ventricular systole. These events cause blood to flow through the heart due to changes in chamber pressures and valvular function during diastole and systole. During diastole, all four heart chambers are relaxed. As a result, the AV valves are open and the semilunar valves are closed. Pressures in all of the chambers are the lowest during diastole, which facilitates ventricular filling. Venous blood returns to the right atrium from the superior and inferior vena cava, then into the right ventricle. On the left side, oxygenated blood returns from the lungs via the four pulmonary veins into the left atrium and ventricle.

Toward the end of this diastolic period, atrial systole occurs as the atrial muscles contract in response to an electrical impulse initiated by the SA node. Atrial systole increases the pressure inside the atria, ejecting the remaining blood into the ventricles. Atrial systole augments ventricular blood volume by 15% to 25% and is sometimes referred to as the atrial kick (Wesley, 2017). At this point, ventricular systole begins in response to propagation of the electrical impulse that began in the SA node some milliseconds earlier.

Beginning with ventricular systole, the pressure inside the ventricles rapidly increases, forcing the AV valves to close. As a result, blood ceases to flow from the atria into the ventricles, and regurgitation (backflow) of blood into the atria is prevented. The rapid increase in pressure inside the right and left ventricles forces the pulmonic and aortic valves to open, and blood is ejected into the pulmonary artery and aorta, respectively. The exit of blood is at first rapid; then, as the pressure in each ventricle and its corresponding artery equalizes, the flow of blood gradually decreases. At the end of systole, pressure within the right and left ventricles rapidly decreases. As a result, pulmonary arterial and aortic pressures decrease, causing closure of the semilunar valves. These events mark the onset of diastole, and the cardiac cycle is repeated.

Chamber pressures can be measured with the use of special monitoring catheters and equipment. This technique is called **hemodynamic monitoring**. Methods of hemodynamic monitoring are covered in more detail at the end of this chapter.

### Cardiac Output

**Cardiac output** refers to the total amount of blood ejected by one of the ventricles in liters per minute. The cardiac output in a resting adult is 4 to 6 L/min but varies greatly depending on the metabolic needs of the body. Cardiac output is computed by multiplying the stroke volume by the heart rate. **Stroke volume** is the amount of blood ejected from one of the ventricles per heartbeat. The average resting stroke volume is about 60 to 130 mL (Wiegand, 2017).

### Effect of Heart Rate on Cardiac Output

The cardiac output responds to changes in the metabolic demands of the tissues associated with stress, physical exercise, and illness. To compensate for these added demands, the cardiac output is enhanced by increases in both stroke volume and heart rate. Changes in heart rate are due to inhibition or stimulation of the SA node mediated by the parasympathetic and sympathetic divisions of the autonomic nervous system. The balance between these two reflex control systems normally determines the heart rate. Branches of the parasympathetic nervous system travel to the SA node by the vagus nerve. Stimulation of the vagus nerve slows the heart rate. The sympathetic nervous system increases heart rate by innervation of the beta-1 receptor sites located within the SA node. The heart rate is increased by the sympathetic nervous system through an increased level of circulating catecholamines (secreted by the adrenal gland) and by excess thyroid hormone, which produces a catecholamine-like effect.

In addition, the heart rate is affected by central nervous system and baroreceptor activity. **Baroreceptors** are specialized nerve cells located in the

aortic arch and in both right and left internal carotid arteries (at the point of bifurcation from the common carotid arteries). The baroreceptors are sensitive to changes in blood pressure (BP). During hypertension (significant elevations in BP), these cells increase their rate of discharge, transmitting impulses to the cerebral medulla. This action initiates parasympathetic activity and inhibits sympathetic response, lowering the heart rate and the BP. The opposite is true during hypotension (low BP). Decreased baroreceptor stimulation during periods of hypotension prompts a decrease in parasympathetic activity and enhances sympathetic responses. These compensatory mechanisms attempt to elevate the BP through vasoconstriction and increased heart rate.

### Effect of Stroke Volume on Cardiac Output

Stroke volume is primarily determined by three factors: preload, afterload, and contractility.

**Preload** refers to the degree of stretch of the ventricular cardiac muscle fibers at the end of diastole. The end of diastole is the period when filling volume in the ventricles is the highest and the degree of stretch on the muscle fibers is the greatest. The volume of blood within the ventricle at the end of diastole determines preload, which directly affects stroke volume. Therefore, preload is commonly referred to as left ventricular end-diastolic pressure. As the volume of blood returning to the heart increases, muscle fiber stretch also increases (increased preload), resulting in stronger contraction and a greater stroke volume. This relationship, referred to as the Frank–Starling (or Starling) law of the heart, is maintained until the physiologic limit of the muscle is reached.

The Frank–Starling law is based on the fact that, within limits, the greater the initial length or stretch of the sarcomeres (cardiac muscle cells), the greater the degree of shortening that occurs. This result is caused by increased interaction between the thick and thin filaments within the cardiac muscle cells. Preload is decreased by a reduction in the volume of blood returning to the ventricles. Diuresis, venodilating agents (e.g., nitrates), excessive loss of blood, or dehydration (excessive loss of body fluids from vomiting, diarrhea, or diaphoresis) reduce preload. Preload is increased by increasing the return of circulating blood volume to the ventricles. Controlling the loss of blood or body fluids and replacing fluids (i.e., blood transfusions and intravenous [IV] fluid administration) are examples of ways to increase preload.

**Afterload**, or resistance to ejection of blood from the ventricle, is the second determinant of stroke volume. The resistance of the systemic BP to left ventricular ejection is called **systemic vascular resistance**. The resistance of the pulmonary BP to right ventricular ejection is called **pulmonary vascular resistance**. There is an inverse relationship between afterload and stroke volume. For example, afterload is increased by arterial vasoconstriction, which leads to decreased stroke volume. The opposite is true with arterial

vasodilation, in which case afterload is reduced because there is less resistance to ejection, and stroke volume increases.

**Contractility** refers to the force generated by the contracting myocardium. Contractility is enhanced by circulating catecholamines, sympathetic neuronal activity, and certain medications (e.g., digoxin, dopamine, or dobutamine). Increased contractility results in increased stroke volume. Contractility is depressed by hypoxemia, acidosis, and certain medications (e.g., beta-adrenergic-blocking agents such as metoprolol).

The heart can achieve an increase in stroke volume (e.g., during exercise) if preload is increased (through increased venous return), if contractility is increased (through sympathetic nervous system discharge), and if afterload is decreased (through peripheral vasodilation with decreased aortic pressure).

The percentage of the end-diastolic blood volume that is ejected with each heartbeat is called the **ejection fraction**. The ejection fraction of the normal left ventricle is 55% to 65% (Wiegand, 2017). The right ventricular ejection fraction is rarely measured. The ejection fraction is used as a measure of myocardial contractility. An ejection fraction of less than 40% indicates that the patient has decreased left ventricular function and likely requires treatment of HF (refer to [Chapter 25](#) for further discussion).



## Gerontologic Considerations

Changes in cardiac structure and function occur with age. A loss of function of the cells throughout the conduction system leads to a slower heart rate. The size of the heart increases due to hypertrophy (thickening of the heart walls), which reduces the volume of blood that the chambers can hold. Hypertrophy also changes the structure of the myocardium, reducing the strength of contraction. Both of these changes negatively affect cardiac output. The valves, due to stiffening, no longer close properly. The resulting backflow of blood creates heart murmurs, a common finding in older adults (Bickley, 2017; Pappano & Weir, 2019).

As a result of these age-related changes, the cardiovascular system takes longer to compensate from increased metabolic demands due to stress, exercise, or illness. In these situations, older adults may become symptomatic with fatigue, shortness of breath, or palpitations and present with new physical examination findings (Bickley, 2017; Zipes, Libby, Bonow, et al., 2019). The structural and functional changes with aging and associated history and physical examination findings are summarized in [Table 21-1](#).

## Gender Considerations

Structural differences between the hearts of men and women have significant implications. The heart of a woman tends to be smaller than that of a man. The coronary arteries of a woman are also narrower in diameter than a man's arteries. When atherosclerosis occurs, these differences make procedures such as cardiac catheterization and angioplasty technically more difficult.

Women typically develop CAD 10 years later than men, as women have the benefit of the cardioprotective effects of the female hormone estrogen. The three major effects of estrogen are (1) an increase in high-density lipoprotein (HDL) that transports cholesterol out of arteries; (2) a reduction in low-density lipoprotein (LDL) that deposits cholesterol in the artery; and (3) dilation of the blood vessels, which enhance blood flow to the heart. As testosterone increases and estrogen decreases, women who are postmenopausal have a greater risk of CAD, CVD, and HF (Zhao, Guallar, Ouyang, et al., 2018). Hormone therapy (HT) is not recommended for routine prevention of CAD in women who are postmenopausal, although there are possible benefits to cardiovascular health if HT is initiated soon after the initiation of menopause (Keck & Taylor, 2018).

## Assessment of the Cardiovascular System

The frequency and extent of the nursing assessment of cardiovascular function are based on several factors, including the severity of the patient's symptoms, the presence of risk factors, the practice setting, and the purpose of the assessment. Although the key components of the cardiovascular assessment remain the same, the assessment priorities vary according to patient needs. For example, an emergency department (ED) nurse performs a rapid and focused assessment of a patient in which **acute coronary syndrome** (ACS), signs and symptoms caused by ruptured atheromatous plaque in a diseased coronary artery, is suspected. Diagnosis and treatment must be started immediately, which includes an electrocardiogram (ECG) within 10 minutes of arrival to the ED (Yiadom, Baugh, McWade, et al., 2017). The physical assessment is ongoing and concentrates on evaluating the patient for ACS complications, such as a myocardial infarction (MI), arrhythmias, and HF, and determining the effectiveness of medical treatment.

**TABLE 21-1**

## Age-Related Changes of the Cardiac System

Cardiovascular Structure	Structural Changes	Functional Changes	History and Physical Findings
Atria	↑ Size of left atrium Thickening of the endocardium	↑ Atrial irritability	Irregular heart rhythm from atrial arrhythmias
Left ventricle	Endocardial fibrosis Myocardial hypertrophy (thickening) Infiltration of fat into myocardium	Left ventricle stiff and less compliant Progressive decline in cardiac output  ↑ Risk for ventricular arrhythmias Prolonged systole	Fatigue ↓ Exercise tolerance Signs and symptoms of heart failure or ventricular arrhythmias  Point of maximal impulse palpated lateral to the midclavicular line ↓ Intensity S <sub>1</sub> , S <sub>2</sub> ; split S <sub>2</sub> S <sub>4</sub> may be present
Valves	Thickening and rigidity of AV valves Calcification of aortic valve	Abnormal blood flow across valves during cardiac cycle	Murmurs may be present Thrill may be palpated if significant murmur is present
Conduction system	Connective tissue collects in SA node, AV node, and bundle branches ↓ Number of SA node cells ↓ Number of AV, bundle of His, and right and left bundle branch cells	Slower SA node rate of impulse discharge Slowed conduction across AV node and ventricular conduction system	Bradycardia Heart block ECG changes consistent with slowed conduction (↑ PR interval, widened QRS complex)
Sympathetic nervous system	↓ Response to beta-adrenergic stimulation	↓ Adaptive response to exercise: contractility and heart rate slower to respond to exercise demands  Heart rate takes more time to return to baseline	Fatigue Diminished exercise tolerance ↓ Ability to respond to stress
Aorta and arteries	Stiffening of vasculature	Left ventricular hypertrophy	Progressive increase in systolic BP;

	↓ Elasticity and widening of aorta Elongation of aorta, displacing the brachiocephalic artery upward		slight ↑ in diastolic BP Widening pulse pressure Pulsation visible above right clavicle
Baroreceptor response	↓ Sensitivity of baroreceptors in the carotid artery and aorta to transient episodes of hypertension and hypotension	Baroreceptors unable to regulate heart rate and vascular tone, causing slow response to postural changes in body position	Postural BP changes and reports of feeling dizzy, fainting when moving from lying to sitting or standing position

AV, atrioventricular; BP, blood pressure; ECG, electrocardiographic; SA, sinoatrial.

Adapted from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

## Health History

The patient's ability to recognize cardiac symptoms and to know what to do when they occur is essential for effective self-care management. All too often, a patient's new symptoms or those of progressing cardiac dysfunction go unrecognized, resulting in lifesaving treatment delays. Research findings suggest that ethnic and socioeconomic factors can lead to patient-related delays (Iacobe, Ratner, Wong, et al., 2018). Women, African Americans, and older patients are reported to delay seeking treatment when they have had MIs (Zipes et al., 2019). Socioeconomic factors such as poverty, high health care costs, lack of insurance, transportation, and proximity to a health care provider or hospital can also lead to delays in care. Patients may also lack knowledge about ACS symptoms, or experience denial, fear, or uncertainty about what to do when having chest pain. Some women have the misperception that the risk for cardiac disease is only linked to weight. A female patient who is embarrassed from the social stigma of being overweight may not see a health care provider, or could be reluctant to disclose important cardiac symptoms (Merz, Andersen, Sprague, et al., 2017). Therefore, during the health history, the nurse determines if the patient and involved family members are able to recognize symptoms of an acute cardiac problem, such as ACS or HF, and seek timely treatment of these symptoms. Responses to this level of inquiry will help the nurse individualize the plan for patient and family education.

## Common Symptoms

The signs and symptoms experienced by people with CVD are related to arrhythmias and conduction problems (see Chapter 22); CAD (see Chapter

[23](#)); structural, infectious, and inflammatory disorders of the heart (see [Chapter 24](#)); and complications of CAD such as HF and cardiogenic shock (see [Chapters 11](#) and [25](#)). These disorders have many signs and symptoms in common; therefore, the nurse must be skillful at recognizing these signs and symptoms so that patients are given timely and often lifesaving care.

The following are the most common signs and symptoms of CVD, with related medical diagnoses in parentheses:

- Chest pain or discomfort (angina pectoris, ACS, arrhythmias, valvular heart disease)
- Pain or discomfort in other areas of upper body, including one or both arms, back, neck, jaw, or stomach (ACS)
- Shortness of breath or dyspnea (ACS, cardiogenic shock, HF, valvular heart disease)
- Peripheral edema, weight gain, abdominal distention due to enlarged spleen and liver or ascites (HF)
- Palpitations (tachycardia from a variety of causes, including ACS, caffeine or other stimulants, electrolyte imbalances, stress, valvular heart disease, ventricular aneurysms)
- Unusual fatigue, sometimes referred to as vital exhaustion (an early warning symptom of ACS, HF, or valvular heart disease, characterized by feeling unusually tired or fatigued, irritable, and dejected)
- Dizziness, syncope, or changes in level of consciousness (cardiogenic shock, cerebrovascular disorders, arrhythmias, hypotension, orthostatic hypotension, vasovagal episode)

Symptoms of ACS can differ between men and women. Chest pain and discomfort related to ACS can occur in both men and women. However, women may experience more atypical or nonspecific symptoms such as chest pain at rest; pain in the jaw, arm, neck, shoulder, middle back, or epigastrium; nausea; vomiting; syncope; sweating; anxiety; and fatigue (Lichtman, Leifheit, Safdar, et al., 2018; Merz et al., 2017).

### Chest Pain

Chest pain and chest discomfort are common symptoms that may be caused by a number of cardiac and noncardiac problems. [Table 21-2](#) summarizes the characteristics and patterns of common causes of chest pain or discomfort. To differentiate among these causes of pain, the nurse asks the patient to identify the quantity (0 = no pain to 10 = worst pain), location, and quality of pain. The nurse assesses for radiation of the pain to other areas of the body and determines if associated signs and symptoms are present, such as diaphoresis or nausea. It is important to identify the events that precipitate the onset of

symptoms, the duration of the symptoms, and measures that aggravate or relieve the symptoms.

The nurse keeps the following important points in mind when assessing patients reporting chest pain or discomfort:

- The location of chest symptoms is not well correlated with the cause of the pain. For example, substernal chest pain can result from a number of causes as outlined in [Table 21-2](#).
- The severity or duration of chest pain or discomfort does not predict the seriousness of its cause. For example, when asked to rate pain using a 0 to 10 scale, patients experiencing esophageal spasm may rate their chest pain as a 10. In contrast, patients having an acute MI, which is a potentially life-threatening event, may report having moderate pain rated as a 4 to 6 on the pain scale.
- More than one clinical cardiac condition may occur simultaneously. During an MI, patients may report chest pain from myocardial ischemia, shortness of breath from HF, and palpitations from arrhythmias. Both HF and arrhythmias can be complications of an acute MI. (See [Chapter 23](#) for discussion of clinical manifestations of ACS, including MI.)

### Past Health, Family, and Social History

The health history provides an opportunity for the nurse to assess patients' understanding of their personal risk factors for peripheral vascular, cerebrovascular, and CAD and any measures that they are taking to modify these risks. Some risk factors, such as increasing age, male gender, and heredity, including race are not modifiable. However, there are a number of risk factors, such as smoking, hypertension, high cholesterol, diabetes, obesity, and physical inactivity that can be modified by lifestyle changes or medications (Arnett, Blumenthal, Albert, et al., 2019). Online tools from the AHA and the American College of Cardiology (ACC) can be used to screen a person's risk for CVD, including risk for having an MI or stroke (ACC, 2019; AHA, 2018; see Resources at the end of this chapter for a link to these tools).

In an effort to determine how patients perceive their current health status, the nurse asks the following questions:

- How is your health? Have you noticed any changes from last year? From 5 years ago?
- Do you have a cardiologist or primary provider? How often do you go for checkups?
- What health concerns do you have?
- Do you have a family history of genetic disorders that place you at risk for CVD ([Chart 21-1](#))?
- What are your risk factors for CAD (see [Chapter 23](#), [Chart 23-1](#))?

- What do you do to stay healthy and take care of your heart?

Patients who do not understand the connection between risk factors and CAD may be unwilling to make recommended lifestyle changes or manage their illness effectively. In contrast, patients who have this understanding may be more motivated to alter their lifestyle to avoid the risk of future cardiac events. The AHA published lifestyle management guidelines that identify interventions and treatment goals for each of these risk factors (Arnett et al., 2019). [Chapter 23](#) provides an overview of this information.

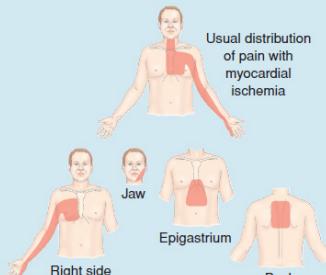
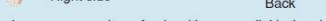
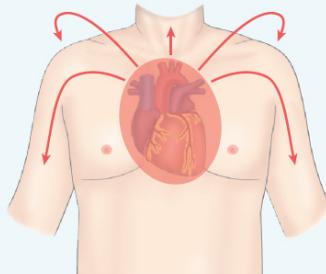
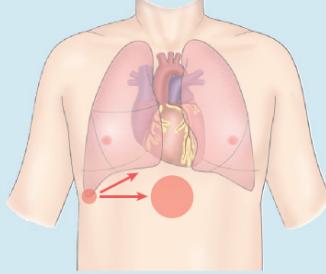
## Medications

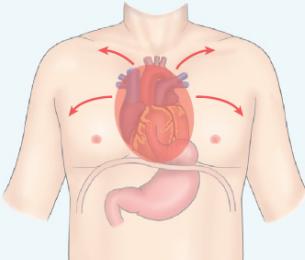
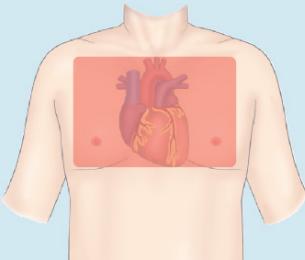
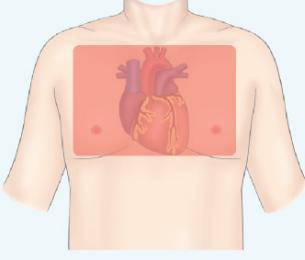
Nurses collaborate with other health care providers, including pharmacists, to obtain a complete list of the patient's medications, including dose and frequency. Vitamins, herbals, and other over-the-counter medications are included on this list. During this aspect of the health assessment, the nurse asks the following questions to ensure that the patient is safely and effectively taking the prescribed medications.

- What are the names and doses of your medications?
- What is the purpose of each of these medications?
- How and when are these medications taken? Do you ever skip a dose or forget to take them?
- Are there any special precautions associated with any of these medications?
- What symptoms or problems do you need to report to your primary provider?

**TABLE 21-2**

## Assessing Chest Pain

Location	Character	Duration	Precipitating Events and Aggravating Factors	Alleviating Factors
<b>Angina Pectoris, ACS (unstable angina, MI)</b>  <b>Less common sites of pain with myocardial ischemia</b> 	<p><b>Angina:</b> Uncomfortable pressure, squeezing, or fullness in substernal chest area Can radiate across chest to the medial aspect of one or both arms and hands, jaw, shoulders, upper back, or epigastrium</p> <p><b>Radiation:</b> Radiation to arms and hands, described as numbness, tingling, or aching</p> <p><b>ACS:</b> Same as angina pectoris Pain or discomfort ranges from mild to severe Associated with shortness of breath, diaphoresis, palpitations, unusual fatigue, and nausea or vomiting</p>	<b>Angina:</b> 5–15 min  <b>ACS:</b> >15 min	<b>Angina:</b> Physical exertion, emotional upset, eating large meal, or exposure to extremes in temperature  <b>ACS:</b> Emotional upset or unusual physical exertion occurring within 24 h of symptom onset Can occur at rest or while asleep	<b>Angina:</b> Rest, nitroglycerin, oxygen  <b>ACS:</b> Morphine, reperfusion of coronary artery with thrombolytic (fibrinolytic) agent or percutaneous coronary intervention
<b>Pericarditis</b> 	Sharp, severe substernal or epigastric pain Can radiate to neck, arms, and back Associated symptoms include fever, malaise, dyspnea, cough, nausea, dizziness, and palpitations	Intermittent	Sudden onset Pain increases with inspiration, swallowing, coughing, and rotation of trunk	Sitting upright, analgesia, anti-inflammatory medications
<b>Pulmonary Disorders (pneumonia, pulmonary embolism)</b> 	Sharp, severe substernal or epigastric pain arising from inferior portion of pleura (referred to as pleuritic pain) Patient may be able to localize the pain	≥30 min	Follows an infectious or noninfectious process (MI, cardiac surgery, cancer, immune disorders, uremia)  Pleuritic pain increases with inspiration, coughing, movement, and supine positioning  Occurs in conjunction with community- or hospital-acquired lung infections (pneumonia) or venous thromboembolism (pulmonary embolism)	Treatment of underlying cause

Esophageal Disorders (hiatal hernia, reflux esophagitis or spasm)	Substernal pain described as sharp, burning, or heavy Often mimics angina Can radiate to neck, arm, or shoulders	5–60 min	Recumbency, cold liquids, exercise	Food or antacid Nitroglycerin
				
Anxiety and Panic Disorders	Pain described as stabbing to dull ache Associated with diaphoresis, palpitations, shortness of breath, tingling of hands or mouth, feeling of unreality, or fear of losing control	>30 min	Can occur at any time including during sleep Can be associated with a specific trigger	Removal of stimulus, relaxation, medications to treat anxiety or underlying disorder
				
Musculoskeletal Disorders (costochondritis)	Sharp or stabbing pain localized in anterior chest Most often unilateral Can radiate across chest to epigastrium or back	Hours to days	Most often follows respiratory tract infection with significant coughing, vigorous exercise, or posttrauma Some cases are idiopathic. Exacerbated by deep inspiration, coughing, sneezing, and movement of upper torso or arms	Rest, ice, or heat Analgesic or anti-inflammatory medications
				

ACS, acute coronary syndrome; MI, myocardial infarction.

Adapted from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins; Rushton, S., & Carman, M. J. (2018). Chest pain: If it is not the heart, what is it? *Nursing Clinics of North America*, 53(3), 421–431; Zipes, D. P., Libby, P., Bonow, R. O., et al. (2019). *Braunwald's heart disease: A textbook of cardiovascular medicine* (11th ed.). Philadelphia, PA: Elsevier.

Patients recovering from ACS, including coronary stent placement or coronary artery bypass graft (CABG), are commonly prescribed dual antiplatelet therapy (DAPT). DAPT means that two antiplatelet drugs are prescribed for the patient. Aspirin, an OTC antiplatelet medication, is often prescribed for life. In addition, a second antiplatelet P2Y12 inhibitor medication (clopidogrel, prasugrel, or ticagrelor) is prescribed for 1 to 12 months, depending upon a variety of factors, including the patient's diagnosis and the type of procedure done (see [Chapter 23](#) for further discussion of ACS treatment and pharmacologic management) (Levine, Bates, Bittl, et al., 2016). During a careful medication history, the nurse reinforces the necessity for adherence to the medication regimen.

## Nutrition

Dietary modifications, exercise, weight loss, and careful monitoring are important strategies for managing three major cardiovascular risk factors: hyperlipidemia, hypertension, and diabetes. Diets that are restricted in sodium,

fat, cholesterol, or calories are commonly prescribed. The nurse obtains the following information:

- The patient's current height and weight (to determine body mass index [BMI]); waist measurement; BP; and any laboratory test results such as blood glucose, glycosylated hemoglobin (diabetes), total blood cholesterol, HDL and LDL levels, and triglyceride levels (hyperlipidemia)
- How often the patient self-monitors BP, blood glucose, and weight as appropriate to the medical diagnoses
- The patient's level of awareness regarding their target goals for each of the risk factors and any problems achieving or maintaining these goals
- What the patient normally eats and drinks in a typical day and any food preferences (including cultural or ethnic preferences)
- Eating habits (canned or commercially prepared foods vs. fresh foods, restaurant meals vs. home cooking, assessing for high-sodium foods, dietary intake of fats)
- Who shops for groceries and prepares meals

Chart 21-1



## GENETICS IN NURSING PRACTICE

## **Cardiovascular Disorders**

Several cardiovascular disorders are associated with genetic abnormalities. Some examples are:

- Arrhythmogenic right ventricular dysplasia (ARVD)
- Brugada syndrome
- Familial hypercholesterolemia
- Hypertrophic cardiomyopathy
- Long QT syndrome
  - Jervell and Lange-Nielsen syndrome (autosomal recessive form)
  - Romano–Ward syndrome (autosomal dominant form)
  - Genetic connective tissue disorders that impact the cardiovascular system:
- Ehlers–Danlos syndrome
- Loeys–Dietz syndrome
- Marfan syndrome

Genetic blood disorders that can impair the function of the cardiovascular system:

- Factor V Leiden
- Hemochromatosis
- Sickle cell disease

## **Nursing Assessments**

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

### **Family History Assessment Specific to Cardiovascular Disorders**

- Assess all patients with cardiovascular symptoms for coronary artery disease, regardless of age.
- Inquire about a family history of sudden death or unexplained death.
- Ask about other family members with biochemical or neuromuscular conditions (e.g., hemochromatosis or muscular dystrophy).

### **Patient Assessment Specific to Cardiovascular Disorders**

- Assess for signs and symptoms of hyperlipidemias (xanthomas, corneal arcus, or abdominal pain of unexplained

origin).

- Obtain an electrocardiogram and an echocardiogram.
- Assess for muscular weakness.
- Assess for episodes of shortness of breath, dizziness, or palpitations.
- Review laboratory data for abnormal values.
- Gather dietary history.
- Assess for secondary risk factors (e.g., diet, smoking, overweight, high stress, alcohol use).

## Elimination

Typical bowel and bladder habits need to be identified. Nocturia (awakening at night to urinate) is common in patients with HF. Fluid collected in gravity-dependent tissues (extremities) during the day (i.e., edema) redistributes into the circulatory system once the patient is recumbent at night. The increased circulatory volume is excreted by the kidneys (increased urine production).

When straining during defecation, the patient bears down (the Valsalva maneuver), which momentarily increases pressure on the baroreceptors. This triggers a vagal response, causing the heart rate to slow and resulting in syncope in some patients. Straining during urination can produce the same response.

Because many cardiac medications can cause gastrointestinal side effects or bleeding, the nurse asks about bloating, diarrhea, constipation, stomach upset, heartburn, loss of appetite, nausea, and vomiting. Screening for bloody urine or stools should be done for patients taking antiplatelet medications (aspirin, clopidogrel, prasugrel, ticagrelor), platelet aggregation inhibitors (abciximab, eptifibatide, tirofiban), or anticoagulants (low-molecular-weight heparins such as dalteparin or enoxaparin; heparin; or oral anticoagulants such as warfarin, rivaroxaban, or apixaban).

## Activity and Exercise

Changes in the patient's activity tolerance are often gradual and may go unnoticed. The nurse determines if there are recent changes by comparing the patient's current activity level with that performed in the past 6 to 12 months. New symptoms or a change in the usual symptoms during activity is a significant finding. Activity-induced angina or shortness of breath may indicate CAD. These CAD-related symptoms occur when myocardial ischemia is present, due to an inadequate arterial blood supply to the myocardium, in the setting of increased demand (e.g., exercise, stress, or anemia). Patients experiencing these kinds of symptoms need to seek medical attention. Fatigue, associated with a low left ventricular ejection fraction (less than 40%) and

certain medications (e.g., beta-adrenergic-blocking agents), can result in activity intolerance. Patients with fatigue may benefit from having their medications adjusted and learning energy conservation techniques.

Additional areas to explore include the presence of architectural barriers in the home (stairs, multilevel home); the patient's participation in cardiac rehabilitation; and his or her current exercise pattern including intensity, duration, and frequency.

### Sleep and Rest

Clues to worsening cardiac disease, especially HF, can be revealed by sleep-related events. Patients with worsening HF often experience *orthopnea*, a term used to indicate the need to sit upright or stand to avoid feeling short of breath. Patients experiencing orthopnea will report that they need to sleep upright in a chair or add extra pillows to their bed. Sudden awakening with shortness of breath, called *paroxysmal nocturnal dyspnea*, is an additional symptom of worsening HF. This nighttime symptom is caused by the reabsorption of fluid from dependent areas of the body (arms and legs) back into the circulatory system within hours of lying in bed. This sudden fluid shift increases preload and places increased demand on the heart of patients with HF, causing sudden pulmonary congestion.

Sleep-disordered breathing (SDB) is an abnormal respiratory pattern due to intermittent episodes of upper airway obstruction causing apnea and hypopnea (shallow respirations) during sleep. These abnormal sleep events cause intermittent hypoxemia, sympathetic nervous system activation, and increased intrathoracic pressure that puts mechanical stress on the heart and large artery walls. SDB impacts the length and quality of sleep. Short sleep duration (less than 7 hours per night) and poor sleep quality (interrupted sleep) are associated with the cardiovascular risks of hypertension, atherosclerosis, CAD, and stroke (Domínguez, Fuster, Fernández-Alvira, et al., 2019). Untreated SDB is also linked to HF and arrhythmias. Obstructive sleep apnea (OSA) is a type of SDB that is treated by the use of continuous positive airway pressure (CPAP), although many patients have difficulty with CPAP adherence. Other treatment options include weight loss, positional therapy, mandibular advancement devices (MADs), oral appliances, or surgery (Won, Mohsenin, & Kryger, 2018; see [Chapter 18](#) for further discussion of OSA, including risks).

During the health history, the nurse assesses for SDB by asking patients at risk if they snore loudly, have frequent bouts of awaking from sleep, awaken with a headache, or experience hypersomnolence (severe daytime sleepiness). For patients with a diagnosis of SDB or OSA, the nurse determines if the patient has been prescribed a CPAP, MAD, or oral appliance, and the frequency of its use. Patients who are being admitted to the hospital or going for an ambulatory surgical procedure should be instructed to bring their sleep aid devices with them.

## Self-Perception and Self-Concept

Self-perception and self-concept are both related to the cognitive and emotional processes that people use to formulate their beliefs and feelings about themselves. Having a chronic cardiac illness, such as HF, or experiencing an acute cardiac event, such as an MI, can alter a person's self-perception and self-concept. Patients' beliefs and feelings about their health are key determinants of adherence to self-care recommendations and recovery after an acute cardiac event. To reduce the risk of future cardiovascular-related health problems, patients are asked to make difficult lifestyle changes, such as quitting smoking. Patients who have misperceptions about the health consequences of their illness are at risk for nonadherence to these recommended lifestyle changes. The health history is used to discover how patients perceive their health by asking questions that may include the following:

- What is your cardiac condition?
- How has this illness changed your feelings about your health?
- What do you think caused this illness?
- What consequences do you think this illness will have on your physical activity, work, social relationships, and role in your family?
- How much of an influence do you think you have on controlling this illness?

The patient's responses to these questions can guide the nurse in planning interventions to ensure that the patient is prepared to manage the illness and that adequate services are in place to support the patient's recovery and self-care needs.

## Roles and Relationships

Patients with CVD are being managed with complex medical regimens and sophisticated technology, such as implantable cardioverter defibrillators (ICDs) and left ventricular assist devices. Hospital stays for cardiac disorders have shortened. Many invasive diagnostic cardiac procedures, such as cardiac catheterization, are being performed in the ambulatory setting. Support from family members helps to lessen the patient's burden of managing self-care for cardiac illnesses. Social support is closely linked to patient depression and CVD outcomes. Patients who are depressed and who have poor social support have an increased risk of poor cardiac outcomes (Kim, Kang, Bae, et al., 2019).

To assess patients' roles in their families and their relationships, both components of social support, the nurse asks each patient: Who do you live with? Who is your primary caregiver at home? Who helps you manage your health? The nurse also assesses for any significant effects that the cardiac illness has had on the patient's role in the family. Are there adequate finances

and health insurance? The answers to these questions help the nurse determine if consultation with social services or others is necessary to tailor the plan of care to meet the patient's self-care needs.

### Sexuality and Reproduction

Sexual dysfunction affects twice as many people with CVD compared with the general population. Depression, anxiety, erectile dysfunction, and major cardiac events such as an MI are common reasons that patients report decreased sexual activity. Patients and their partners are concerned about the effects of physical exertion on the heart and if the activity may cause another heart attack, sudden death, or untoward symptoms such as angina, dyspnea, or palpitations. Couples often lack adequate information about the physical demands related to sexual activity, which is considered a low-intensity exercise. The nurse can help patients by initiating discussions about sexuality and encouraging them to discuss problems with their primary provider or cardiologist. The exercise and counseling provided in cardiac rehabilitation programs may also improve sexual activity (Boothby, Dada, Rabi, et al., 2018).

Chart 21-2



### NURSING RESEARCH PROFILE

## **Depression, Self-Efficacy, and Physical Activity among Patients with Coronary Artery Disease**

Siow, E., Leung, D. Y., Wong, E. M., et al. (2018). Do depressive symptoms moderate the effects of exercise self-efficacy on physical activity among patients with coronary heart disease? *Journal of Cardiovascular Nursing*, 33(4), e26–e34.

### **Purpose**

Physical activity and exercise can reduce cardiac complications in patients with coronary artery disease (CAD). Patients with higher levels of self-efficacy are more likely to start and maintain an exercise program. Patients with CAD are also at risk for developing depression, which can lead to isolation and low activity levels. Therefore, the purpose of this study was to examine the relationship between the symptoms of depression, exercise self-efficacy, and physical activity among patients with CAD. The nurse researchers also explored if depression could impact the relationship between exercise self-efficacy and physical activity in this population.

### **Design**

A cross-sectional, exploratory study was conducted with adult participants with CAD who were hospitalized in an emergency medicine or general medical unit. This study was performed at two hospitals in Hong Kong. The nurse researchers conducted a survey interview with 149 participants before discharge from the hospital. Participants completed a survey with questions about their socioeconomic status, current level of physical activity, self-efficacy about exercise, and symptoms of depression. Participants were then asked about their physical activity using the Godin-Shephard Leisure-Time Physical Activity questionnaire, about exercise self-efficacy using the Self-Efficacy for Exercise scale, and about depression using the Centre for Epidemiological Studies-Depression tool. Other information such as body mass index (BMI), medical diagnosis, and how many times participants were admitted to the hospital was obtained from the hospital records.

### **Findings**

Most participants in this study were older adults with a mean age of  $73 \pm 13$  years, were male, married, living with family, and had a lower socioeconomic status. Approximately 50% of this sample had a normal BMI, and over half did some level of exercise. Participants with greater exercise self-efficacy were more likely to engage in physical activity. However, this relationship was stronger among participants with symptoms of depression. Participants who were depressed reported lower levels of self-efficacy and engaging in less physical activity.

### **Nursing Implications**

The results of this study demonstrate the importance of assessing self-efficacy, physical activity, and depression among patients with CAD. It is

important to intervene and educate patients about lifestyle changes that improve exercise self-efficacy. Improving this level of confidence can help patients who are depressed start or increase their level of physical activity, which may improve cardiovascular health.

A reproductive history is necessary for women of childbearing age, particularly those with seriously compromised cardiac function. The reproductive history includes information about previous pregnancies, plans for future pregnancies, oral contraceptive use (especially in women older than 35 years who smoke), menopausal status, and the use of HT. Women who have a history of preeclampsia during pregnancy, preterm labor, or giving birth to an infant that was small for gestational age have a higher risk for developing CVD (Lane-Cordova, Khan, Grobman, et al., 2019).

### Coping and Stress Tolerance

Anxiety, depression, and stress are known to influence both the development of and recovery from CAD and HF. Depression is twice as prevalent in women compared to men and has a negative impact on quality of life and overall prognosis. Patients who are depressed are more likely to be readmitted to the hospital after having a heart attack. The risk of depression is lower if the patient has relationship and work stability, a higher educational level, a healthy lifestyle, and the absence of comorbidities such as diabetes. Although the association between depression and CAD is not completely understood, biologic factors (e.g., platelet abnormalities, inflammatory responses, insulin resistance), lifestyle factors (e.g., diet, exercise, smoking), and behavioral factors (e.g., substance abuse, unemployment, social isolation) contribute to this link. Patients who are depressed are less motivated to adhere to follow-up appointments, take prescribed medications, or make recommended lifestyle changes such as smoking cessation, losing weight, exercising, or participating in cardiac rehabilitation (Jha, Qamar, Vaduganathan, et al., 2019; Vaccarino, Badimon, Bremner, et al., 2019; Yuan, Fang, Liu, et al., 2019). See [Chart 21-2](#) for a Nursing Research Profile on depression, self-efficacy, and physical activity.

Patients with CAD or HF should be assessed for depression. Patients who have depression exhibit common signs and symptoms, such as feelings of worthlessness or guilt, problems falling asleep or staying asleep, having little interest or pleasure in doing things that they usually enjoy, having difficulty concentrating, restlessness, and recent changes in appetite or weight. The Patient Health Questionnaire (PHQ-2) is a two-question self-reported patient assessment tool recommended by the AHA. The nurse asks the patient:

- *Do you have little interest or pleasure in doing things over the last 2 weeks?*
- *Are you feeling down, depressed or hopeless over the last 2 weeks?*

The nurse scores the patient's responses to each question by assigning 0 for "not at all," 1 for "several days," 2 for "more than half the days," or 3 for "nearly every day." The PHQ-2 score ranges from 0 to 6. Patients with a positive score greater than or equal to 3 complete a focused screening called the PHQ-9 and are referred to their primary providers for further evaluation (Jha et al., 2019).

Stress initiates a variety of responses, including increased levels of catecholamines and cortisol, and has been strongly linked to cardiovascular events, such as an MI. Therefore, patients need to be assessed for sources of stress; the nurse asks about recent or ongoing stressors, previous coping styles and effectiveness, and the patient's perception of their current mood and coping ability. A widely used tool used to measure life stress is the Social Readjustment Rating Scale (Homes & Rahe, 1967). Examples of items on this scale include death of a spouse, divorce, and change in responsibilities at work. Each item is assigned a score of 11 to 100. Patients identify the items that happened to them in the previous year. Patients with a score less than 150 have a slight risk for future illness, whereas a score of 150 to 299 indicates a moderate risk. A score of 300 or higher indicates a high risk for future illness. Consultation with a psychiatric advanced practice nurse, psychologist, psychiatrist, or social worker is indicated for patients who are anxious or depressed or for patients who are having difficulty coping with their cardiac illness.

## Physical Assessment



Physical assessment is conducted to confirm information obtained from the health history, to establish the patient's current or baseline condition, and, in subsequent assessments, to evaluate the patient's response to treatment. Once the initial physical assessment is completed, the frequency of future assessments is determined by the purpose of the encounter and the patient's condition. For example, a focused cardiac assessment may be performed each time the patient is seen in the outpatient setting, whereas patients in the acute care setting may require a more extensive assessment at least every 8 hours. During the physical assessment, the nurse evaluates the cardiovascular system for any deviations from normal with regard to the following (examples of abnormalities are in parentheses):

- The heart as a pump (reduced pulse pressure, displaced PMI from fifth intercostal space midclavicular line, gallop sounds, murmurs)
- Atrial and ventricular filling volumes and pressures (elevated jugular venous distention, peripheral edema, ascites, crackles, postural changes in BP)

- Cardiac output (reduced pulse pressure, hypotension, tachycardia, reduced urine output, lethargy, or disorientation)
- Compensatory mechanisms (peripheral vasoconstriction, tachycardia)

## General Appearance

This part of the assessment evaluates the patient's level of consciousness (alert, lethargic, stuporous, comatose) and mental status (oriented to person, place, time; coherence). Changes in level of consciousness and mental status may be attributed to inadequate perfusion of the brain from a compromised cardiac output or thromboembolic event (stroke). Patients are observed for signs of distress, which include pain or discomfort, shortness of breath, or anxiety.

The nurse notes the size of the patient (normal, overweight, underweight, or cachectic). The patient's height and weight are measured to calculate BMI, as well as the waist circumference (see [Chapter 4](#)). These measures are used to determine if obesity (BMI greater than  $30 \text{ kg/m}^2$ ) and abdominal fat (males: waist greater than 40 inches; females: waist greater than 35 inches) are placing the patient at risk for CAD.

## Assessment of the Skin and Extremities

Examination of the skin includes all body surfaces, starting with the head and finishing with the lower extremities. Skin color, temperature, and texture are assessed for acute and chronic problems with arterial or venous circulation. [Table 21-3](#) summarizes common skin and extremity assessment findings in patients with CVD. The most noteworthy changes include the following:

- Signs and symptoms of acute obstruction of arterial blood flow in the extremities, referred to as the six *Ps*, are *pain*, *palor*, *pulselessness*, *paresthesia*, *poikilothermia* (coldness), and *paralysis*. During the first few hours after invasive cardiac procedures (e.g., cardiac catheterization, percutaneous coronary intervention [PCI], or cardiac electrophysiology testing), affected extremities should be assessed frequently for these acute vascular changes.
- Major blood vessels of the arms and legs may be used for catheter insertion. During these procedures, systemic anticoagulation with heparin is necessary, and bruising or small hematomas may occur at the catheter access site. However, large hematomas are a serious complication that can compromise circulating blood volume and cardiac output. Patients who have undergone these procedures must have catheter access sites frequently observed until hemostasis is adequately achieved.
- Edema of the feet, ankles, or legs is called *peripheral edema*. Edema can be observed in the sacral area of patients on bed rest. The nurse assesses the patient for edema by using the thumb to place firm

pressure over the dorsum of each foot, behind each medial malleolus, over the shins or sacral area for 5 seconds. *Pitting edema* is the term used to describe an indentation in the skin created by this pressure (see [Chapter 25](#), Fig. 25-2). The degree of pitting edema relies on the clinician's judgment of depth of edema and time the indentation remains after release of pressure. Pitting edema is graded as absent (0) or as present on a scale from trace ( $1+ \leq 0.25$  inch) to severe ( $4+ \geq 1$  inch) (Urden, Stacy, & Lough, 2017). It is important that clinicians use a consistent scale in order to ensure reliable clinical measurements and management. Peripheral edema is a common finding in patients with HF and peripheral vascular diseases, such as deep vein thrombosis or chronic venous insufficiency.

- Prolonged capillary refill time indicates inadequate arterial perfusion to the extremities. To test capillary refill time, the nurse compresses the nail bed briefly to occlude perfusion and the nail bed blanches. Then, the nurse releases pressure and determines the time it takes to restore perfusion. Normally, reperfusion occurs within 2 seconds, as evidenced by the return of color to the nail bed. Prolonged capillary refill time indicates compromised arterial perfusion, a problem associated with cardiogenic shock and HF.
- Clubbing of the fingers and toes indicates chronic hemoglobin desaturation and is associated with congenital heart disease.
- Hair loss, brittle nails, dry or scaling skin, atrophy of the skin, skin color changes, and ulcerations are indicative of chronically reduced oxygen and nutrient supply to the skin observed in patients with arterial or venous insufficiency (see [Chapter 26](#) for a complete description of these conditions) (Weber & Kelley, 2018).

**TABLE 21-3**

Common Assessment Findings Associated with  
Cardiovascular Disease

Assessment Findings	Associated Causes and Conditions
Clubbing of the fingers or toes (thickening of the skin under the fingers or toes)	Chronic hemoglobin desaturation most often due to congenital heart disease, advanced pulmonary diseases
Cool/cold skin and diaphoresis	Low cardiac output (e.g., cardiogenic shock, acute myocardial infarction) causing sympathetic nervous system stimulation with resultant vasoconstriction
Cold, pain, pallor of the fingertips or toes	Intermittent arteriolar vasoconstriction (Raynaud disease). Skin may change in color from white, blue, and red accompanied by numbness, tingling, and burning pain
Cyanosis, central (a bluish tinge observed in the tongue and buccal mucosa)	Serious cardiac disorders (pulmonary edema, cardiogenic shock, congenital heart disease) result in venous blood passing through the pulmonary circulation without being oxygenated
Cyanosis, peripheral (a bluish tinge, most often of the nails and skin of the nose, lips, earlobes, and extremities)	Peripheral vasoconstriction, allowing more time for the hemoglobin molecules to become desaturated. It can be caused by exposure to cold environment, anxiety, or ↓ cardiac output
Ecchymosis or bruising (a purplish-blue color fading to green, yellow, or brown)	Blood leaking outside of the blood vessels Excessive bruising is a risk for patients on anticoagulants or platelet-inhibiting medications
Edema, lower extremities (collection of fluid in the interstitial spaces of the tissues)	Heart failure and vascular problems (PAD, chronic venous insufficiency, deep vein thrombosis, thrombophlebitis)
Hematoma (localized collection of clotted blood in the tissue)	Bleeding after catheter removal/tissue injury in patients on anticoagulant/antithrombotic agents
Pallor (↓ skin color in fingernails, lips, oral mucosa, and lower extremities)	Anemia or ↓ arterial perfusion. Suspect PAD if feet develop pallor after elevating legs 60 degrees from a supine position
Rubor (a reddish-blue discoloration of the legs, seen within 20 s to 2 min after placing in a dependent position)	Filling of dilated capillaries with deoxygenated blood, indicative of PAD
Ulcers, feet and ankles: Superficial, irregular ulcers at medial malleolus. Red to yellow granulation tissue	Rupture of small skin capillaries from chronic venous insufficiency
Ulcers, feet and ankles: Painful, deep, round ulcers on feet or from exposure to pressure. Pale to black wound base	Prolonged ischemia to tissues due to PAD. Can lead to gangrene
Thinning of skin around a cardiac implantable electronic device	Erosion of the device through the skin

Xanthelasma (yellowish, raised plaques observed along nasal portion of eyelids)      Elevated cholesterol levels (hypercholesterolemia)

PAD, peripheral arterial disease.

Adapted from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

## Blood Pressure

Systemic arterial BP is the pressure exerted on the walls of the arteries during ventricular systole and diastole. It is affected by factors such as cardiac output; distention of the arteries; and the volume, velocity, and viscosity of the blood. A normal adult BP is considered a systolic BP less than 120 mm Hg over a diastolic BP less than 80 mm Hg. High BP, called **hypertension**, is defined in two stages and is based upon BP taken on at least two instances. Stage 1 hypertension is a systolic BP between 130 and 139 mm Hg or a diastolic BP between 80 and 89 mm Hg. Stage 2 hypertension is a systolic BP over 140 mm Hg or a diastolic over 90 mm Hg (Whelton, Carey, Aronow, et al., 2018). **Hypotension** refers to an abnormally low systolic and diastolic BP that can result in lightheadedness or fainting. (See [Chapter 27](#) for additional definitions, measurement, and management.)

## Pulse Pressure

The difference between the systolic and the diastolic pressures is called the *pulse pressure*. A normal pulse pressure is 40 mm Hg. A narrow pulse pressure (e.g., BP of 92/74 mm Hg and pulse pressure of 18 mm Hg) occurs when there is vasoconstriction that is compensating for a low stroke volume and ejection velocity (shock, HF, hypovolemia, mitral regurgitation) or obstruction to blood flow during systole (mitral or aortic stenosis). This compensation allows for adequate organ perfusion. A wide pulse pressure (e.g., BP of 88/38 mm Hg and pulse pressure of 50 mm Hg) is associated with conditions that elevate the stroke volume (anxiety, exercise, bradycardia), or cause vasodilation (fever, septic shock). Abnormal pulse pressures require further cardiovascular assessment (Urden et al., 2017).

## Orthostatic (Postural) Blood Pressure Changes

There is a gravitational redistribution of approximately 500 mL of blood into the lower extremities immediately upon standing. This venous pooling reduces blood return to the heart, compromising preload that ultimately reduces stroke volume and cardiac output. As a consequence, the autonomic nervous system is activated. The sympathetic nervous system increases heart rate and enhances peripheral vasoconstriction, whereas parasympathetic activity of the heart via the vagus nerve is decreased. This stabilization occurs within 1 minute (Ricci, De Caterina, & Fedorowski, 2015).

Normal postural responses that occur when a person moves from a lying to a standing position include (1) a heart rate increase of 5 to 20 bpm above the resting rate; (2) an unchanged systolic pressure, or a slight decrease of up to 10 mm Hg; and (3) a slight increase of 5 mm Hg in diastolic pressure.

**Orthostatic (postural) hypotension** is a sustained decrease of at least 20 mm Hg in systolic BP or 10 mm Hg in diastolic BP within 3 minutes of moving from a lying or sitting to a standing position. It is usually accompanied by dizziness, lightheadedness, or syncope. The risk of orthostatic hypotension increases with age and is associated with fall risk (Ricci et al., 2015).

Orthostatic hypotension in patients with CVD is most often due to a significant reduction in preload, which compromises cardiac output. Reduced preload, which is reflective of intravascular volume depletion, is caused by dehydration from overdiuresis, bleeding (due to antiplatelet or anticoagulant medications or post intravascular procedures), or medications that dilate the blood vessels (e.g., nitrates and antispasmodic agents). In these situations, the usual mechanisms needed to maintain cardiac output (increased heart rate and peripheral vasoconstriction) cannot compensate for the significant loss in intravascular volume. As a result, the BP drops and heart rate increases with changes from lying or sitting to upright positions ([Chart 21-3](#)).

The following is an example of BP and heart rate measurements in a patient with orthostatic hypotension:

*Supine:* BP 120/70 mm Hg, heart rate 70 bpm

*Sitting:* BP 100/55 mm Hg, heart rate 90 bpm

*Standing:* BP 98/52 mm Hg, heart rate 94 bpm

## Arterial Pulses

The arteries are palpated to evaluate the pulse rate, rhythm, amplitude, contour, and obstruction to blood flow.

### Pulse Rate

The normal pulse rate varies from a low of 50 bpm in healthy, athletic young adults to rates well in excess of 100 bpm after exercise or during times of excitement. Anxiety can raise the pulse rate during the physical examination. If the rate is higher than expected, the nurse should reassess the pulse near the end of the physical examination, when the patient may be more relaxed.

Chart 21-3



### ASSESSMENT

### Assessing Patients for Orthostatic Hypotension

The following steps are recommended when assessing patients for orthostatic hypotension:

- Position the patient supine for 10 minutes before taking the initial blood pressure (BP) and heart rate measurements.
- Reposition the patient to a sitting position with legs in the dependent position, wait 2 minutes, then reassess both BP and heart rate measurements.
- If the patient is symptom free or has no significant decreases in systolic or diastolic BP, assist the patient into a standing position, obtain measurements immediately, and recheck in 2 minutes; continue measurements every 2 minutes for a total of 10 minutes to rule out orthostatic hypotension.
- Return the patient to a supine position if orthostatic hypotension is detected or if the patient becomes symptomatic.
- Document heart rate and BP measured in each position (e.g., supine, sitting, standing) and any signs or symptoms that accompany the postural changes.

Adapted from Momeyer, M. A., & Mion, L. C. (2018). Orthostatic hypotension: An often overlooked risk factor for falls. *Geriatric Nursing*, 39(4), 483–486; Urden, L. D., Stacy, K. M., & Lough, M. E. (2017). *Critical care nursing: Diagnosis and management* (8th ed.). St. Louis, MO: Elsevier Mosby.

### Pulse Rhythm

The rhythm of the pulse is normally regular. Minor variations in regularity of the pulse may occur with respirations. The pulse rate may increase during inhalation and slow during exhalation due to changes in blood flow to the heart during the respiratory cycle. This phenomenon, called *sinus arrhythmia*, occurs most commonly in children and young adults.

For the initial cardiac examination, or if the pulse rhythm is irregular, the heart rate should be counted by auscultating the apical pulse, located at the PMI, for a full minute while simultaneously palpating the radial pulse. Any discrepancy between contractions heard and pulses felt is noted. Disturbances of rhythm (arrhythmias) often result in a **pulse deficit**, which is a difference between the apical and radial pulse rates. Pulse deficits commonly occur with atrial fibrillation, atrial flutter, and premature ventricular contractions. These arrhythmias stimulate the ventricles to contract prematurely, before diastole is finished. As a result, these early ventricular contractions produce a smaller stroke volume, which can be heard during auscultation but do not produce a palpable pulse (see [Chapter 22](#) for a detailed discussion of these arrhythmias).

### Pulse Amplitude

The pulse amplitude, indicative of the BP in the artery, is used to assess peripheral arterial circulation. The nurse assesses pulse amplitude bilaterally and describes and records the amplitude of each artery. The simplest method characterizes the pulse as absent, diminished, normal, or bounding. Scales are also used to rate the strength of the pulse. The following is an example of a 0 to 4 scale:

- 0: Not palpable or absent
- +1: Diminished—weak, thready pulse; difficult to palpate; obliterated with pressure
- +2: Normal—cannot be obliterated
- +3: Moderately increased—easy to palpate, full pulse; cannot be obliterated
- +4: Markedly increased—strong, bounding pulse; may be abnormal

The numerical classification is subjective; therefore, when documenting the pulse amplitude, specify location of the artery and scale range (e.g., “left radial +3/+4”) (Weber & Kelley, 2018).

If the pulse is absent or difficult to palpate, the nurse can use a continuous wave Doppler. This portable ultrasound device has a transducer that is placed over the artery. The transducer emits and receives ultrasound beams. Rhythmic changes are heard as blood cells flow through patent arteries, whereas obstruction to blood flow is evidenced by no changes in sound. (Ultrasound techniques are discussed in more detail in [Chapter 26](#).)

### Pulse Contour

The contour of the pulse conveys important information. In patients with stenosis of the aortic valve, the valve opening is narrowed, reducing the amount of blood ejected into the aorta. The pulse pressure is narrow, and the pulse feels feeble. In aortic insufficiency, the aortic valve does not close completely, allowing blood to flow back from the aorta into the left ventricle. The rise of the pulse wave is abrupt and strong, and its fall is precipitous—a “collapsing” or “water hammer” pulse. The true contour of the pulse is best appreciated by palpating over the carotid artery rather than the distal radial artery, because the dramatic characteristics of the pulse wave may be distorted when the pulse is transmitted to smaller vessels.

### Palpation of Arterial Pulses

To assess peripheral circulation, the nurse locates and evaluates all arterial pulses. Arterial pulses are palpated at points where the arteries are near the skin surface and are easily compressed against bones or firm musculature. Pulses are detected over the right and left temporal, common carotid, brachial, radial, femoral, popliteal, dorsalis pedis, and posterior tibial arteries (see [Chapter 26](#), Fig. 26-2). A reliable assessment of the pulses depends on accurate identification of the location of the artery and careful palpation of the

area. Light palpation is essential; firm finger pressure can obliterate the temporal, dorsalis pedis, and posterior tibial pulses and confuse the examiner. In approximately 10% of patients, the dorsalis pedis pulses are not palpable (Sidawy & Perler, 2019). In such circumstances, both are usually absent and the posterior tibial arteries alone provide adequate blood supply to the feet. Arteries in the extremities are often palpated simultaneously to facilitate comparison of quality.



#### **Quality and Safety Nursing Alert**

*Do not simultaneously palpate both the temporal and carotid arteries, because it is possible to decrease the blood flow to the brain.*

### **Jugular Venous Pulsations**

Right-sided heart function can be estimated by observing the pulsations of the jugular veins of the neck, which reflects central venous pressure (CVP). CVP is the pressure in the right atria or the right ventricle at the end of diastole. If the internal jugular pulsations are difficult to see, pulsations of the external jugular veins may be noted. These veins are more superficial and are visible just above the clavicles, adjacent to the sternocleidomastoid muscles.

In patients with euvoolemia (normal blood volume), the jugular veins are normally visible in the supine position with the head of the bed elevated to 30 degrees (Bickley, 2017). Obvious distention of the veins with the patient's head elevated 45 to 90 degrees indicates an abnormal increase in CVP. This abnormality is observed in patients with right-sided HF, due to hypervolemia, pulmonary hypertension, and pulmonary stenosis; less commonly with obstruction of blood flow in the superior vena cava; and rarely with acute massive pulmonary embolism.

### **Heart Inspection and Palpation**

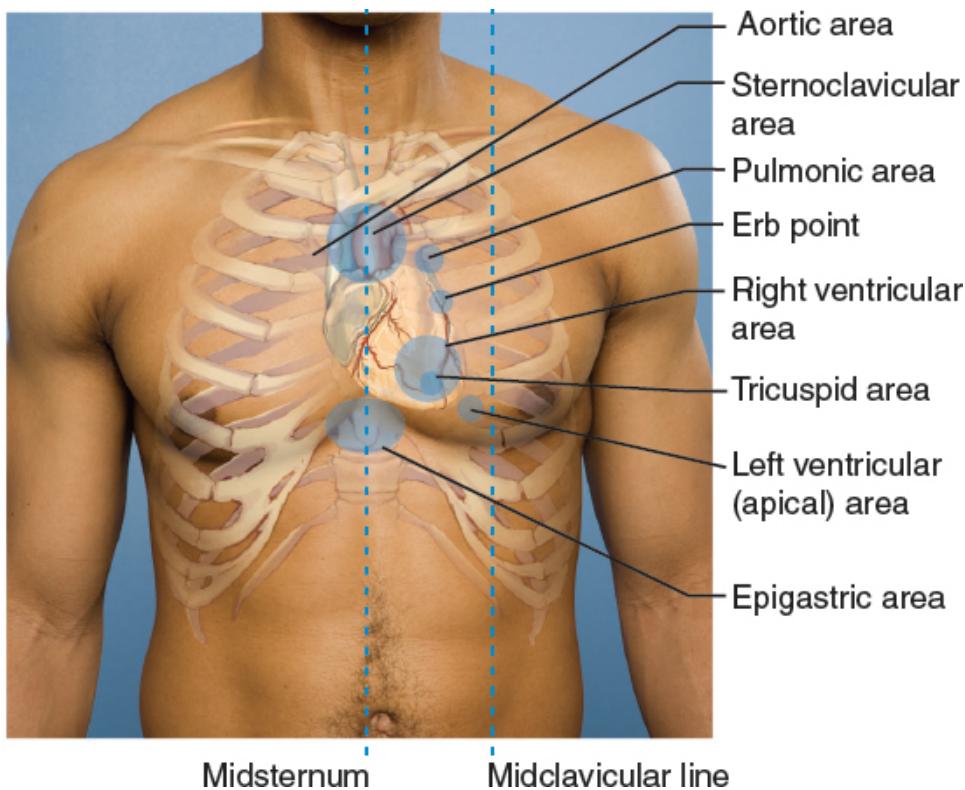


The heart is examined by inspection, palpation, and auscultation of the precordium or anterior chest wall that covers the heart and lower thorax. A systematic approach is used to examine the precordium in the following six areas. [Figure 21-5](#) identifies these important landmarks:

1. *Aortic area*—second intercostal space to the right of the sternum. To determine the correct intercostal space, the nurse first finds the angle of Louis by locating the bony ridge near the top of the sternum, at the junction of the sternum and the manubrium. From this angle, the second intercostal space is located by sliding one finger to the left or right of the

sternum. Subsequent intercostal spaces are located from this reference point by palpating down the rib cage.

2. *Pulmonic area*—second intercostal space to the left of the sternum
3. *Erb point*—third intercostal space to the left of the sternum
4. *Tricuspid area*—fourth and fifth intercostal spaces to the left of the sternum
5. *Mitral (apical) area*—left fifth intercostal space at the midclavicular line
6. *Epigastric area*—below the xiphoid process



**Figure 21-5 •** Areas of the precordium to be assessed when evaluating heart function.



**Figure 21-6 • Palpating the apical impulse.** **A.** Remain on the patient's right side, and ask the patient to remain supine. Use the finger pads to palpate the apical impulse in the mitral area (fifth intercostal space at the midclavicular line). **B.** You may ask the patient to roll to the left side to better feel the impulse using the palmar surfaces of your hand. Photos used with permission from Weber, J. R., & Kelley, J. H. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

For most of the examination, the patient lies supine, with the head of the bed or the examination table slightly elevated. A right-handed examiner stands at the right side of the patient, a left-handed examiner at the left side.

Each area of the precordium is inspected for pulsations and is then palpated. An apical impulse is a normal finding observed in young patients and adults who have thin chest walls.

The apical impulse may be felt as a light pulsation, 1 to 2 cm in diameter. It is felt at the onset of the first heart sound and lasts for only half of ventricular systole (see the next section for a discussion of heart sounds). The nurse uses the palm of the hand to locate the apical impulse initially and the finger pads to assess its size and quality. Palpation of the apical pulse may be facilitated by repositioning the patient to the left lateral position, which puts the heart in closer contact with the chest wall ([Fig. 21-6](#)).

There are several abnormalities that the nurse may find during palpation of the precordium. Normally, the apical impulse is palpable in only one intercostal space; palpability in two or more adjacent intercostal spaces indicates left ventricular enlargement. An apical impulse below the fifth intercostal space or lateral to the midclavicular line usually denotes left ventricular enlargement from left ventricular HF. If the apical impulse can be palpated in two distinctly separate areas and the pulsation movements are paradoxical (not simultaneous), a ventricular aneurysm may be suspected. A broad and forceful apical impulse is known as a left ventricular heave or lift because it appears to lift the hand from the chest wall during palpation.

A vibration or purring sensation may be felt over areas where abnormal, turbulent blood flow is present. It is best detected by using the palm of the

hand. This vibration is called a *thrill* and is associated with a loud murmur. Depending on the location of the thrill, it may be indicative of serious valvular heart disease; an atrial or ventricular septal defect (abnormal opening); or stenosis of a large artery, such as the carotid artery.

## Heart Auscultation

A stethoscope is used to auscultate each of the locations identified in [Figure 21-5](#), with the exception of the epigastric area. The purpose of cardiac auscultation is to determine heart rate and rhythm and evaluate heart sounds. The apical area is auscultated for 1 minute to determine the apical pulse rate and the regularity of the heartbeat. Normal and abnormal heart sounds detected during auscultation are described next.

### Normal Heart Sounds

**Normal heart sounds**, referred to as  $S_1$  and  $S_2$ , are produced by closure of the AV valves and the semilunar valves, respectively. The period between  $S_1$  and  $S_2$  corresponds with ventricular systole ([Fig. 21-7](#)). When the heart rate is within the normal range, systole is much shorter than the period between  $S_2$  and  $S_1$  (diastole). However, as the heart rate increases, diastole shortens.

Normally,  $S_1$  and  $S_2$  are the only sounds heard during the cardiac cycle (Bickley, 2017).

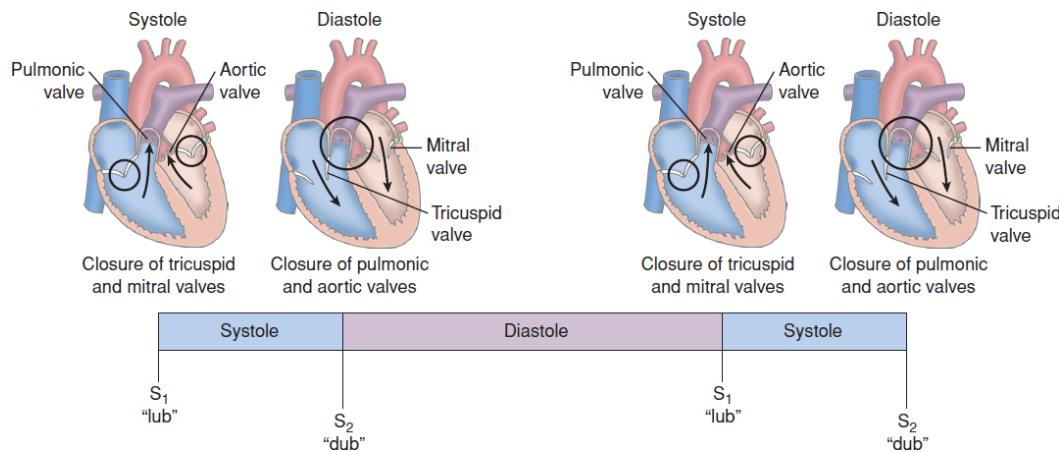
#### **$S_1$ —First Heart Sound**

Tricuspid and mitral valve closure creates the first heart sound ( $S_1$ ). The word “lub” is used to replicate its sound.  $S_1$  is usually heard the loudest at the apical area.  $S_1$  is easily identifiable and serves as the point of reference for the remainder of the cardiac cycle.

The intensity of  $S_1$  increases during tachycardias or with mitral stenosis. In these circumstances, the AV valves are wide open during ventricular contraction. The accentuated  $S_1$  occurs as the AV valves close with greater force than normal. Similarly, arrhythmias can vary the intensity of  $S_1$  from beat to beat due to lack of synchronized atrial and ventricular contraction.

#### **$S_2$ —Second Heart Sound**

Closure of the pulmonic and aortic valves produces the second heart sound ( $S_2$ ), commonly referred to as the “dub” sound. The aortic component of  $S_2$  is heard the loudest over the aortic and pulmonic areas. However, the pulmonic component of  $S_2$  is a softer sound and is heard best over the pulmonic area.



**Figure 21-7 • Normal heart sounds.** The first heart sound ( $S_1$ ) is produced by closure of the mitral and tricuspid valves ("lub"). The second heart sound ( $S_2$ ) is produced by closure of the aortic and pulmonic valves ("dub"). Arrows represent the direction of blood flow.

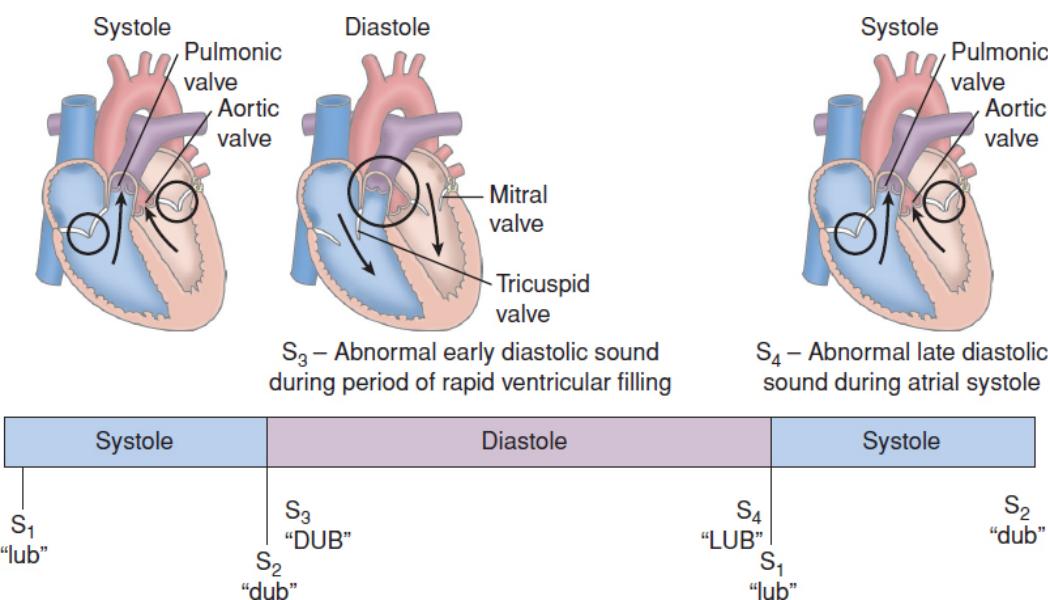
Although these valves close almost simultaneously, the pulmonic valve lags slightly behind the aortic valve. In some individuals, it is possible to distinguish between the closure of the aortic and pulmonic valves. When this situation occurs, the patient is said to have a split  $S_2$ . Normal physiologic splitting of  $S_2$  is accentuated on inspiration and disappears on expiration. During inspiration, there is a decrease in intrathoracic pressure and subsequent increase in venous return to the right atrium and ventricle. The right ventricle takes a little longer to eject this extra volume, which causes the pulmonic valve to close a little later than normal. Splitting of  $S_2$  that remains constant during inspiration and expiration is an abnormal finding. Abnormal splitting of the second heart sound can be caused by a variety of disease states (valvular heart disease, septal defects, bundle branch blocks). Splitting of  $S_2$  is best heard over the pulmonic area.

### Abnormal Heart Sounds

Abnormal sounds develop during systole or diastole when structural or functional heart problems are present. These sounds are called  $S_3$  or  $S_4$  gallops, opening snaps, systolic clicks, and murmurs.  $S_3$  and  $S_4$  gallop sounds are heard during diastole. These sounds are created by the vibration of the ventricle and surrounding structures as blood meets resistance during ventricular filling. The term *gallop* evolved from the cadence that is produced by the addition of a third or fourth heart sound, similar to the sound of a galloping horse. Gallop sounds are very low-frequency sounds and are heard with the bell of the stethoscope placed very lightly against the chest.

### S<sub>3</sub>—Third Heart Sound

An S<sub>3</sub> (“DUB”) is heard early in diastole during the period of rapid ventricular filling as blood flows from the atrium into a noncompliant ventricle. It is heard immediately after S<sub>2</sub>. “Lub-dub-DUB” is used to imitate the abnormal sound of a beating heart when an S<sub>3</sub> is present. It represents a normal finding in children and adults up to 35 or 40 years of age. In these cases, it is referred to as a physiologic S<sub>3</sub> (Fig. 21-8). In older adults, an S<sub>3</sub> is a significant finding, suggesting HF. It is best heard with the bell of the stethoscope. If the right ventricle is involved, a right-sided S<sub>3</sub> is heard over the tricuspid area with the patient in a supine position. A left-sided S<sub>3</sub> is best heard over the apical area with the patient in the left lateral position.



**Figure 21-8 • Gallop sounds.** An S<sub>3</sub> (“DUB”) is an abnormal sound heard immediately following S<sub>2</sub> (closure of semilunar valves). This sound is generated very early in diastole as blood flowing into the right or left ventricle is met with resistance. S<sub>4</sub> (“LUB”) is an abnormal sound created during atrial systole as blood flowing into the right or left ventricle is met with resistance. Arrows represent the direction of blood flow.

### S<sub>4</sub>—Fourth Heart Sound

S<sub>4</sub> (“LUB”) occurs late in diastole (see Fig. 21-8). S<sub>4</sub> heard just before S<sub>1</sub> is generated during atrial contraction as blood forcefully enters a noncompliant ventricle. This resistance to blood flow is due to ventricular hypertrophy caused by hypertension, CAD, cardiomyopathies, aortic stenosis, and numerous other conditions. “LUB lub-dub” is the mnemonic used to imitate

this gallop sound. S<sub>4</sub>, produced in the left ventricle, is auscultated using the bell of the stethoscope over the apical area with the patient in the left lateral position. A right-sided S<sub>4</sub>, although less common, is heard best over the tricuspid area with the patient in supine position. There are times when both S<sub>3</sub> and S<sub>4</sub> are present, creating a quadruple rhythm, which sounds like “LUB lub-dub DUB.” During tachycardia, all four sounds combine into a loud sound, referred to as a **summation gallop**.

### Opening Snaps and Systolic Clicks

Normally, no sound is produced when valves open. However, diseased valve leaflets create abnormal sounds as they open during diastole or systole. **Opening snaps** are abnormal diastolic sounds heard during opening of an AV valve. For example, mitral stenosis can cause an opening snap, which is an unusually high-pitched sound very early in diastole. This sound is caused by high pressure in the left atrium that abruptly displaces or “snaps” open a rigid valve leaflet. Timing helps to distinguish an opening snap from the other gallop sounds. It occurs too long after S<sub>2</sub> to be mistaken for a split S<sub>2</sub> and too early in diastole to be mistaken for an S<sub>3</sub>. The high-pitched, snapping quality of the sound is another way to differentiate an opening snap from an S<sub>3</sub>. Hearing a murmur or the sound of turbulent blood flow is expected following the opening snap. An opening snap is heard best using the diaphragm of the stethoscope placed medial to the apical area and along the lower left sternal border.

In a similar manner, stenosis of one of the semilunar valves creates a short, high-pitched sound in early systole, immediately after S<sub>1</sub>. This sound, called a **systolic click**, is the result of the opening of a rigid and calcified aortic or pulmonic valve during ventricular contraction. Mid to late systolic clicks may be heard in patients with mitral or tricuspid valve prolapse as the malfunctioning valve leaflet is displaced into the atrium during ventricular systole. Murmurs are expected to be heard following these abnormal systolic sounds. These sounds are the loudest in the areas directly over the malfunctioning valve.

### Murmurs

**Murmurs** are created by turbulent flow of blood in the heart. The causes of the turbulence may be a critically narrowed valve, a malfunctioning valve that allows regurgitant blood flow, a congenital defect of the ventricular wall, a defect between the aorta and the pulmonary artery, or an increased flow of blood through a normal structure (e.g., with fever, pregnancy, hyperthyroidism). Murmurs are characterized and consequently described by several characteristics, including their timing in the cardiac cycle, location on the chest wall, intensity, pitch, quality, and pattern of radiation ([Chart 21-4](#)).

## Friction Rub

A harsh, grating sound that can be heard in both systole and diastole is called a *friction rub*. It is caused by abrasion of the inflamed pericardial surfaces from pericarditis. Because a friction rub may be confused with a murmur, care should be taken to identify the sound and to distinguish it from murmurs that may be heard in both systole and diastole. A pericardial friction rub can be heard best using the diaphragm of the stethoscope, with the patient sitting up and leaning forward.

## Auscultation Procedure

During auscultation, the patient remains supine and the examining room is as quiet as possible. A stethoscope with both diaphragm and bell functions is necessary for accurate auscultation of the heart.

Using the diaphragm of the stethoscope, the examiner starts at the apical area and progresses upward along the left sternal border to the pulmonic and aortic areas. Alternatively, the examiner may begin the examination at the aortic and pulmonic areas and progress downward to the apex of the heart. Initially,  $S_1$  is identified and evaluated with respect to its intensity and splitting. Next,  $S_2$  is identified, and its intensity and any splitting are noted. After concentrating on  $S_1$  and  $S_2$ , the examiner listens for extra sounds in systole and then in diastole.

Sometimes it helps to ask the following questions: Do I hear snapping or clicking sounds? Do I hear any high-pitched blowing sounds? Is this sound in systole, or diastole, or both? The examiner again proceeds to move the stethoscope to all of the designated areas of the precordium, listening carefully for these sounds. Finally, the patient is turned on the left side and the stethoscope is placed on the apical area, where an  $S_3$ , an  $S_4$ , and a mitral murmur are more readily detected.

Once an abnormality is heard, the entire chest surface is reexamined to determine the exact location of the sound and its radiation. The patient may be concerned about the prolonged examination and must be supported and reassured. The auscultatory findings, particularly murmurs, are documented by identifying the following characteristics (see [Chart 21-4](#)): location on chest wall, timing, intensity, pitch, quality, and radiation.

## Interpretation of Heart Sounds

Interpreting heart sounds requires detailed knowledge of cardiac physiology and pathophysiology. However, all nurses should have adequate knowledge and skill to recognize normal heart sounds ( $S_1$ ,  $S_2$ ) and the presence of abnormal sounds. When assessment is at this very basic level of practice, abnormal findings are reported for further evaluation and treatment. More advanced skills are required of nurses caring for critically ill patients with

CVD or those nurses functioning in advanced practice roles. Nurses in these roles readily identify abnormal heart sounds, recognize the diagnostic significance of their findings, and use their assessment skills to evaluate patients' responses to medical interventions. For example, these highly skilled nurses monitor heart sounds in patients with HF to detect the resolution of an S<sub>3</sub> after treatment with a diuretic.

#### Chart 21-4

## Characteristics of Heart Murmurs

Heart murmurs are described in terms of location, timing, intensity, pitch, quality, and radiation. These characteristics provide information needed to determine the cause of the murmur and its clinical significance.

### Location

Pinpointing the location of the murmur helps to determine the underlying structures that are involved in generating the abnormal sounds. The locations described in [Figure 21-5](#) are used to identify where the loudest sounds are detected. The description should include the exact location from which the sound emanates, such as the location of the intercostal space and other important landmarks (right or left sternal border; midsternal, midclavicular, anterior axillary, or midaxillary lines). For example, a ventricular septal defect can be located at the left sternal border in the third and fourth intercostal spaces.

### Timing

A murmur is described in terms of when it occurs during the cardiac cycle (systole or diastole). Murmurs are further differentiated by identifying exactly when during systole or diastole they are heard. A skilled clinician can detect that the murmur is occurring during early, mid, or late systole or diastole. Some murmurs have sounds that occur in both systole and diastole.

### Intensity

A grading system is used to describe the intensity or loudness of a murmur.

*Grade 1:* Very faint and difficult for the inexperienced clinician to hear

*Grade 2:* Quiet but readily perceived by the experienced primary provider

*Grade 3:* Moderately loud

*Grade 4:* Loud and may be associated with a thrill

*Grade 5:* Very loud; heard when stethoscope is partially off the chest;  
associated with a thrill

*Grade 6:* Extremely loud; detected with the stethoscope off the chest;  
associated with a thrill

### Pitch

Pitch describes the sound frequency, identified as high, medium, or low pitched. High-pitched murmurs are heard best with the stethoscope's diaphragm, whereas low-pitched sounds are detected using the bell of the stethoscope placed lightly on the chest wall.

### Quality

Quality describes the sound that the murmur resembles. Murmurs can produce a rumbling, blowing, whistling, harsh, or musical sound. For

example, murmurs caused by mitral or tricuspid regurgitation have a blowing quality, whereas mitral stenosis generates a rumbling sound.

### Radiation

Radiation refers to the transmission of the murmur from the point of maximal intensity to other areas in the upper chest. The examiner determines if radiation is present by listening carefully to areas of the heart adjacent to the point where the murmur is the loudest. If radiation is present, the exact location is described. A murmur associated with aortic stenosis, for example, can radiate into the neck, down the left sternal border, and into the apical area.

Adapted from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

## Assessment of Other Systems

### Lungs

The details of respiratory assessment are described in [Chapter 17](#). Findings frequently exhibited by patients with cardiac disorders include the following:

*Hemoptysis*: Pink, frothy sputum is indicative of acute pulmonary edema.

*Cough*: A dry, hacking cough from irritation of small airways is common in patients with pulmonary congestion from HF.

*Crackles*: HF or atelectasis associated with bed rest, splinting from ischemic pain, or the effects of analgesic, sedative, or anesthetic agents often results in the development of crackles. Typically, crackles are first noted at the bases (because of gravity's effect on fluid accumulation and decreased ventilation of basilar tissue), but they may progress to all portions of the lung fields.

*Wheezes*: Compression of the small airways by interstitial pulmonary edema may cause wheezing. Beta-adrenergic-blocking agents (beta-blockers), particularly noncardioselective beta-adrenergic-blocking agents such as propranolol, may cause airway narrowing, especially in patients with underlying pulmonary disease.

### Abdomen

For the patient with CVD, several components of the abdominal examination are relevant:

*Abdominal distention*: A protuberant abdomen with bulging flanks indicates ascites. Ascites develops in patients with right ventricular or biventricular HF (both right- and left-sided HF). In the failing right heart, abnormally high chamber pressures impede the return of venous blood. As a result, the liver and spleen become engorged with excessive venous blood (hepatosplenomegaly). As pressure in the portal system rises, fluid shifts

from the vascular bed into the abdominal cavity. Ascitic fluid, found in the dependent or lowest points in the abdomen, will shift with position changes.

*Hepatojugular reflux:* This test is performed when right ventricular or biventricular HF is suspected. The patient is positioned so that the jugular venous pulse is visible in the lower part of the neck. While observing the jugular venous pulse, firm pressure is applied over the right upper quadrant of the abdomen for 30 to 60 seconds. An increase of 1 cm or more in jugular venous pressure is indicative of a positive hepatojugular reflux. This positive test aids in confirming the diagnosis of HF.

*Bladder distention:* Urine output is an important indicator of cardiac function. Reduced urine output may indicate inadequate renal perfusion or a less serious problem such as one caused by urinary retention. When urine output is decreased, the patient must be assessed for a distended bladder or difficulty voiding. The bladder may be assessed with an ultrasound scanner (see [Chapter 47](#), Fig. 47-8) or the suprapubic area palpated for an oval mass and percussed for dullness, indicative of a full bladder.



## Gerontologic Considerations

When performing a cardiovascular examination on an older patient, the nurse may note such differences as more readily palpable peripheral pulses because of decreased elasticity of the arteries and a loss of adjacent connective tissue. Palpation of the precordium in older adults is affected by the changes in the shape of the chest. For example, a cardiac impulse may not be palpable in patients with chronic obstructive pulmonary disease, because these patients usually have an increased anterior–posterior chest diameter. Kyphoscoliosis, a spinal deformity that occurs in many older adult patients, may move the cardiac apex downward so that palpation of the apical impulse is obscured.

Hypertension affects 46% of adults. The prevalence increases with age and is also impacted by race and ethnicity. The risk of a middle-age adult developing hypertension is over 90% for African Americans and Hispanic/Latino Americans. Untreated hypertension is associated with significant cardiovascular morbidity and mortality, including stroke (Whelton et al., 2018). Isolated systolic hypertension is of concern in adults over 55 year of age, and occurs because of stiffening of the vasculature and a decrease in vascular elasticity due to the aging process (Zipes et al., 2019). Another common BP problem in the older adult is orthostatic hypotension, which is a result of impaired baroreceptor function necessary to regulate BP. Other factors that heighten the risk for orthostatic hypotension include prolonged bed rest, dehydration, and many cardiovascular medications (e.g., beta-blockers,

angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, diuretics, nitrates).

An S<sub>4</sub> that is associated with hypertension is common in older adults. It is thought to be due to a decrease in compliance of the left ventricle. The S<sub>2</sub> is usually split. At least 60% of older patients have murmurs, the most common being a soft systolic ejection murmur resulting from sclerotic changes of the aortic leaflets (Bickley, 2017) (see [Table 21-1](#)).

## Diagnostic Evaluation

A wide range of diagnostic studies may be performed in patients with cardiovascular conditions. The nurse should educate the patient on the purpose, what to expect, and any possible side effects related to these examinations prior to testing. The nurse should note trends in results because they provide information about disease progression as well as the patient's response to therapy.

## Laboratory Tests

Samples of the patient's blood are sent to the laboratory for the following reasons:

- To screen for risk factors associated with CAD
- To establish baseline values before initiating other diagnostic tests, procedures, or therapeutic interventions
- To monitor response to therapeutic interventions
- To assess for abnormalities in the blood that affect prognosis

Normal values for laboratory tests may vary depending on the laboratory and the health care institution. This variation is due to the differences in equipment and methods of measurement across organizations.

## Cardiac Biomarker Analysis

The diagnosis of MI is made by evaluating the history and physical examination, the 12-lead ECG, and the results of laboratory tests that measure serum cardiac biomarkers. Myocardial cells that become necrotic from prolonged ischemia or trauma release specific enzymes (creatine kinase [CK]), CK isoenzymes (CK-MB), and proteins (myoglobin, troponin T, and troponin I). These substances leak into the interstitial spaces of the myocardium and are carried by the lymphatic system into general circulation. As a result, abnormally high levels of these substances can be detected in serum blood samples. (See [Chapter 23](#) for further discussion of cardiac biomarker analysis.)

## Blood Chemistry, Hematology, and Coagulation Studies

Table 21-4 provides information about some common serum laboratory tests and the implications for patients with CVD. Discussion of lipid, brain (B-type) natriuretic peptide (BNP), C-reactive protein (CRP), and homocysteine measurements follows.

### Lipid Profile

Cholesterol, triglycerides, and lipoproteins are measured to evaluate a person's risk of developing CAD, especially if there is a family history of premature heart disease, or to diagnose a specific lipoprotein abnormality. Cholesterol and triglycerides are transported in the blood by combining with plasma proteins to form lipoproteins called LDL and HDL. Although cholesterol levels remain relatively constant over 24 hours, the blood specimen for the lipid profile should be obtained after a 12-hour fast.

### Cholesterol Levels

Cholesterol is a lipid required for hormone synthesis and cell membrane formation. It is found in large quantities in brain and nerve tissue. Two major sources of cholesterol are diet (animal products) and the liver, where cholesterol is synthesized. Factors that contribute to variations in cholesterol levels include age, gender, diet, exercise patterns, genetics, menopause, tobacco use, and stress levels. Total cholesterol level is calculated by adding the HDL, LDL, and 20% of the triglyceride level.

High cholesterol levels increase the risk of CVD regardless of the patient's age. Lifestyle changes are recommended to lower cholesterol levels. After determining a person's 10-year risk for atherosclerotic vascular disease, medication is prescribed if necessary. Statins, a class of cholesterol-lowering medications, are often prescribed. Other nonstatins may be added to medication management to further reduce cholesterol; these include ezetimibe, bile acid sequestrants, and PCSK9 inhibitors (Grundy, Stone, Bailey, et al., 2019) (see Chapter 23 for more details).

**TABLE 21-4**

Common Serum Laboratory Tests and Implications  
for Patients with Cardiovascular Disease

Laboratory Test Reference Range	Implications
<b>Blood Chemistries</b>	
Blood urea nitrogen (BUN): 8–20 mg/dL	BUN and creatinine are end products of protein metabolism excreted by the kidneys. Elevated BUN reflects reduced renal perfusion from decreased cardiac output or intravascular fluid volume deficit as a result of diuretic therapy or dehydration.
Calcium (Ca <sup>++</sup> ): 8.8–10.4 mg/dL	Calcium is necessary for blood coagulability, neuromuscular activity, and automaticity of the nodal cells (sinus and atrioventricular nodes). <i>Hypocalcemia:</i> Decreased calcium levels slow nodal function and impair myocardial contractility. The latter effect increases the risk for heart failure. <i>Hypercalcemia:</i> Increased calcium levels can occur with the administration of thiazide diuretics because these medications reduce renal excretion of calcium. Hypercalcemia potentiates digitalis toxicity, causes increased myocardial contractility, and increases the risk for varying degrees of heart block and sudden death from ventricular fibrillation.
Creatinine <i>Male:</i> 0.6–1.2 mg/dL <i>Female:</i> 0.4–1.0 mg/dL	Both BUN and creatinine are used to assess renal function, although creatinine is a more sensitive measure. Renal impairment is detected by an increase in both BUN and creatinine. A normal creatinine level and an elevated BUN suggest an intravascular fluid volume deficit.
Magnesium (Mg <sup>++</sup> ): 1.8–2.6 mg/dL	Magnesium is necessary for the absorption of calcium, maintenance of potassium stores, and metabolism of adenosine triphosphate. It plays a major role in protein and carbohydrate synthesis and muscular contraction. <i>Hypomagnesemia:</i> Decreased magnesium levels are due to enhanced renal excretion of magnesium from the use of diuretic or digitalis therapy. Low magnesium levels predispose patients to atrial or ventricular tachycardias. <i>Hypermagnesemia:</i> Increased magnesium levels are commonly caused by the use of cathartics or antacids containing magnesium. Increased magnesium levels depress contractility and excitability of the myocardium, causing heart block and, if severe, asystole.
Potassium (K <sup>+</sup> ): 3.5–5 mEq/L	Potassium has a major role in cardiac electrophysiologic function. <i>Hypokalemia:</i> Decreased potassium levels due to administration of potassium-excreting diuretics can cause many forms of arrhythmias, including life-threatening ventricular tachycardia or ventricular fibrillation, and predispose patients taking digitalis preparations to digitalis toxicity.

	<p><b>Hyperkalemia:</b> Increased potassium levels can result from an increased intake of potassium (e.g., foods high in potassium or potassium supplements), decreased renal excretion of potassium, the use of potassium-sparing diuretics (e.g., spironolactone), or the use of angiotensin-converting enzyme inhibitors that inhibit aldosterone function. Serious consequences of hyperkalemia include heart block, asystole, and life-threatening ventricular arrhythmias.</p>
Sodium ( $\text{Na}^+$ ): 135–145 mEq/L	<p>Low or high serum sodium levels do not directly affect cardiac function.</p> <p><b>Hyponatremia:</b> Decreased sodium levels indicate fluid excess and can be caused by heart failure or administration of thiazide diuretics.</p> <p><b>Hypernatremia:</b> Increased sodium levels indicate fluid deficits and can result from decreased water intake or loss of water through excessive sweating or diarrhea.</p>
<b>Coagulation Studies</b>	<p>Injury to a vessel wall or tissue initiates the formation of a thrombus. This injury activates the coagulation cascade, the complex interactions among phospholipids, calcium, and clotting factors that convert prothrombin to thrombin. The coagulation cascade has two pathways: the intrinsic and extrinsic pathways. Coagulation studies are routinely performed before invasive procedures, such as cardiac catheterization, electrophysiology testing, and cardiac surgery.</p>
Activated partial thromboplastin time (aPTT) <i>Lower limit of normal:</i> 21–35 s	<p>aPTT measures the activity of the intrinsic pathway and is used to assess the effects of unfractionated heparin. A therapeutic range is 1.5–2.5 times baseline values. Adjustment of heparin dose is required for aPTT &lt;50 s (<math>\uparrow</math> dose) or &gt;100 s (<math>\downarrow</math> dose).</p>
Prothrombin time (PT) <i>Lower limit of normal:</i> 11–13 s	<p>PT measures the extrinsic pathway activity and is used to monitor the level of anticoagulation with warfarin.</p>
International normalized ratio (INR): 0.8–1.2	<p>The INR, reported with the PT, provides a standard method for reporting PT levels and eliminates the variation of PT results from different laboratories. The INR, rather than the PT alone, is used to monitor the effectiveness of warfarin. The therapeutic range for INR is 2–3.5, although specific ranges vary based on diagnosis.</p>
<b>Hematologic Studies</b>	
Complete blood count (CBC)	<p>The CBC identifies the total number of white and red blood cells and platelets, and measures hemoglobin and hematocrit. The CBC is carefully monitored in patients with cardiovascular disease.</p>
Hematocrit <i>Male:</i> 42–52% <i>Female:</i> 36–48%	<p>The hematocrit represents the percentage of red blood cells found in 100 mL of whole blood. The red blood cells contain hemoglobin, which transports oxygen to the cells.</p>

Hemoglobin <i>Male:</i> 14–17.4 g/dL <i>Female:</i> 12–16 g/dL	Low hemoglobin and hematocrit levels have serious consequences for patients with cardiovascular disease, such as more frequent angina episodes or acute myocardial infarction.
Platelets: 140,000–400,000/mm <sup>3</sup>	Platelets are the first line of protection against bleeding. Once activated by blood vessel wall injury or rupture of atherosclerotic plaque, platelets undergo chemical changes that form a thrombus. Several medications inhibit platelet function, including aspirin, clopidogrel, and intravenous glycoprotein IIb/IIIa inhibitors (abciximab, eptifibatide, and tirofiban). When these medications are given, it is essential to monitor for thrombocytopenia (low platelet counts).
White blood cell (WBC) count: 4500–11,000/mm <sup>3</sup>	WBC counts are monitored in patients who are immunocompromised, including patients with heart transplants or in situations where there is concern for infection (e.g., after invasive procedures or surgery).

Adapted from Fischbach, F. T., & Fischbach, M. A. (2018). *A manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Wolters Kluwer; Urden, L. D., Stacy, K. M., & Lough, M. E. (2017). *Critical care nursing: Diagnosis and management* (8th ed.). St. Louis, MO: Elsevier Mosby.

LDL is the primary transporter of cholesterol and triglycerides into the cell. One harmful effect of LDL is the deposition of these substances in the walls of arterial vessels. HDL has a protective action because it transports cholesterol away from the tissue and cells of the arterial wall to the liver for excretion (Grundy et al., 2019).

### Triglycerides

Triglycerides, composed of free fatty acids and glycerol, are stored in the adipose tissue and are a source of energy. Triglyceride levels increase after meals and are affected by stress. Diabetes, alcohol use, and obesity can elevate triglyceride levels. These levels have a direct correlation with LDL and an inverse one with HDL.

### Brain (B-Type) Natriuretic Peptide

BNP is a neurohormone that helps regulate BP and fluid volume. It is primarily secreted from the ventricles in response to increased preload with resulting elevated ventricular pressure. The level of BNP in the blood increases as the ventricular walls expand from increased pressure, making it a helpful diagnostic, monitoring, and prognostic tool in the setting of HF. Because this serum laboratory test can be quickly obtained, BNP levels are useful for prompt diagnosis of HF in settings such as the ED. Elevations in BNP can occur from a number of other conditions such as pulmonary embolus, MI, and ventricular hypertrophy. Therefore, the primary provider correlates BNP levels with abnormal physical assessment findings and other diagnostic tests before

making a definitive diagnosis of HF. A BNP level greater than 100 pg/mL is suggestive of HF.

### C-Reactive Protein

CRP is a protein produced by the liver in response to systemic inflammation. Inflammation is thought to play a role in the development and progression of atherosclerosis. The high-sensitivity CRP (hs-CRP) test is used as an adjunct to other tests to predict CVD risk. People with high hs-CRP levels (3 mg/L or greater) may be at greatest risk for CVD compared to people with moderate (1 to 3 mg/L) or low (less than 1 mg/L) hs-CRP levels (Sidawy & Perler, 2019).

### Homocysteine

Homocysteine, an amino acid, is linked to the development of atherosclerosis because it can damage the endothelial lining of arteries and promote thrombus formation. Therefore, an elevated blood level of homocysteine is thought to indicate a high risk for CAD, stroke, and peripheral vascular disease, although it is not an independent predictor of CAD. Genetic factors and a diet low in folate, vitamin B<sub>6</sub>, and vitamin B<sub>12</sub> are associated with elevated homocysteine levels. A 12-hour fast is necessary before drawing a blood sample for an accurate serum measurement. Test results are interpreted as optimal (less than 12 mcmol/L), borderline (12 to 15 mcmol/L), and high risk (greater than 15 mcmol/L) (Zipes et al., 2019).

## Chest X-Ray and Fluoroscopy

A chest x-ray is obtained to determine the size, contour, and position of the heart. It reveals cardiac and pericardial calcifications and demonstrates physiologic alterations in the pulmonary circulation. Although it does not help diagnose acute MI, it can help diagnose some complications (e.g., HF). Correct placement of pacemakers and pulmonary artery catheters is also confirmed by chest x-ray.

Fluoroscopy is an x-ray imaging technique that allows visualization of the heart on a screen. It shows cardiac and vascular pulsations and unusual cardiac contours. This technique uses a movable x-ray source, which makes it a useful aid for positioning transvenous pacing electrodes and for guiding the insertion of arterial and venous catheters during cardiac catheterization and other cardiac procedures.

## Electrocardiography

The ECG is a graphic representation of the electrical currents of the heart. The ECG is obtained by placing disposable electrodes in standard positions on the skin of the chest wall and extremities. Recordings of the electrical current

flowing between two electrodes are made on graph paper or displayed on a monitor. Several different recordings can be obtained by using a variety of electrode combinations, called *leads*. Simply stated, a lead is a specific view of the electrical activity of heart. The standard ECG is composed of 12 leads or 12 different views, although it is possible to record 15 or 18 leads.

The 12-lead ECG is used to diagnose arrhythmias, conduction abnormalities, and chamber enlargement, as well as myocardial ischemia, injury, or infarction. It can also suggest cardiac effects of electrolyte disturbances (high or low calcium and potassium levels) and the effects of antiarrhythmic medications. A 15-lead ECG adds three additional chest leads across the right precordium and is used for early diagnosis of right ventricular and left posterior (ventricular) infarction. The 18-lead ECG adds three posterior leads to the 15-lead ECG and is useful for early detection of myocardial ischemia and injury. To enhance interpretation of the ECG, the patient's age, gender, BP, height, weight, symptoms, and medications (especially digitalis and antiarrhythmic agents) are noted on the ECG requisition. (See [Chapter 22](#) for a more detailed discussion of ECG.)

## Continuous Electrocardiographic Monitoring

Continuous ECG monitoring is the standard of care for patients who are at high risk for arrhythmias. This form of cardiac monitoring detects abnormalities in heart rate and rhythm. Many systems have the capacity to monitor for changes in ST segments, which are used to identify the presence of myocardial ischemia or injury (see [Chapter 23](#)). Two types of continuous ECG monitoring techniques are used in health care settings: hardwire cardiac monitoring, found in EDs, critical care units, and progressive care units; and telemetry, found in general nursing care units or outpatient cardiac rehabilitation programs. Hardwire cardiac monitoring and telemetry systems vary in sophistication; however, most systems have the following features in common:

- Monitor more than one ECG lead simultaneously.
- Monitor ST segments (ST-segment depression is a marker of myocardial ischemia; ST-segment elevation provides evidence of an evolving MI).
- Provide graded visual and audible alarms (based on priority, asystole merits the highest grade of alarm).
- Interpret and store alarms.
- Trend data over time.
- Print a copy of rhythms from one or more specific ECG leads over a set time (called a *rhythm strip*).
- Save electronic copies of cardiac rhythms into the electronic health record (EHR).



### Quality and Safety Nursing Alert

*Patients placed on continuous ECG monitoring must be informed of its purpose and cautioned that it does not detect shortness of breath, chest pain, or other ACS symptoms. Thus, patients are instructed to report new or worsening symptoms immediately.*



### Hardwire Cardiac Monitoring

Hardwire cardiac monitoring is used to continuously observe the heart for arrhythmias and conduction disorders using one or two ECG leads. A real-time ECG is displayed on a bedside monitor and at a central monitoring station. In critical care units, additional components can be added to the bedside monitor to continuously monitor hemodynamic parameters (noninvasive BP, arterial pressures, pulmonary artery pressures), respiratory parameters (respiratory rate, oxygen saturation), and ST segments for myocardial ischemia. The nurse must know the specific indication for each patient's ECG monitoring.

### Telemetry

In addition to hardwire cardiac monitoring, the ECG can be continuously observed by **telemetry**—the transmission of radio waves from a battery-operated transmitter to a central bank of monitors. The primary benefit of using telemetry is that the system is wireless, which allows patients to ambulate while one or two ECG leads are monitored. The patient has electrodes placed on the chest with a lead cable that connects to the transmitter. The transmitter can be placed in a disposable pouch and worn around the neck, or simply secured to the patient's clothing. Most transmitter batteries are changed every 24 to 48 hours.

### Lead Systems

The number of electrodes needed for hardwire cardiac monitoring and telemetry is dictated by the lead system used in the clinical setting. Electrodes need to be securely and correctly placed on the chest wall to accurately capture arrhythmias (Sandau, Funk, Auerbach, et al., 2017). [Chart 21-5](#) provides helpful hints on how to apply these electrodes. There are three-, four-, or five-lead systems available for ECG monitoring. The type of lead system used determines the number of lead options for monitoring. For example, the five-lead system provides up to seven different lead selections. Unlike the other two systems, the five-lead system can monitor the activity of the anterior wall of the left ventricle. [Figure 21-9](#) presents diagrams of electrode placement.

The two ECG leads most often selected for continuous ECG monitoring are leads II and V<sub>1</sub>. Lead II provides the best visualization of atrial depolarization (represented by the P wave). Lead V<sub>1</sub> best records ventricular depolarization and is most helpful when monitoring for certain arrhythmias (e.g., premature ventricular contractions, tachycardias, bundle branch blocks) (see [Chapter 22](#)). In addition to proper lead and electrode placement, it is important for the nurse to review and customize alarm settings for each patient to reduce false alarms and alarm fatigue (Jepsen, Sendelbach, Ruppel, et al., 2018).

## Chart 21-5

### Applying Electrodes

The monitoring system requires an adequate electrical signal to analyze the patient's cardiac rhythm. Proper use of this technology includes the correct application of electrodes to reduce false alarms on the cardiac monitor. When applying electrodes, the recommendations below should be followed to optimize skin adherence and conduction of the heart's electrical current:

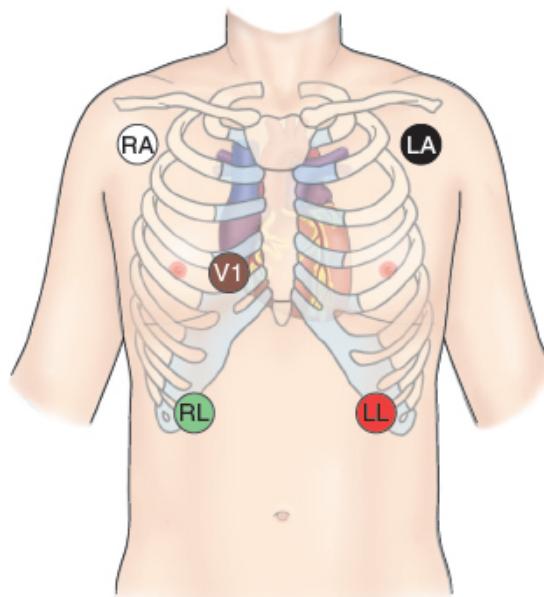
- Débride the skin surface of dead cells with soap and water; dry well using a wash cloth or gauze.
- Clip (do not shave) hair from around the electrode site, if needed.
- Connect the electrodes to the lead wires prior to placing them on the chest (connecting lead wires when electrodes are in place may be uncomfortable for some patients).
- Peel the backing off the electrode, and check to make sure the center is moist with electrode gel.
- Locate the appropriate lead placement, and apply the electrode to the skin, securing it in place with light pressure.
- Change the electrodes every 24 hours, examine the skin for irritation, and apply the electrodes to different locations.
- If the patient is sensitive to the electrodes, use hypoallergenic electrodes.

Adapted from Jepsen, S., Sendelbach, S., Ruppel, H., et al. (2018). AACN Practice Alert: Managing alarms in acute care across the life span: Electrocardiography and pulse oximetry. *Critical Care Nurse*, 38(2), e16–e20.

### Ambulatory Electrocardiography

Ambulatory electrocardiography is a form of continuous or intermittent ECG home monitoring. It is used for longer-term monitoring, since some arrhythmias occur intermittently and are difficult to capture with a 12-lead ECG in the office setting. This monitoring can help to identify the etiology of chest pain, syncope or palpitation caused by arrhythmias, detect episodes of

myocardial ischemia, evaluate effectiveness of treatment of HF and arrhythmias, and evaluate the functioning of ICDs and pacemakers. Several types of devices are available and are worn either externally or implanted under the skin. The ECG is transmitted to a centralized monitoring station via telephone or wireless technology to a secure Web site (Sampson, 2019).



- RA – Right arm (white)
- LA – Left arm (black)
- RL – Right leg (green)
- LL – Left leg (red)
- V<sub>1</sub> – Chest or precordium (brown)

**Figure 21-9 •** Electrode placement used in continuous electrocardiographic monitoring for three-lead system, placement on RA, LA, and LL; four-lead system, placement on RA, LA, RL, and LL; five-lead system, placement on RA, LA, RL, LL, and V<sub>1</sub>.

### Continuous Monitors

Commonly called Holter monitors, these small portable recorders are connected to chest electrodes (number varies based on model used) that record all ECG activity using two or more leads onto a digital memory device. The patient usually wears the recorder for 24 to 48 hours. The patient is also asked to keep a diary to note the date and time of symptoms and activities. The diary is used by the primary provider to correlate symptoms with detected arrhythmias. Once monitoring is completed, the patient returns the device and diary to the primary provider's office. Data from the digital memory device are then uploaded into a computer for analysis, and rhythms that need further

evaluation by the primary provider are identified. Therefore, Holter monitors do not provide real-time ECG recordings or analysis. The effectiveness of this form of monitoring is dependent upon the patient's adherence with wearing the monitor, keeping an accurate diary, and if an arrhythmia occurred during the monitoring period (Sampson, 2019; Urden et al., 2017).

A novel alternative to the use of the Holter monitor is ECG patch monitoring, which uses Bluetooth technology. An ECG patch with adhesive backing is placed over the left pectoral area, eliminating the need for multiple ECG electrodes, wires, and recorders. The patch is single use, waterproof, and easily concealed under clothing. The patient wears the patch for 7 to 14 days and then returns it to the manufacturer for analysis. This device can detect more arrhythmias compared to the Holter monitor since it is worn by the patient for a longer period of time (Sampson, 2019).

### Intermittent Monitors

Intermittent cardiac event monitors are devices that can capture arrhythmias when the patient experiences symptoms such as palpitations, dizziness, or lightheadedness. Patients may need to record events for several days to a month. The recorded ECGs are transmitted to the primary provider by telephone or a wireless transmission. The symptom event monitor and external loop recorder are two common forms of external intermittent cardiac event monitors.

The symptom event monitor is used to record and store the ECG during only during times when the patient is experiencing symptoms. Patients activate the symptom event monitor by pressing a button for devices that are worn on the wrist or by placing a small handheld device over the chest.

The loop recorder, a small battery-operated device, can record and store short periods of ECG activity. The monitor is inserted under the skin or worn on a wrist band. Some loop recorders are programmed to detect bradycardia, tachycardia, and irregular rhythms and do not require patient interaction. Other loop recorders require the patient to activate ECG recordings by pushing a button. The device records the patient's ECG for a predetermined time before and after the device activation. This is a preferred method over the symptom event monitor because it has more monitoring capabilities.

Real-time smart phone monitoring is a novel approach to cardiac event recording. The KardiaMobile® is a small device that connects to a smart phone app. The patient places two fingers on the device which generates a PDF file that can be sent electronically to the provider. Similarly, a patient can place a finger on the digital section of the Apple watch® to generate a rhythm strip. This rhythm is displayed on the watch face and a PDF file is sent to an app on the Apple iPhone® (Sampson, 2019).

### Cardiac Implantable Electronic Devices

Cardiac implantable electronic devices include pacemakers and ICDs. These lifesaving devices are used to manage patients with serious cardiac illnesses. The technology available today allows for remote wireless monitoring of these devices to determine battery life, pacing parameters and therapies, and occurrence of serious atrial and ventricular arrhythmias. A transmitter, which is placed in the patient's home, sends device data to a secure data repository on a secure Web portal. A unique feature of these implantable devices is that they have programmable alerts that automatically detect and transmit arrhythmias without the need for patient interaction (see [Chapter 22](#) for further discussion).

An implantable cardiac monitor such as the Reveal LINQ™ is another type of electronic device. This small device is implanted subcutaneously under the skin and has a battery life of 3 years. Recordings are triggered when an arrhythmia is detected by the device. Recordings are sent wirelessly via a home monitoring device for expert evaluation. Compared with event recorders described previously, this device offers advantages such as eliminating the patient's need to change electrodes and wear or carry the monitoring device. This type of monitoring is recommended for patients who have infrequent symptoms or require longer-term ECG monitoring (Sampson, 2019).

### Nursing Interventions for Inpatient Cardiac Monitoring

A body of evidence indicates that most alarms occurring during inpatient ECG monitoring are false alarms. Nurses dealing with excessive alarms become desensitized to these sounds and develop alarm fatigue. Alarm fatigue delays response time or results in missed alarms. Several nursing interventions facilitate acquisition of accurate data, reduce risk of alarm fatigue, and ensure patient safety when using cardiac monitoring (Jepsen et al., 2018; Sandau et al., 2017).

To minimize false alarms, the ECG recordings must be free of artifact, which is an abnormal ECG pattern caused by muscular activity, patient movement, electrical interference, or lead cable or electrode malfunction. Artifact can mimic arrhythmias and cause unnecessary false alarms. Key to the elimination of artifact is using proper skin preparation before applying electrodes and changing the electrodes every 24 hours. During electrode changes, the skin should be assessed for allergic responses (itchy, reddened skin) to the adhesive or electrode gel. If present, the electrodes are replaced with hypoallergenic electrodes. Rotation of electrode placement on the skin will reduce the risk for skin breakdown (see [Fig. 21-9](#)).

Electrodes and lead connections need to be positioned correctly. Improper positioning can result in artifact that mimics ischemia or arrhythmias. Two leads should be selected that provide the best tracing for arrhythmia monitoring, which are usually lead II and the chest lead V<sub>1</sub>. Electrical equipment in use around the patient should be inspected to be certain that it is functioning properly and has been recently checked by the medical

engineering department per organization policy, because improperly functioning equipment may cause false alarms from artifact.

An effort should be made to individualize the ECG alarm parameters to meet the patient's monitoring needs. For example, if the patient has atrial fibrillation, it is appropriate to turn off the irregular heart rate alarm. Keeping it on will create unnecessary alarms, contributing to alarm fatigue. Similarly, the bradycardia and tachycardia alarms should be adjusted, slightly below or above the patient's underlying heart rate (Jepsen et al., 2018; Sandau et al., 2017).

The nurse's role is to respond to and correct all monitor alarms immediately. Inoperative (inop) monitoring alarms—used to communicate that electrodes have fallen off, that leads are loose, or that the system's battery power is low (e.g., telemetry)—are just as significant as arrhythmia alarms indicating that the patient is tachycardic, bradycardic, or experiencing another potentially life-threatening arrhythmia. Timely responses to all alarms can prevent serious consequences, including death.

Hospital-acquired infections can be transmitted through lead wire cables. This may be prevented by using disposable lead wire cables, or by keeping reusable cables and transmitter equipment clean, per organizational policy. A patient should never be connected to monitoring equipment that has not been thoroughly cleaned between patients. If a patient is scheduled for a device implant, such as a pacemaker, electrodes should not be placed over the planned incision site. Likewise, electrodes should never be placed over an incision, implanted device, open wounds, or inflamed skin.

Electrodes should be removed once monitoring is discontinued and skin cleansed to remove excess electrode gel and adhesive. Metal-containing electrodes must be removed before sending a patient for any magnetic resonance scan, including magnetic resonance angiography (MRA).

Telemetry transmitters and other monitoring equipment should be maintained according to the manufacturer's recommendations. Monitoring devices of any type should not be submerged in water. A monitoring device may break if dropped; therefore, it should be secured to the patient's gown or clothing.

## Cardiac Stress Testing

Normally, the coronary arteries dilate to four times their usual diameter in response to increased metabolic demands for oxygen and nutrients. However, coronary arteries affected by atherosclerosis dilate less, compromising blood flow to the myocardium and causing ischemia. Therefore, abnormalities in cardiovascular function are more likely to be detected during times of increased oxygen demand, or “stress.” The **cardiac stress test** procedures—the exercise stress test, pharmacologic stress test, and radionucleotide imaging

studies—are noninvasive ways to evaluate if there is myocardial ischemia and higher myocardial oxygen requirement during these tests. Cardiac imaging is performed during the resting state and immediately after stress testing. The results can identify specific coronary artery lesions and ischemic areas of the heart (King, 2017). Since complications of stress testing can be life-threatening (MI, cardiac arrest, HF, and bradycardia and tachycardia with hemodynamic compromise), testing facilities must have staff and equipment ready to provide treatment, including advanced cardiac life support.

## **Exercise Stress Testing**

### **Procedure**

During an exercise stress test, the patient walks or runs on a treadmill (most common) or pedals a stationary bicycle. A protocol guides exercise intensity based upon the patient's age and heart rate goal (King, 2017). During the test, the following are monitored: two or more ECG leads for heart rate, rhythm, and ischemic changes; BP; skin temperature; physical appearance; perceived exertion; and symptoms, including chest pain, dyspnea, dizziness, leg cramping, and fatigue. The test is stopped when the target heart rate is achieved or if the patient experiences signs of myocardial ischemia. Abnormal findings include chest pain, ventricular arrhythmia, ST-segment depression, and lack of heart rate or BP elevation with exercise (King, 2017).

### **Nursing Interventions**

In preparation for the exercise stress test, the patient is instructed to fast for several hours before the test and to avoid stimulants such as tobacco and caffeine. Medications may be taken with sips of water. The primary provider may instruct the patient to hold beta-blockers, calcium channel blockers, and digitalis for up to 48 hours before the stress test. Clothes and sneakers or rubber-soled shoes suitable for exercising are to be worn. The nurse prepares the patient for the stress test by describing how the stress test is performed, the type of monitoring equipment used, the rationale for insertion of an IV catheter, and what symptoms to report. The exercise method is reviewed, and patients are asked to put forth their best exercise effort. If the test is to be performed with echocardiography or radionuclide imaging (described in the next section), this information is reviewed as well. After the test, the patient is monitored for 10 to 15 minutes until vital signs and assessment findings return to normal. Once stable, patients may resume their usual activities.

## **Pharmacologic Stress Testing**

### **Procedure**

Patients who are cognitively impaired and unable to follow directions or physically disabled or deconditioned will not be able to achieve their target

heart rate by exercising on a treadmill or bicycle. Vasodilating agents such as dipyridamole, adenosine, or regadenoson given as an IV infusion are used to mimic the effects of exercise by maximally dilating normal coronary arteries and identifying stenotic arteries that cannot vasodilate. The side effects of these agents are related to the vasodilating action and include chest pain, headache, flushing, nausea, heart block, and dyspnea. If necessary the effects of these drugs can be reversed with IV aminophylline. Adenosine has an extremely short half-life (less than 10 seconds), so any severe effects subside rapidly. These vasodilating medications are the agents used in conjunction with radionuclide imaging techniques. Patients undergoing pharmacologic stress tests must avoid xanthine derivatives including theophylline, aminophylline, and caffeine as they block the effects of the vasodilating agents.

Dobutamine is another option for use during a pharmacologic stress test. This medication is a synthetic sympathomimetic agent that increases heart rate, myocardial contractility, and BP, thereby increasing the metabolic demands of the heart. It is the agent of choice when echocardiography is used because of its effects on altering myocardial wall motion (due to enhanced contractility). Dobutamine is also used for patients who have bronchospasm or pulmonary disease and cannot tolerate having doses of theophylline withheld.

### Nursing Interventions

In preparation for the pharmacologic stress test, the patient is instructed not to eat or drink anything for at least 3 hours before the test. The patient must also be told to refrain from eating any liquid or food that contain chocolate or caffeine for 24 hours and to avoid taking medications that contain caffeine. This restriction also includes caffeine-free coffee, tea, and carbonated beverages. If caffeine is ingested before a stress test using vasodilating agents, the test will have to be rescheduled. Patients taking aminophylline, theophylline, or dipyridamole are instructed to stop taking these medications for 24 to 48 hours before the test (if tolerated). The patient is informed about the transient sensations that may occur during infusion of the vasodilating agent, such as flushing or nausea, which will disappear quickly. The patient is instructed to report the occurrence of any other symptoms during the test to the cardiologist or nurse. The stress test may take about 1 hour, or up to 3 hours if imaging is performed.

## Radionuclide Imaging

Radionuclide imaging studies are noninvasive tests that use radioisotopes to evaluate coronary artery perfusion, detect myocardial ischemia and infarction, and/or assess left ventricular function. **Radioisotopes** are unstable atoms that give off small amounts of energy in the form of gamma rays as they decay. When radioisotopes are injected into the bloodstream, the energy emitted can

be detected by a gamma scintillation camera positioned over the body. These radioisotopes are called tracers.

## Myocardial Perfusion Imaging

Myocardial perfusion imaging is performed using two types of techniques: single photon emission computed tomography (SPECT) or positron emission tomography (PET). It is commonly performed after an acute MI to determine if arterial perfusion to the heart is compromised during activity and to evaluate the extent of myocardial damage. It is also used to evaluate if myocardial ischemia from CAD is the cause of chest pain or other CAD-related symptoms.

These imaging techniques are performed in combination with stress testing to compare images obtained when the heart is resting to images of the heart in a stressed state resulting from exercise or medications. An area of the myocardium that shows no perfusion or reduced perfusion is said to have a “defect” present. Comparing resting images with images taken after the stress test helps differentiate ischemic myocardium from infarct-related myocardium. A defect that does not change in size before and after stress is called a fixed defect. Fixed defects indicate that there is no perfusion in that area of the myocardium, which is the case after an MI. Defects that appear or that get larger after the stress test images are taken indicate reduced perfusion to that area of the heart. Because the defect disappears with rest, it is called a reversible defect. Reversible defects constitute positive stress test findings. Typically, cardiac catheterization is recommended after a positive test result to determine the severity of obstructions to blood flow caused by CAD.

The patient undergoing myocardial perfusion imaging with stress testing should be prepared for the type of stressor to be used (exercise or medication) and provided with details of what to expect during imaging. The imaging is performed in two stages. Usually, the resting images are taken first. An IV is inserted to administer the radioisotope, and electrodes are placed on the chest to monitor the heart rate and rhythm. Women who are nursing, pregnant or think they are pregnant should not undergo myocardial perfusion imaging. The nurse alerts the primary provider if any of these conditions are present.

### Single Photon Emission Computed Tomography

SPECT is widely available and is the most common technique of myocardial perfusion imaging. In addition, the ability of SPECT to detect myocardial ischemia is between 80% and 90% (King, 2017).

### Procedure

SPECT is a painless, noninvasive procedure that involves the injection of the nuclear medicine radionucleotide (technetium-99m [ $^{99m}\text{Tc}$ ]; rubidium-82) and imaging. During SPECT, patients are positioned supine on the table with their

arms over their heads. The gamma camera circles around the chest area converting the signals from the traces into pictures of the heart. The procedure takes approximately 30 minutes. The second scan is repeated after an exercise or pharmacologic stress test.

### Nursing Interventions

The nurse's primary role is to prepare the patient for SPECT and insert an IV catheter or assess an existing IV for patency and suitability. The IV is used to inject the tracer. The patient may be concerned about receiving a radioactive substance and needs to be reassured that these tracers are safe—the radiation exposure is similar to that of other diagnostic x-ray studies. No postprocedure radiation precautions are necessary.

### Positron Emission Tomography

PET is another noninvasive procedure in which a radioactive tracer chemical is administered to the patient and images are obtained. These images generally have a higher resolution compared to SPECT. PET technology is expensive and is likely to be found at large or academic medical centers.

### Procedure

During PET, tracers are given by injection; one compound is used to determine blood flow in the myocardium, and another determines the metabolic function. The PET camera provides detailed three-dimensional images of the distributed compounds. The viability of the myocardium is determined by comparing the extent of glucose metabolism in the myocardium to the degree of blood flow. For example, ischemic but viable tissue will show decreased blood flow and elevated metabolism. For a patient with this finding, revascularization through surgery or angioplasty will probably be indicated to improve heart function. Restrictions of food intake before the test vary among institutions, but because PET evaluates glucose metabolism, the patient's blood glucose level should be within the normal range before testing.

### Nursing Interventions

The nurse instructs the patient to refrain from using alcohol and caffeine for 24 hours before undergoing PET because of the stimulating effects they may have on the heart. For patients with diabetes and who are taking insulin, the nurse needs to discuss insulin doses and food restrictions with the primary provider. The nurse assesses patients for fear of closed spaces or claustrophobia. Patients who have this condition are reassured that medications can be given to help them relax. The nurse also reassures patients that radiation exposure is at safe and acceptable levels, similar to those of other diagnostic x-ray studies.

To prepare the patient for PET, the nurse inserts an IV or assesses the existing IV catheter for patency and suitability, and then describes the

procedure to the patient. The patient is positioned on a table with hands above the head. The table then slides into a donut-shaped scanner. While in the scanner, the patient must lie still so that clear images of the heart can be obtained. A baseline scan is performed, which takes about 30 minutes. Then a tracer is injected into the IV and the scan is repeated. The patient's glucose level is monitored throughout the procedure. The scan takes from 1 to 3 hours to complete.

### **Test of Ventricular Function and Wall Motion**

Equilibrium radionuclide angiography (ERNA), also known as multiple-gated acquisition (MUGA) scanning, is a common noninvasive technique that uses a conventional scintillation camera interfaced with a computer to record images of the heart during several hundred heartbeats. The computer processes the data and allows for sequential viewing of the functioning heart. The sequential images are analyzed to evaluate left ventricular function, wall motion, and ejection fraction.

The patient is reassured that there is no known radiation danger and is instructed to remain motionless during the scan.

## **Additional Imaging**

Additional cardiac imaging techniques include computed tomography (CT) and MRA.

### **Computed Tomography**

#### **Procedure**

Cardiac CT scanning is a form of cardiac imaging that uses x-rays to provide accurate cross-sectional “virtual” slices of specific areas of the heart and surrounding structures. Complex mathematical and computer algorithms are used to analyze the slices to create three-dimensional images. Multidetector CT (MDCT) is a fast form of CT scanning that takes multiple slices at the same time. This technology produces high-resolution clear images of cardiac anatomy (Liddy, Buckley, Kok, et al., 2018). Two types of cardiac CT scanning include coronary CT angiography and electron beam CT (EBCT) (for coronary calcium scoring).

Coronary CT angiography requires the use of an IV contrast agent to enhance the x-rays and improve visualization of cardiac structures. This test is used to evaluate coronary arteries for stenosis, the aorta for aneurysms or dissections, graft patency after coronary artery bypass grafting, pulmonary veins in patients with atrial fibrillation, and cardiac structures for congenital anomalies. Patients may receive beta-blockers prior to the scan to control heart rate and rhythm and reduce artifact. Another way to minimize artifact is to

have patients hold their breath periodically throughout the scan. Coronary CT angiography is used with caution in patients with renal insufficiency. The contrast agent used during the CT scan is excreted through the kidneys; therefore, renal function should be assessed prior to the scan. It may be necessary to administer IV hydration before and after the scan to minimize the effect of the contrast on renal function. Patients will require premedication with corticosteroids and antihistamines if they experienced a reaction to a contrast agent in the past (Mervak, Cohan, Ellis, et al., 2017).

EBCT is used to calculate a coronary artery calcium score that is based on the amount of calcium deposits in the coronary arteries. This score is used to predict the likelihood of cardiac events, such as MI, or the need for a revascularization procedure in the future. Coronary artery calcium scoring is used for the evaluation of individuals without known CAD and offers limited incremental prognostic value for individuals with known CAD, such as those with stents and bypass grafts. Currently, EBCT is thought to be a reasonable test to consider in patients with low to intermediate risk for future CAD-related events. Results of the test may help to reclassify them to higher risk and thus intensify primary prevention measures (Arnett et al., 2019).

### Nursing Interventions

The nurse provides details of the procedure to help prepare the patient for the test. Patients need to be prepared to hold their breath at certain times during the procedure, so it is important for the nurse to practice with the patient before going for CT scan. The patient is positioned on a table, and the scanner rotates around the table during the test. The procedure is noninvasive and painless. However, to obtain adequate images, the patient must lie completely still during the scanning process. An IV is necessary if contrast is to be used to enhance the images. The patient should be told to expect transient flushing, metallic taste, nausea, or bradycardia during the contrast infusion.

## Magnetic Resonance Angiography

### Procedure

MRA is a noninvasive, painless technique that is used to examine both the physiologic and anatomic properties of the heart. MRA uses a powerful magnetic field and computer-generated pictures to image the heart and great vessels. It is valuable in diagnosing diseases of the aorta, heart muscle, and pericardium, as well as congenital heart lesions. The application of this technique to the evaluation of coronary artery anatomy is limited because the quality of the images is distorted by respirations, the beating heart, and certain implanted devices (stents and surgical clips). In addition, this technique cannot adequately visualize the small distal coronary arteries as accurately as conventional angiography performed during a cardiac catheterization.

## Nursing Interventions

Because of the magnetic field used during MRA, patients must be screened for contraindications for its use. Patients with any type of cardiac implantable electronic device need to be screened to see if it is safe for the patient to undergo the test (Indik, Gimbel, Abe, et al., 2017). MRA cannot be performed on patients who have metal plates, prosthetic joints, or other metallic implants that can become dislodged if exposed to MRA. Patients are instructed to remove any jewelry, watches, or other metal items (e.g., ECG leads). Transdermal patches that contain a heat-conducting aluminized layer (e.g., nitroglycerin, clonidine, fentanyl) must be removed before MRA to prevent burning of the skin.

During MRA, the patient is positioned supine on a table that is placed into an enclosed imager or tube containing the magnetic field. A patient who is claustrophobic may need to receive a mild sedative before undergoing an MRA. An intermittent clanking or thumping that can be annoying is generated by the magnetic coils, so the patient may be offered a headset to listen to music. The scanner is equipped with a microphone so that the patient can communicate with the staff. The patient is instructed to remain motionless during the scan.

## Echocardiography

### Transthoracic Echocardiography

Echocardiography is a noninvasive ultrasound test that is used to measure the ejection fraction and examine the size, shape, and motion of cardiac structures. It is particularly useful for diagnosing pericardial effusions; determining chamber size and the etiology of heart murmurs; evaluating the function of heart valves, including prosthetic heart valves; and evaluating ventricular wall motion.

### Procedure

Echocardiography involves transmission of high-frequency sound waves into the heart through the chest wall and the recording of the return signals. With the traditional transthoracic approach, the ultrasound is generated by a handheld transducer applied to the front of the chest. The transducer picks up the echoes and converts them to electrical impulses that are recorded and displayed on a monitor. It creates sophisticated, spatially correct images of the heart. An ECG is recorded simultaneously to assist in interpretation of the echocardiogram.

With the use of Doppler techniques, an echocardiogram can also show the direction and velocity of the blood flow through the heart. These techniques are used to assess for “leaking valves,” conditions referred to as valvular

regurgitation, and will also detect abnormal blood flow between the septum of the left and right heart.

Echocardiography may be performed with an exercise or pharmacologic stress test. Images are obtained at rest and then immediately after the target heart rate is reached. Myocardial ischemia from decreased perfusion during stress causes abnormalities in ventricular wall motion and is easily detected by echocardiography. A stress test using echocardiography is considered positive if abnormalities in ventricular wall motion are detected during stress but not during rest. These findings are highly suggestive of CAD and require further evaluation, such as a cardiac catheterization.

### Nursing Interventions

Before transthoracic echocardiography, the nurse informs the patient about the test, explaining that it is painless. Echocardiographic monitoring is performed while a transducer that emits sound waves is moved over the surface of the chest wall. Gel applied to the skin helps transmit the sound waves. Periodically, the patient is asked to turn onto the left side or hold a breath. The test takes about 30 to 45 minutes. If the patient is to undergo an exercise or pharmacologic stress test with echocardiography, information on stress testing is also reviewed with the patient.

## Transesophageal Echocardiography

### Procedure

A significant limitation of transthoracic echocardiography is the poor quality of the images produced. Ultrasound loses its clarity as it passes through tissue, lung, and bone. An alternative technique involves threading a small transducer through the mouth and into the esophagus. This technique, called *transesophageal echocardiography* (TEE), provides clearer images because ultrasound waves pass through less tissue. A topical anesthetic agent and sedation are used during TEE because of the discomfort associated with the positioning of the transducer in the esophagus (refer to [Chapter 15](#) for further discussion of sedation for procedures). Once the patient is comfortable, the transducer is inserted into the mouth and the patient is asked to swallow several times until it is positioned in the esophagus.

The high-quality imaging obtained during TEE makes this technique an important first-line diagnostic tool for evaluating patients with many types of CVD, including HF, valvular heart disease, arrhythmias, and many other conditions that place the patient at risk for atrial or ventricular thrombi. Pharmacologic stress testing using dobutamine and TEE can also be performed. It is frequently used during cardiac surgery to continuously monitor the response of the heart to the surgical procedure (e.g., valve replacement or coronary artery bypass). Complications are uncommon during TEE; however,

if they do occur, they are serious. These complications are caused by sedation and impaired swallowing resulting from the topical anesthesia (respiratory depression and aspiration) and by insertion and manipulation of the transducer into the esophagus and stomach (vasovagal response or esophageal perforation). The patient must be assessed before TEE for a history of dysphagia or radiation therapy to the chest, which increases the likelihood of complications.

### Nursing Interventions

Prior to the test, the nurse provides preprocedure education and ensures that the patient has a clear understanding of what the test entails and why it is being performed, instructs the patient not to eat or drink anything for 6 hours prior to the study, and checks to make sure that informed consent has been obtained. The nurse also inserts an IV line or assesses an existing IV for patency and suitability and asks the patient to remove full or partial dentures. During the test, the nurse provides emotional support and monitors level of consciousness, BP, ECG, respiration, and oxygen saturation ( $\text{SpO}_2$ ). During the recovery period, the patient must maintain bed rest with the head of the bed elevated to 45 degrees. Following the procedural sedation policy of the agency, the nurse monitors the patient for dyspnea and assesses vital signs,  $\text{SpO}_2$ , level of consciousness, and gag reflex as recommended. Food and oral fluids are withheld until the patient is fully alert and the effects of the topical anesthetic agent are reversed, usually 2 hours after the procedure; if the gag reflex is intact, the nurse begins feeding with sips of water, then advances to the preprocedure diet. Patients are informed that a sore throat may be present for the next 24 hours; they are instructed to report the presence of a persistent sore throat, shortness of breath, or difficulty swallowing to the medical staff. If the procedure is performed in an outpatient setting, a family member or friend must be available to transport the patient home from the test site.

## Cardiac Catheterization

**Cardiac catheterization** is a common invasive procedure used to diagnose structural and functional diseases of the heart and great vessels. The results guide treatment decisions including the need for revascularization (PCI or CABG) and other interventions to manage structural defects of the valves or septum (see [Chapter 23](#)).

This procedure involves the percutaneous insertion of radiopaque catheters into a large vein and an artery. Fluoroscopy is used to guide the advancement of the catheters through the right and left heart, referred to as right and left heart catheterizations, respectively. In most situations, patients undergo both right and left heart catheterizations. However, right heart catheterization is performed without a left heart catheterization when patients only need

myocardial biopsies or measurement of pulmonary artery pressures. Of note, left heart catheterization involves the use of a contrast agent. These agents are necessary to visualize patency of the coronary arteries and evaluate left ventricular function.

In preparation for the procedure, patients have blood tests performed to evaluate metabolic function (electrolytes and glucose) and renal function (blood urea nitrogen and creatinine level). Baseline coagulation studies (activated partial thromboplastin time [aPTT], international normalized ratio [INR], and prothrombin time [PT]) are obtained to guide dosing of anticoagulation during the procedure. Because bleeding and hematoma formation are procedural risks, a complete blood cell count (CBC; includes the hematocrit, hemoglobin, and platelets) is necessary to establish baseline values. Later these results are compared with postprocedure results to monitor for blood loss.

A health history is obtained to assess for previous reactions to a contrast agent and determine if the patient has any risk factors for contrast-induced nephropathy (CIN). This uncommon complication is a form of acute kidney injury that is usually reversible. Patients with chronic kidney disease or renal insufficiency, diabetes, HF, hypotension, dehydration, use of nephrotoxic medications, and advanced age are at risk for CIN. CIN is defined as an increase in the baseline serum creatinine by 25% or more or an absolute increase of 0.5 mg/dL within 48 to 72 hours after the administration of contrast (Urden et al., 2017). See [Chapter 47, Chart 47-5](#) for further discussion of nursing care of patient undergoing imaging study with the use of a contrast agent.

During a cardiac catheterization, the patient has one or more IV catheters for administration of fluids, sedatives, heparin, and other medications. The patient is continuously monitored for chest pain or dyspnea and for changes in BP and ECG, which are indicative of myocardial ischemia, hemodynamic instability, or arrhythmias. Resuscitation equipment must be readily available, and staff must be prepared to provide advanced cardiac life support measures as necessary.

Postprocedure, patients remain on bed rest for 2 to 6 hours before they are permitted to ambulate. Variations in time to ambulation are related to the size of the catheters used during the procedure, the site of catheter insertion (femoral or radial artery), the patient's anticoagulation status, and other factors (e.g., advanced age, obesity, bleeding disorder). The use of a radial access site and smaller (4- or 6-Fr) arterial catheters are associated with shorter bed rest restrictions.

Cardiac catheterization may be performed in the ambulatory setting. Unless the results demonstrate the need for immediate treatment, patients are discharged home. Hospitalized patients undergoing cardiac catheterization for

diagnostic and interventional purposes (PCI, valvuloplasty) are returned to their hospital rooms for recovery (see [Chapter 23](#)).

## Right Heart Catheterization

Right heart catheterization usually precedes left heart catheterization. It is performed to assess the function of the right ventricle and tricuspid and pulmonary valves. The procedure involves the passage of a catheter from a brachial, internal jugular, or femoral vein into the right atrium, right ventricle, pulmonary artery, and pulmonary arterioles. Pressures and oxygen saturations from each of these areas are obtained and recorded. The pulmonary artery pressures are used to diagnose pulmonary hypertension. A biopsy of a small piece of myocardial tissue can also be obtained during a right heart catheterization. The results of the biopsy are used to diagnose the etiology of a cardiomyopathy (abnormality of myocardium) or heart transplant rejection. At the completion of the procedure, the venous catheter is removed and hemostasis of the affected vein is achieved using manual pressure. Although right heart catheterization is considered relatively safe, potential complications include arrhythmias (from contact of the catheter with the endocardium), venous spasm, infection at the insertion site, and right heart perforation.

## Left Heart Catheterization

Prior to left heart catheterization, patients who have previously experienced a reaction to a contrast agent are premedicated with antihistamines (e.g., diphenhydramine) and corticosteroids (e.g., prednisone). Patients at risk for CIN receive pre- and postprocedure preventive strategies. IV saline hydration increases vascular volume, facilitates removal of contrast from the kidneys, and reduces the risk of CIN (Liu, Hong, Wang, et al., 2019).

Left heart catheterization is performed to evaluate the aortic arch and its major branches, patency of the coronary arteries, and the function of the left ventricle and mitral and aortic valves. Left heart catheterization is performed by retrograde catheterization of the left ventricle. In this approach, the interventional cardiologist usually inserts the catheter into the right radial or a femoral artery and advances it into the aorta and left ventricle. Potential complications include arrhythmias, MI, perforation of the left heart or great vessels, and systemic embolization.

During a left heart catheterization, angiography is performed. Angiography is an imaging technique that involves the injection of the contrast agent into the arterial catheter. The contrast agent is filmed as it passes through the chambers of the left heart, aortic arch, and its major arteries. Coronary angiography is another technique used to observe the coronary artery anatomy and evaluate the degree of stenosis from atherosclerosis. To perform this test, a catheter is positioned into one of the coronary arteries. Once in position, the

contrast agent is injected directly into the artery and images are obtained. The procedure is then repeated using the opposite coronary artery. Ventriculography is also performed to evaluate the size and function of the left ventricle. For this test, a catheter is positioned in the left ventricle and a large amount of contrast agent (30 mL) is rapidly injected into the ventricle.

The manipulation of catheters in the coronary arteries and left ventricle as well as injection of the contrast agent can cause intermittent myocardial ischemia. Vigilant monitoring throughout left heart catheterization is needed to detect myocardial ischemia, which can trigger chest pain and life-threatening arrhythmias.

Once the procedure is completed, the arterial catheter is withdrawn. There are several options available to achieve arterial hemostasis, including applying manual pressure and hemostatic devices available from numerous vendors. For the radial artery, a compression device, such as the Terumo TR Band®, is positioned over the artery. It has a mechanism that is inflated with air to put pressure against the artery. It remains in place for about 2 hours. A radial approach and a compression device are both common practices and are associated with lower risks for bleeding and vascular complications, as well as a shorter time to ambulation postprocedure (Mason, Shah, Tamis-Holland, et al., 2018).

For the femoral approach, manual pressure may be used alone or in combination with mechanical compression devices such as the FemoStop™. Many types of percutaneously deployed vascular closure devices are also available. These devices are positioned at the femoral arterial puncture site after completion of the procedure. They deploy a saline-soaked gelatin sponge (QUICKSEAL), collagen (VasoSeal), sutures (Perclose ProGlide™), or a combination of both collagen and sutures (Angio-Seal). Other products that expedite arterial hemostasis include external patches (Syvek Patch, Clo-Sur P.A.D.). These products are placed over the puncture site as the catheter is removed and manual pressure is applied for 4 to 10 minutes. Once hemostasis is achieved, the patch is covered with a dressing that remains in place for 24 hours. The interventional cardiologist determines which closure device, if any, will be deployed based on the artery used to insert the catheter, patient's condition, device availability, and personal preference.

Major benefits of the vascular closure devices include reliable, immediate hemostasis and a shorter time on bed rest without a significant increase in bleeding or other complications. Rare complications associated with these devices include bleeding around the closure device, infection, and arterial obstruction.

## Nursing Interventions

Nursing responsibilities before cardiac catheterization include:

- Instructing the patient to fast, usually for 8 to 12 hours, before the procedure.
- Informing the patient that if catheterization is to be performed as an outpatient procedure, a friend, family member, or other responsible person must transport the patient home.
- Informing the patient about the expected duration of the procedure and advising that it will involve lying on a hard table for less than 2 hours.
- Reassuring the patient that IV medications are given to maintain comfort.
- Informing the patient about sensations that will be experienced during the catheterization. Knowing what to expect can help the patient cope with the experience. The nurse explains that an occasional pounding sensation (palpitation) may be felt in the chest because of extra heartbeats that almost always occur, particularly when the catheter tip touches the endocardium. The patient may be asked to cough and to breathe deeply, especially after the injection of the contrast agent. Coughing may help disrupt an arrhythmia and clear the contrast agent from the arteries. Breathing deeply and holding the breath help lower the diaphragm for better visualization of heart structures. The injection of a contrast agent into either side of the heart may produce a flushed feeling throughout the body and a sensation similar to the need to void, which subsides in 1 minute or less.
- Encouraging the patient to express fears and anxieties. The nurse provides education and reassurance to reduce apprehension.

Nursing responsibilities after cardiac catheterization are guided by hospital policy and primary provider preferences and may include:

- Observing the catheter access site for bleeding or hematoma formation and assessing peripheral pulses in the affected extremity (dorsalis pedis and posterior tibial pulses in the lower extremity, radial pulse in the upper extremity) every 15 minutes for 1 hour, every 30 minutes for 1 hour, and hourly for 4 hours or until discharge. BP and heart rate are also assessed during these same time intervals.
- Evaluating temperature, color, and capillary refill of the affected extremity during these same time intervals. The patient is assessed for affected extremity pain, numbness, or tingling sensations that may indicate arterial insufficiency. The best technique to use is to compare the examination findings between the affected and unaffected extremities. Any changes are reported promptly.

- Screening carefully for arrhythmias by observing the cardiac monitor or by assessing the apical and peripheral pulses for changes in rate and rhythm. A vasovagal reaction, consisting of bradycardia, hypotension, and nausea, can be precipitated by a distended bladder or by discomfort from manual pressure that is applied during removal of an arterial or venous catheter. The vasovagal response is reversed by promptly elevating the lower extremities above the level of the heart, infusing a bolus of IV fluid, and administering IV atropine to treat the bradycardia.
- Maintaining activity restrictions for 2 to 6 hours after the procedure. The determination of bed rest, chair activity, and time to commence ambulation is dependent upon location of arterial approach, size of the catheter used during the procedure, medications administered, and method used to maintain hemostasis. If manual pressure or a mechanical device was used during a femoral artery approach, the patient remains on bed rest for up to 6 hours with the affected leg straight and the head of the bed elevated no greater than 30 degrees. For comfort, the patient may be turned from side to side with the affected extremity straight. If a percutaneous vascular closure device or patch was deployed, the nurse checks local nursing care standards and anticipates that the patient will have fewer activity restrictions. If the radial closure device was used, the patient can sit up in a chair until the effects of sedation have dissipated, and early ambulation is encouraged. After the vascular closure device removal, a dressing is applied over the catheter access site. Patients can return to normal activities the day after the procedure but must avoid strenuous wrist activities for several days (Mason et al., 2018). Analgesic medication is given as prescribed for discomfort.
- Instructing the patient to report chest pain and bleeding or sudden discomfort from the catheter insertion sites promptly.
- Monitoring the patient for CIN by observing for elevations in serum creatinine levels. IV hydration is used to increase urinary output and flush the contrast agent from the urinary tract; accurate oral and IV intake and urinary output are recorded.
- Ensuring patient safety by instructing the patient to ask for help when getting out of bed the first time after the procedure. The patient is monitored for bleeding from the catheter access site and for orthostatic hypotension, indicated by complaints of dizziness or lightheadedness.

For patients being discharged from the hospital on the same day as the procedure, additional instructions are provided ([Chart 21-6](#)).

## Electrophysiologic Testing

The electrophysiology study (EPS) is an invasive procedure that plays a major role in the diagnosis and management of serious arrhythmias. EPS may be indicated for patients with syncope, palpitations, or both, and for survivors of cardiac arrest from ventricular fibrillation (sudden cardiac death) (Kusumoto, Schoenfeld, Barrett, et al., 2019). EPS is used to distinguish atrial from ventricular tachycardias when the determination cannot be made from the 12-lead ECG; to evaluate how readily a life-threatening arrhythmia (e.g., ventricular tachycardia, ventricular fibrillation) can be induced; to evaluate AV node function; to evaluate the effectiveness of antiarrhythmic medications in suppressing the arrhythmia; or to determine the need for other therapeutic interventions, such as a cardiac implantable electronic device, or radiofrequency ablation. (See [Chapter 22](#) for a detailed discussion of EPS.)

### Chart 21-6 PATIENT EDUCATION



## Self-Management After Cardiac Catheterization

After discharge from the hospital for cardiac catheterization, patients should follow these guidelines for self-care:

- *If the artery in your wrist artery was used:* Return to normal activities tomorrow. Strenuous activities of the wrist such as manual labor, tennis, or driving may be restricted for a few days per your provider's orders.
- *If the artery in your groin was used:* For the next 24 hours, do not bend at the waist, strain, or lift heavy objects.
- Do not submerge the puncture site in water. Avoid tub baths, but shower as desired.
- Talk with your primary provider about when you may return to work, drive, or resume strenuous activities.
- If bleeding occurs, sit (arm or wrist approach) or lie down (groin approach) and apply firm pressure to the puncture site for 10 minutes. Notify your primary provider as soon as possible and follow instructions. If there is a large amount of bleeding, call 911. Do not drive to the hospital.
- Call your primary provider if any of the following occur: swelling, new bruising or pain from your procedure puncture site, temperature of 101°F or more.
- If test results show that you have coronary artery disease, talk with your primary provider about options for treatment, including cardiac rehabilitation programs in your community.
- Talk with your primary provider about lifestyle changes to reduce your risk for further or future heart problems, such as quitting smoking, lowering your cholesterol level, initiating dietary changes, beginning an exercise program, cardiac rehabilitation, or losing weight.
- Your primary provider may prescribe one or more new medications depending on your risk factors (medications to lower your blood pressure or cholesterol; aspirin or clopidogrel to prevent blood clots). Take all of your medications as instructed. If you feel that any of them are causing side effects, call your primary provider immediately. Do not stop taking any medications before talking to your primary provider.

Adapted from Mason, P. J., Shah, B., Tamis-Holland, J. E., et al. (2018). An update on radial artery access and best practices for transradial coronary angiography and intervention in acute coronary syndrome: A scientific statement from the American Heart Association. *Circulation: Cardiovascular Interventions*. Retrieved on 5/14/2019 at: [www.ahajournals.org/toc/circinterventions/11/9](http://www.ahajournals.org/toc/circinterventions/11/9)



## Hemodynamic Monitoring

Critically ill patients require continuous assessment of their cardiovascular system to diagnose and manage their complex medical conditions. This type of assessment is achieved by the use of direct pressure monitoring systems, referred to as hemodynamic monitoring. Common forms include CVP, pulmonary artery pressure, and intra-arterial BP monitoring. Patients requiring hemodynamic monitoring are cared for in critical care units. Some progressive care units also admit stable patients with CVP, pulmonary artery catheters, or intra-arterial BP monitoring. To perform hemodynamic monitoring, a CVP, pulmonary artery, or arterial catheter is introduced into the appropriate blood vessel or heart chamber. It is connected to a pressure monitoring system that has several components, including:

- A disposable flush system, composed of IV normal saline solution, tubing, stopcocks, and a flush device, which provides continuous and manual flushing of the system.
- A pressure bag placed around the flush solution that is maintained at 300 mm Hg of pressure. The pressurized flush system delivers 3 to 5 mL of solution per hour through the catheter to prevent clotting and backflow of blood into the pressure monitoring system.
- A transducer to convert the pressure coming from the artery or heart chamber into an electrical signal.
- An amplifier or monitor, which increases the size of the electrical signal for display on an oscilloscope.

Nurses caring for patients who require hemodynamic monitoring receive training prior to using this sophisticated technology. The nurse helps ensure safe and effective care by adhering to the following guidelines:

- Ensuring that the system is set up and maintained properly. For example, the pressure monitoring system must be kept patent and free of air bubbles.
- Checking that the stopcock of the transducer is positioned at the level of the atrium before the system is used to obtain pressure measurements. This landmark is referred to as the phlebostatic axis ([Fig. 21-10](#)). The nurse uses a marker to identify this level on the chest wall, which provides a stable reference point for subsequent pressure readings.
- Establishing the zero reference point in order to ensure that the system is properly functioning at atmospheric pressure. This process is accomplished by placing the stopcock of the transducer at the phlebostatic axis, opening the transducer to air, and activating the zero function key on the bedside monitor. Measurements of CVP, BP, and pulmonary artery pressures can be made with the head of the bed elevated up to 60 degrees;

however, the system must be repositioned to the phlebostatic axis to ensure an accurate reading (Urden et al., 2017).

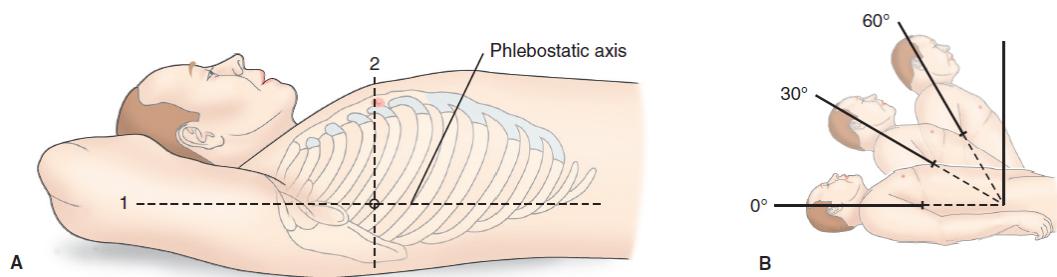
Complications from the use of hemodynamic monitoring systems are uncommon and can include pneumothorax, infection, and air embolism. The nurse observes for signs of pneumothorax during the insertion of catheters using a central venous approach (CVP and pulmonary artery catheters). The longer any of these catheters are left in place (after 72 to 96 hours), the greater the risk of infection. Air emboli can be introduced into the vascular system if the stopcocks attached to the pressure transducers are mishandled during blood drawing, administration of medications, or other procedures that require opening the system to air. Therefore, nurses handling this equipment must demonstrate competence prior to caring independently for a patient requiring hemodynamic monitoring.

Catheter-related bloodstream infections are the most common preventable complication associated with hemodynamic monitoring systems. The Centers for Disease Control and Prevention (CDC) has published comprehensive guidelines for the prevention of these infections (O’Grady, Alexander, Burns, et al., 2011; Talbot, Stone, Irwin, et al., 2017). To minimize the risk of infection, a group of evidence-based interventions, called a care *bundle*, should be implemented.

The CDC and Infusion Nursing Society have additional infection control guidelines that pertain to the ongoing care of these patients, including skin care, dressing changes, and line and connector care that are outlined in [Table 21-5](#).

### Central Venous Pressure Monitoring

CVP is a measurement of the pressure in the vena cava or right atrium. The pressure in the vena cava, right atrium, and right ventricle is equal at the end of diastole; thus, the CVP also reflects the filling pressure of the right ventricle (preload). The normal CVP is 2 to 6 mm Hg. It is measured by positioning a catheter in the vena cava or right atrium and connecting it to a pressure monitoring system. The CVP is most valuable when it is monitored over time and correlated with the patient’s clinical status. A CVP greater than 6 mm Hg indicates an elevated right ventricular preload. There are many problems that can cause an elevated CVP, but the most common problem is hypervolemia (excessive fluid circulating in the body) or right-sided HF. In contrast, a low CVP (less than 2 mm Hg) indicates reduced right ventricular preload, which is most often from hypovolemia. Dehydration, excessive blood loss, vomiting or diarrhea, and overdiuresis can result in hypovolemia and a low CVP. This diagnosis can be substantiated when a rapid IV infusion of fluid causes the CVP to increase.



**Figure 21-10 • A.** The phlebostatic axis is the reference point for the atrium when the patient is positioned supine. It is the intersection of two lines on the chest wall: (1) the midaxillary line drawn between the anterior and posterior surfaces of the chest and (2) the line drawn through the fourth intercostal space. Its location is identified with a skin marker. The stopcock of the transducer used in hemodynamic monitoring is “leveled” at this mark prior to taking pressure measurements. **B.** Measurements can be taken with the head of the bed (HOB) elevated up to 60 degrees. Note the phlebostatic axis changes as the HOB is elevated; thus, the stopcock and transducer must be repositioned after each position change.

**TABLE 21-5**

## Nursing Interventions to Prevent Intravascular Catheter-Related Bloodstream Infections

Topic	Intervention
Hand hygiene	<ul style="list-style-type: none"> <li>Wash hands with soap and water or use alcohol-based hand rubs before and after contact with the catheter for any reason.</li> </ul>
Dressing	<ul style="list-style-type: none"> <li>Wear clean or sterile gloves when changing the dressing.</li> <li>Cleanse the skin during dressing changes with a &gt;0.5% chlorhexidine preparation with alcohol.</li> <li>Use a chlorhexidine-impregnated dressing at the catheter insertion site. Do not use topical antibiotic ointment or creams on insertion sites.</li> <li>Dress the site with sterile gauze or sterile, transparent, semipermeable dressing to cover the catheter site. If the patient is diaphoretic or if the site is bleeding or oozing, use a gauze dressing until it is resolved.</li> <li>Change gauze dressings every 2 d or transparent dressings at least every 7 d and whenever dressings become damp, loosened, or visibly soiled.</li> </ul>
Catheter site	<ul style="list-style-type: none"> <li>Assess the site regularly—visually when changing the dressing or by palpation through an intact dressing. Remove the dressing for a thorough assessment if the patient has tenderness at the insertion site, fever without obvious source, or other signs of local or bloodstream infection.</li> </ul>
Needleless catheter systems	<ul style="list-style-type: none"> <li>Change needleless connectors, administration sets, and pressure tubing per institutional policy, usually every 96 h.</li> <li>Scrub ports, connectors, and hubs with alcohol, chlorhexidine/alcohol, or povidone–iodine before and after access.</li> <li>Apply alcohol-impregnated caps to needless connectors between uses.</li> </ul>
Bathing	<ul style="list-style-type: none"> <li>Clean the skin daily with a 2% chlorhexidine wash.</li> <li>Do not submerge the catheter or catheter site in water.</li> <li>Showering is permitted if the catheter and related tubing are placed in an impermeable cover.</li> </ul>

#### Patient education

- Ask patients to report any new discomforts from the catheter site.

Adapted from Gorski, L. A., Hadaway, L., Hagle, M., et al. (2016). Infusion therapy standards of practice. *Journal of Infusion Nursing*, 39(1 Suppl.), S1–S159; O’Grady, N. P., Alexander, A., Burns, L., et al. (2011). 2017 Update of the guidelines for the prevention of intravascular catheter-related infections, 2011. Retrieved on 5/15/2019 at: [www.cdc.gov/infectioncontrol/pdf/guidelines/bsi-guidelines-H.pdf](http://www.cdc.gov/infectioncontrol/pdf/guidelines/bsi-guidelines-H.pdf); Talbot, T. R., Stone, E. C., Irwin, K., et al. (2017). 2017 Updated recommendations on the use of chlorhexidine-impregnated dressings for prevention of intravascular catheter-related infections. Retrieved on 5/15/ 2019 at: [www.cdc.gov/infectioncontrol/pdf/guidelines/c-i-dressings-H.pdf](http://www.cdc.gov/infectioncontrol/pdf/guidelines/c-i-dressings-H.pdf)

Before insertion of a CVP catheter, the site is prepared as recommended by the CDC (see [Chapter 11, Chart 11-2](#)). The preferred site is the subclavian vein; the femoral vein is generally avoided (O’Grady et al., 2011). A local anesthetic agent is used. During this sterile procedure, the physician threads a single-lumen or multilumen catheter through the vein into the vena cava just above or within the right atrium. Once the CVP catheter is inserted, it is secured and a dry sterile dressing is applied. Position of the catheter is confirmed by a chest x-ray.

### Nursing Interventions

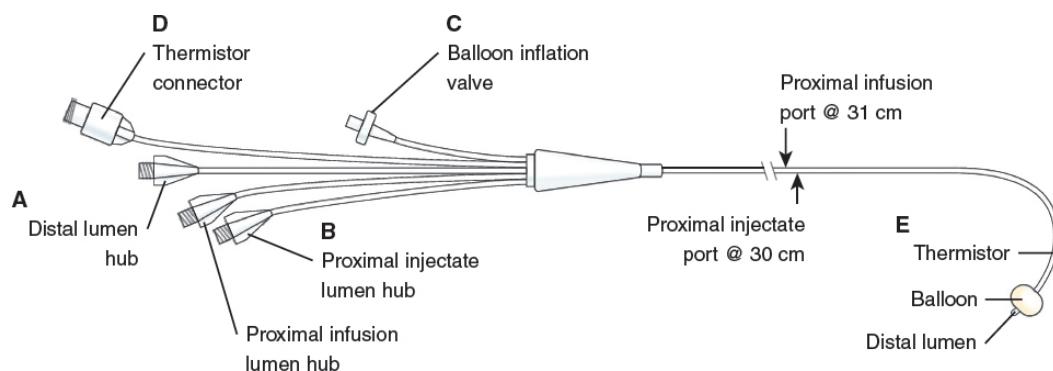
The frequency of CVP measurements is dictated by the patient’s condition and the treatment plan. In addition to obtaining pressure readings, the CVP catheter is used for infusing IV fluids, administering IV medications, and drawing blood specimens. Nursing care of the patient with a CVP catheter follows central line and pressure tubing guidelines (see [Table 21-5](#)).

### Pulmonary Artery Pressure Monitoring

Pulmonary artery pressure monitoring is used in critical care for assessing left ventricular function, diagnosing the etiology of shock, and evaluating the patient’s response to medical interventions (e.g., fluid administration, vasoactive medications). A pulmonary artery catheter and a pressure monitoring system are used. A variety of catheters are available for cardiac pacing, oximetry, cardiac output measurement, or a combination of functions. Pulmonary artery catheters are balloon-tipped, flow-directed catheters that have distal and proximal lumens ([Fig. 21-11](#)). The distal lumen has a port that opens into the pulmonary artery. Once connected by its hub to the pressure monitoring system, it is used only to continuously measure pulmonary artery pressures. The proximal lumen has a port that opens into the right atrium. It is used to administer IV medications and fluids or to monitor right atrial pressures (i.e., CVP). Each catheter has a balloon inflation hub and valve. A syringe is connected to the hub, which is used to inflate or deflate the balloon

with air (maximum 1.5-mL capacity). The valve opens and closes the balloon inflation lumen.

A pulmonary artery catheter with specialized capabilities has additional components. For example, the thermodilution catheter has three additional features that enable it to measure cardiac output: a thermistor connector attached to the cardiac output computer of the bedside monitor, a proximal injectate port used for injecting fluids when obtaining the cardiac output, and a thermistor (positioned near the distal port) (see Fig. 21-11).



**Figure 21-11 •** The pulmonary artery catheter used for obtaining pressure measurements and cardiac output. **A.** The pressure monitoring system is connected to the distal lumen hub. **B.** Intravenous solutions are infused through the proximal infusion and injectate lumen hubs. **C.** An air-filled syringe connected to the balloon inflation valve is used for balloon inflation during catheter insertion and pulmonary artery wedge pressure measurements. **D.** To obtain cardiac output, the thermistor connector is inserted into the cardiac output component of the bedside cardiac monitor, and 5 to 10 mL of normal saline is injected in 4 seconds into the proximal injectate port. **E.** The thermistor located near the balloon is used to calculate the cardiac output. Redrawn courtesy of Baxter Healthcare Corporation, Edwards Critical Care Division, Santa Ana, California.

The pulmonary artery catheter, covered with a sterile sleeve, is inserted into a large vein, preferably the subclavian, through a sheath. As noted previously, the femoral vein is avoided; insertion techniques and protocols mirror those used for inserting a CVP catheter (see previous discussion) (O'Grady et al., 2011). The sheath is equipped with a side port for infusing IV fluids and medications. The catheter is then passed into the vena cava and right atrium. In the right atrium, the balloon tip is inflated, and the catheter is carried rapidly by the flow of blood through the tricuspid valve into the right ventricle, through the pulmonic valve, and into a branch of the pulmonary artery. When the catheter reaches the pulmonary artery, the balloon is deflated and the catheter is secured with sutures (Fig. 21-12). Fluoroscopy may be used during

insertion to visualize the progression of the catheter through the right heart chambers to the pulmonary artery. This procedure can be performed in the operating room, in the cardiac catheterization laboratory, or at the bedside in the critical care unit. During insertion of the pulmonary artery catheter, the bedside monitor is observed for pressure and waveform changes, as well as arrhythmias, as the catheter progresses through the right heart to the pulmonary artery.

Once the catheter is in position, the following are measured: right atrial, pulmonary artery systolic, pulmonary artery diastolic, mean pulmonary artery, and pulmonary artery wedge pressures (see Fig. 21-2 for normal chamber pressures). Monitoring of the pulmonary artery diastolic and pulmonary artery wedge pressures is particularly important in critically ill patients because they are used to evaluate left ventricular filling pressures (i.e., left ventricular preload).

It is important to note that the pulmonary artery wedge pressure is achieved by inflating the balloon tip for a maximum of 15 seconds, which causes it to float more distally into a smaller portion of the pulmonary artery until it is wedged into position. This is an occlusive maneuver that impedes blood flow through that segment of the pulmonary artery. Therefore, the wedge pressure is measured immediately and the balloon deflated promptly to restore blood flow.



#### **Quality and Safety Nursing Alert**

*After measuring the pulmonary artery wedge pressure, the nurse ensures that the balloon is deflated and that the catheter has returned to its normal position. This important intervention is verified by evaluating the return of the pulmonary artery systolic and diastolic waveform displayed on the bedside monitor.*

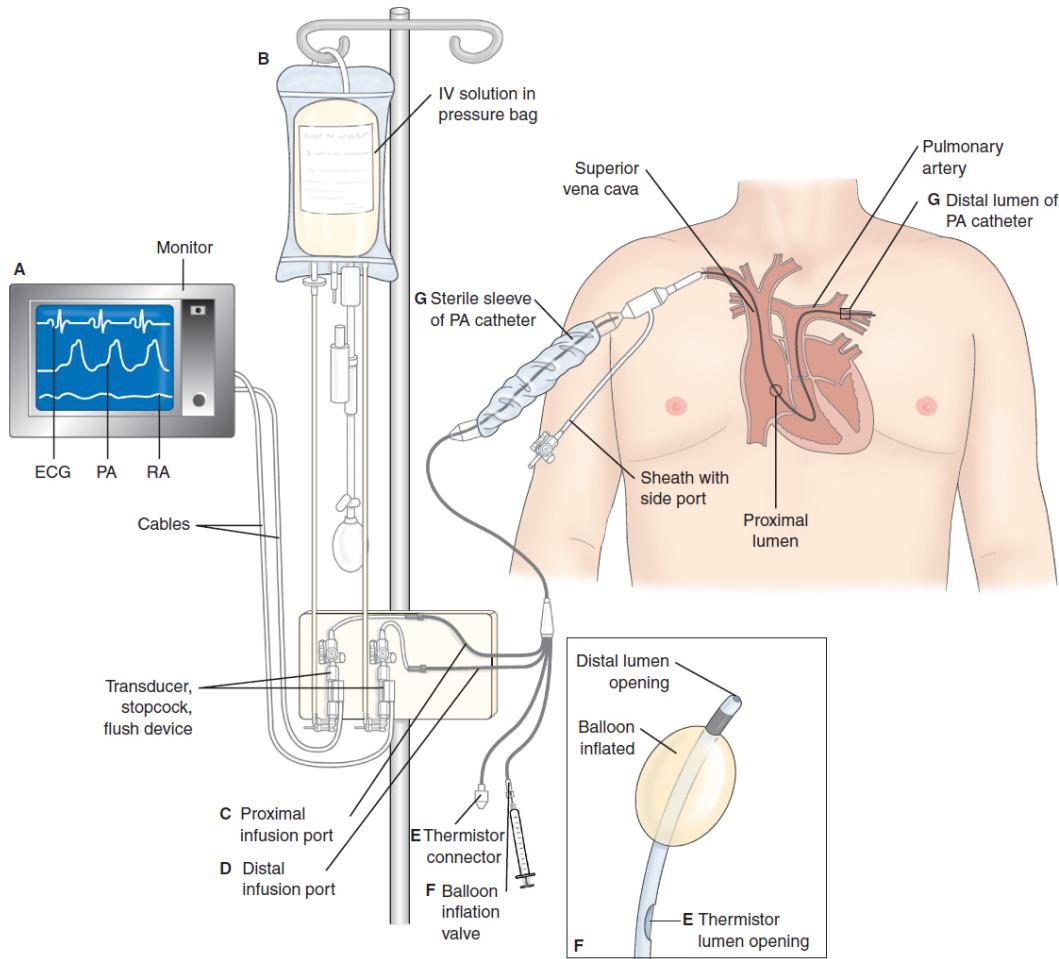
### **Nursing Interventions**

Catheter site care is essentially the same as for a CVP catheter. Similar to CVP measurement, the transducer must be positioned at the phlebostatic axis to ensure accurate readings (see Fig. 21-10). Serious complications include pulmonary artery rupture, pulmonary thromboembolism, pulmonary infarction, catheter kinking, arrhythmias, and air embolism.

### **Intra-Arterial Blood Pressure Monitoring**

Intra-arterial BP monitoring is used to obtain direct and continuous BP measurements in critically ill patients who have severe hypertension or hypotension. Arterial catheters are also useful when arterial blood gas measurements and blood samples need to be obtained frequently.

The radial artery is the usual site selected. However, placement of a catheter into the radial artery can further impede perfusion to an area that has poor circulation. As a result, the tissue distal to the cannulated artery can become ischemic or necrotic. Patients with diabetes, peripheral vascular disease, or hypotension, receiving IV vasopressors, or having had previous surgery are at highest risk for this complication. Before arterial line insertion, two tests may be considered to assess circulation; namely, a Doppler ultrasound or a modified Allen's test. A Doppler ultrasound assesses blood flow of the artery. The modified Allen's test assesses collateral circulation. To perform the Allen's test, the patient's hand is elevated and the patient is asked to make a fist. The nurse compresses the radial and ulnar arteries simultaneously, causing the hand to blanch. After the patient opens the fist, the nurse releases the pressure on the ulnar artery. If blood flow is restored (hand turns pink) within 7 seconds, the circulation to the hand may be adequate enough to tolerate placement of a radial artery catheter (Wiegand, 2017).



**Figure 21-12 •** Pulmonary artery (PA) catheter and pressure monitoring systems. Bedside monitor that connects with cables (**A**) to the pressure monitoring systems (includes intravenous [IV] solution in a pressure bag, IV tubing, and two transducers with stopcocks and flush devices) (**B**). This system connects to the proximal infusion port that opens in the right atria (**C**) and is used to infuse fluids or medications and monitor central venous pressures and the distal infusion port (**D**). This port opens in the PA and is used to monitor PA pressures. **E.** The thermistor connector is attached to the bedside cardiac monitor to obtain cardiac output. **F.** An air-filled syringe is attached to the balloon inflation valve during catheter insertion and measurement of PA wedge pressure. **G.** PA catheter positioned in the pulmonary artery. Note the sterile sleeve over the PA catheter. The PA catheter is threaded through the sheath until it reaches the desired position in the PA. The side port on the sheath is used to infuse medications or fluids. ECG, electrocardiogram; RA, right atrium.

## Nursing Interventions

Site preparation and care are the same as for CVP catheters. The catheter flush solution is normal saline, which is the same as for CVP and pulmonary artery catheters. A transducer is attached, and pressures are measured in millimeters of mercury (mm Hg). The nurse monitors the patient for complications, which include local obstruction with distal ischemia, external hemorrhage, massive ecchymosis, dissection, air embolism, blood loss, pain, arteriospasm, and infection.

### Minimally Invasive Cardiac Output Monitoring Devices

Monitoring cardiac output using the pulmonary artery catheter has been the standard of practice in critical care since its inception over 50 years ago. Its use has diminished recently with the availability of new, less invasive devices. Several types of devices are commercially available. Selection of a specific device for clinical use is determined by availability, provider preferences, and the patient's clinical condition (Urden et al., 2017).

Pulse pressure analysis uses an arterial pressure waveform to continuously estimate the patient's stroke volume. One such device, the Edwards Lifesciences Vigileo monitoring system, is connected to an existing radial or femoral arterial line via its FloTrac transducer. Using age, gender, body surface area, and BP of the patient, this device calculates continuous cardiac output and other parameters used in the management of critically ill patients. The major drawback to this device is that in order for it to capture accurate data, it must first capture optimal arterial waveforms. Therefore, this type of device has limited usefulness in patients with poor waveform signals, some arrhythmias, hemodynamic instability, and those who may be concomitantly using an intra-aortic balloon pump (see [Chapter 11](#)).

Esophageal Doppler probes are used to noninvasively estimate cardiac output. The esophageal probe measures blood flow velocity within a cross-sectional area of the descending aorta to calculate cardiac output. The use of this device in the perioperative setting has been shown to improve patient outcomes, including decreased lengths of hospital stay and an overall decrease in rates of complications (Urden et al., 2017).

The Fick method, which uses carbon dioxide ( $\text{CO}_2$ ) measures, is an additional method used to estimate cardiac output. To obtain cardiac output using this method, a rebreathing loop is attached to the ventilator along with an infrared  $\text{CO}_2$  sensor, an airflow sensor, and pulse oximeter. Continuous readings of cardiac output may be updated every 3 minutes with the use of this device. As an alternative, a Fick calculation may be done that uses the patient's body surface area and oxygen consumption (arterial and venous saturations and hemoglobin) to determine cardiac output.

### CRITICAL THINKING EXERCISES

**1 pq** You receive report on a 65-year-old male patient being transferred to the cardiac ICU from the cardiac catheterization lab. The nurse reports that the patient has a history of chest pain, ACS, MI, hypertension, and diabetes. An elective right and left heart catheterization was performed and three stents were placed in the coronary arteries. The patient received normal saline IV 250 mL, heparin 4500 units, fentanyl 100 mg, and midazolam 2 mg. A TR Band is in place over the right radial artery which must stay in place for 2 hours. The patient tolerated the procedure well. What are your top nursing priorities when the patient arrives to the unit? Discuss key nursing interventions after cardiac catheterization. Describe how the nursing care of a patient with radial access differs from a patient with femoral access. What education will you provide the patient concerning postprocedure care?

**2 ebp** You are assigned to care for a 48-year-old female patient with a pulmonary artery (PA) catheter. She has advanced heart failure and is on the transplant waiting list. What cardiac assessment findings do you expect to find? The charge nurse rounds and asks you if a care bundle has been implemented. What is a care bundle? How will you care for the pulmonary artery catheter to reduce the risk of catheter-related bloodstream infections? What is the strength of the evidence that guides these interventions?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

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- American Heart Association (AHA) (2018). Check. Change. Control Calculator<sup>TM</sup>, [cccalculator.ccctracker.com](http://cccalculator.ccctracker.com)
- National Institutes of Health (NIH), National Heart, Lung, and Blood Institute (NHLBI), [www.nhlbi.nih.gov](http://www.nhlbi.nih.gov)

# **22** Management of Patients with Arrhythmias and Conduction Problems

**LEARNING OUTCOMES**

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

1. Correlate the components of the normal electrocardiogram with physiologic events of the heart.
2. Define the electrocardiogram as a waveform that represents the cardiac electrical event in relation to the lead (placement of electrodes).
3. Analyze elements of an electrocardiographic rhythm strip: ventricular and atrial rate, ventricular and atrial rhythm, QRS complex and shape, QRS duration, P wave and shape, PR interval, and P:QRS ratio.
4. Identify the electrocardiographic criteria, causes and management of arrhythmias, and use the nursing process as a framework for care of the patient with an arrhythmia, including conduction disturbances.
5. Compare the different types of pacemakers, their uses, possible complications, and nursing implications.
6. Describe the key points of using a defibrillator; identify the purpose of an implantable cardioverter defibrillator, the types available, and the nursing implications.
7. Describe the nursing management of patients with implantable cardiac devices.

## NURSING CONCEPTS

Perfusion

## GLOSSARY

**ablation:** purposeful destruction of heart muscle cells, usually in an attempt to correct or eliminate an arrhythmia

**arrhythmia:** disorder of the formation or conduction (or both) of the electrical impulse within the heart, altering the heart rate, heart rhythm, or both and potentially causing altered blood flow (also referred to as dysrhythmia)

**artifact:** distorted, irrelevant, and extraneous electrocardiographic (ECG) waveforms

**automaticity:** ability of the cardiac cells to initiate an electrical impulse

**cardiac resynchronization therapy (CRT):** biventricular pacing used to correct interventricular, intraventricular, and atrioventricular conduction disturbances that occur in patients with heart failure

**cardioversion:** electrical current given in synchrony with the patient's own QRS complex to stop an arrhythmia

**chronotropy:** rate of impulse formation

**conduction:** transmission of electrical impulses from one cell to another

**defibrillation:** electrical current given to stop an arrhythmia, not synchronized with the patient's QRS complex

**depolarization:** process by which cardiac muscle cells change from a more negatively charged to a more positively charged intracellular state

**dromotropy:** conduction velocity

**electrocardiogram (ECG):** a record of a test that graphically measures the electrical activity of the heart, including each phase of the cardiac cycle

**implantable cardioverter defibrillator (ICD):** a device implanted into the chest wall to treat arrhythmias

**inotropy:** force of myocardial contraction

**P wave:** the part of an ECG that reflects conduction of an electrical impulse through the atrium; atrial depolarization

**paroxysmal:** arrhythmia that has a sudden onset and terminates spontaneously; usually of short duration, but may recur

**PP interval:** the duration between the beginning of one P wave and the beginning of the next P wave; used to calculate atrial rate and rhythm

**PR interval:** the part of an ECG that reflects conduction of an electrical impulse from the sinoatrial node through the atrioventricular node

**QRS complex:** the part of an ECG that reflects conduction of an electrical impulse through the ventricles; ventricular depolarization

**QT interval:** the part of an ECG that reflects the time from ventricular depolarization through repolarization

**repolarization:** process by which cardiac muscle cells return to a more negatively charged intracellular condition, their resting state

**RR interval:** the duration between the beginning of one QRS complex and the beginning of the next QRS complex; used to calculate ventricular rate and rhythm

**sinus rhythm:** electrical activity of the heart initiated by the sinoatrial node

**ST segment:** the part of an ECG that reflects the end of the QRS complex to the beginning of the T wave

**T wave:** the part of an ECG that reflects repolarization of the ventricles

**TP interval:** the part of an ECG that reflects the time between the end of the T wave and the beginning of the next P wave; used to identify the isoelectric line

**U wave:** the part of an ECG that may reflect Purkinje fiber repolarization; usually, it is not seen unless a patient's serum potassium level is low

It is essential for the heart to have a regular rate and rhythm to perform efficiently as a pump to circulate oxygenated blood and other life-sustaining nutrients to all of the body's tissues and organs (including the heart itself). With an irregular or erratic rhythm, the heart is considered to be arrhythmic (also called *dysrhythmic*). This is a potentially dangerous condition.

Nurses may encounter patients with arrhythmias in all health care settings, including primary care settings, skilled nursing facilities, rehabilitation settings, hospitals, and the home health care setting. Some arrhythmias are acute and others chronic; some require emergent interventions, while others may not. Because patients with arrhythmias are frequently encountered in many different types of settings, nurses must be able to identify and provide appropriate first-line treatment of arrhythmias.

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## ARRHYTHMIAS



**Arrhythmias** are disorders of the formation or conduction (or both) of the electrical impulse within the heart. These disorders can cause disturbances of the heart rate, the heart rhythm, or both. Arrhythmias may initially be evidenced by the hemodynamic effect they cause (e.g., a change in conduction may change the pumping action of the heart and cause decreased blood pressure), and are diagnosed by analyzing the electrocardiographic (ECG) waveform. Their treatment is based on the frequency and severity of symptoms produced. Arrhythmias are named according to the site of origin of the electrical impulse and the mechanism of formation or conduction involved.

For example, an impulse that originates in the sinoatrial (SA) node and at a slow rate is called *sinus bradycardia*.

## Normal Electrical Conduction

The electrical impulse that stimulates and paces the cardiac muscle normally originates in the SA node, also called the *sinus node*, an area located near the superior vena cava in the right atrium. In the adult, the electrical impulse usually occurs at a rate of 60 to 100 times a minute. The electrical impulse quickly travels from the SA node through the atria to the atrioventricular (AV) node (see Fig. 22-1); this process is known as **conduction**. The electrical stimulation of the muscle cells of the atria causes them to contract. The structure of the AV node slows the electrical impulse, giving the atria time to contract and fill the ventricles with blood. This part of atrial contraction is frequently referred to as the atrial kick and accounts for nearly one third of the volume ejected during ventricular contraction (Fuster, Harrington, Narula, et al., 2017). The electrical impulse then travels very quickly through the bundle of His to the right and left bundle branches and the Purkinje fibers, located in the ventricular muscle.

The electrical stimulation is called **depolarization**, and the mechanical contraction is called *systole*. Electrical relaxation is called **repolarization**, and mechanical relaxation is called *diasstole*. The process from sinus node electrical impulse generation through ventricular repolarization completes the electromechanical circuit, and the cycle begins again. (See Chapter 21 for a more complete explanation of cardiac function.)

## Influences on Heart Rate and Contractility

The heart rate is influenced by the autonomic nervous system, which consists of sympathetic and parasympathetic fibers. Sympathetic nerve fibers (also referred to as adrenergic fibers) are attached to the heart and arteries as well as several other areas in the body. Stimulation of the sympathetic system results in positive **chronotropy** (increased heart rate), positive **dromotropy** (increased AV conduction), and positive **inotropy** (increased force of myocardial contraction). Sympathetic stimulation also constricts peripheral blood vessels, therefore increasing blood pressure. Parasympathetic nerve fibers are also attached to the heart and arteries. Parasympathetic stimulation reduces the heart rate (negative chronotropy), AV conduction (negative dromotropy), and the force of atrial myocardial contraction. The decreased sympathetic stimulation results in dilation of arteries, thereby lowering blood pressure.

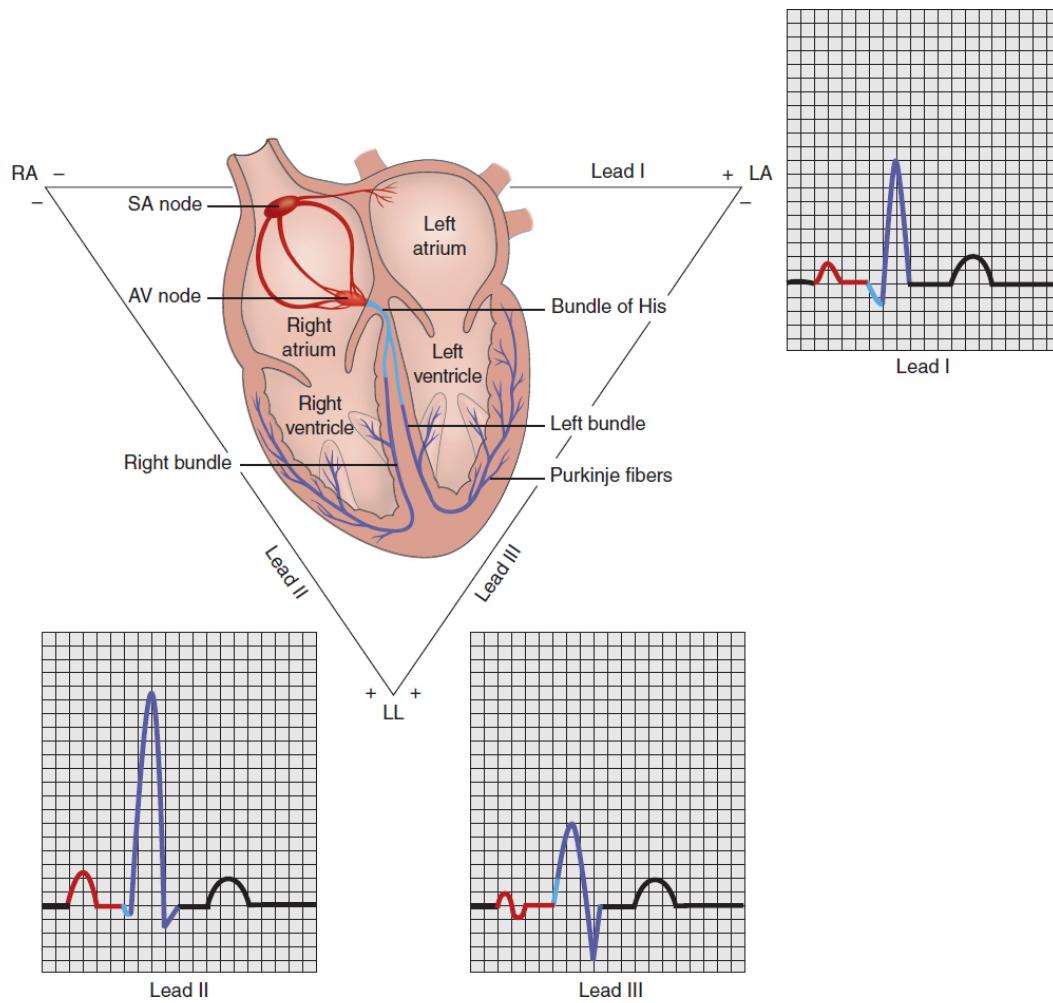
Manipulation of the autonomic nervous system may increase or decrease the incidence of arrhythmias. Increased sympathetic stimulation (e.g., caused by exercise, anxiety, fever, or administration of catecholamines such as dopamine, aminophylline, or dobutamine) may increase the incidence of arrhythmias. Decreased sympathetic stimulation (e.g., with rest, anxiety reduction methods such as therapeutic communication or meditation, or administration of beta-adrenergic blocking agents) may decrease the incidence of arrhythmias.

## The Electrocardiogram

The electrical impulse that travels through the heart can be viewed by means of electrocardiography, the end product of which is an **electrocardiogram (ECG)**. Each phase of the cardiac cycle is reflected by specific waveforms on the screen of a cardiac monitor or on a strip of ECG graph paper.

## Obtaining an Electrocardiogram

An ECG is obtained by placing electrodes on the body at specific areas. Biomonitoring electrodes come in various shapes and sizes, but all have two components: (1) an adhesive substance that attaches to the skin to secure the electrode in place and (2) a substance that reduces the skin's electrical impedance, facilitating the transfer of ions from the tissue to electrons in the electrode, enhancing conductivity. Gently abrading the skin with a clean dry gauze pad or sandpaper edge of the electrode helps to expose the inner conductive layer of epidermis, which will reduce skin impedance. Although cleansing the skin with alcohol removes any oily residue from the skin, it also increases the skin's electrical impedance and hinders detection of the cardiac electrical signal. Washing the area with soap and water prior to electrode placement is recommended. If the amount of chest hair prevents the electrode from having good contact with the skin, the hair may need to be clipped (Sendelbach & Jepsen, 2018; see Chapter 21, [Chart 21-5: Applying Electrodes](#)). Poor electrode adhesion will cause significant **artifact** (distorted, irrelevant, and extraneous ECG waveforms), which may distort capturing an accurate ECG waveform.



**Figure 22-1 •** Relationship of electrocardiographic (ECG) complex, lead system, and electrical impulse. The heart conducts electrical activity, which the ECG measures and shows. The configurations of electrical activity displayed on the ECG vary depending on the lead (or view) of the ECG and on the rhythm of the heart. Therefore, the configuration of a normal rhythm tracing from lead I will differ from the configuration of a normal rhythm tracing from lead II, lead II will differ from lead III, and so on. The same is true for abnormal rhythms and cardiac disorders. To make an accurate assessment of the heart's electrical activity or to identify where, when, and what abnormalities occur, the ECG needs to be evaluated from every lead, not just from lead II. Here the different areas of electrical activity are identified by color. RA, right arm; LA, left arm; SA, sinoatrial; AV, atrioventricular; LL, left leg.

The number and placement site of the electrodes depend on the type of ECG being obtained. Most continuous monitors use 2 to 5 electrodes, usually placed on the limbs and the chest. These electrodes create an imaginary line, called a *lead*, which serves as a reference point from which the electrical

activity is viewed. A lead is like an eye of a camera—it has a narrow peripheral field of vision, looking only at the electrical activity directly in front of it. Therefore, the ECG waveforms that appear on the ECG paper and cardiac monitor represent the electrical impulse in relation to the lead (see [Fig. 22-1](#)). A change in the waveform can be caused by a change in the electrical impulse (where it originates or how it is conducted) or by a change in the lead. Electrodes are attached to cable wires, which are connected to one of the following:

- An ECG machine placed at the patient's side for an immediate recording (standard 12-lead ECG)
- A cardiac monitor at the patient's bedside for continuous reading; this kind of monitoring, usually called *hardwire monitoring*, is used in intensive care units
- A small box that the patient carries and that continuously transmits the ECG information by radiowaves to a central monitor located elsewhere (called *telemetry*)
- A small, lightweight tape recorder-like machine (called *continuous ECG monitoring*, which might include a *Holter monitor* or a *patch monitor*) that the patient wears for a prescribed period of time and that continuously records the ECG, which is later viewed and analyzed with a scanner
- A very small device inserted under the skin or worn externally on a wrist band (called *intermittent monitoring* using a *looped recorder*) can perform ECG monitoring on demand whenever a patient is symptomatic (see [Chapter 21](#) for further discussion of ECG monitoring systems)

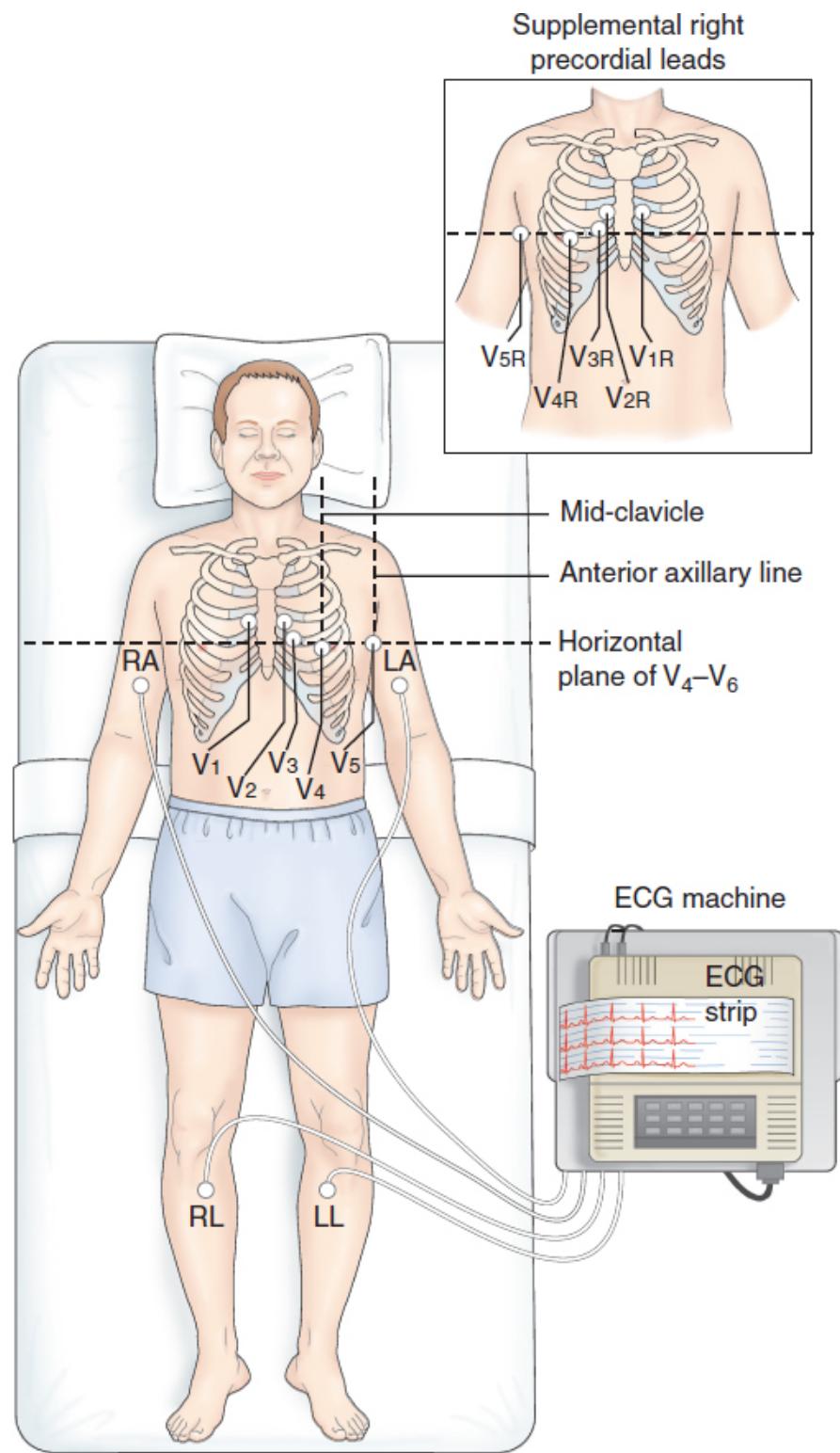
A patient may undergo an electrophysiology study (EPS) in which electrodes are placed inside the heart in order to obtain an intracardiac ECG. This is used not only to diagnose the arrhythmia but also to determine the most effective treatment plan. However, because an EPS is invasive, it is performed in the hospital and may require that the patient be admitted (see later discussion).

During open heart surgery, temporary pacemaker wires may be lightly sutured to the epicardium and brought through the chest wall. These wires may be used not only for temporary pacing but also, when connected to the V lead cable, to obtain an atrial ECG, which can be helpful in the differential diagnosis of tachyarrhythmias (see [Chapter 23](#) for further discussion).

The placement of electrodes for monitoring varies with the type of technology, the purpose of monitoring, and the protocols used in the health care facility. For a standard 12-lead ECG, 10 electrodes (6 on the chest and 4 on the limbs) are placed on the body (see [Fig. 22-2](#)). To prevent interference from the electrical activity of skeletal muscle, the limb electrodes are placed

on areas that are not bony and that do not have significant movement. The limb electrodes provide the first six leads: leads I, II, III, aVR (augmented voltage right arm), aVL (augmented voltage left arm), and aVF (augmented voltage left leg/foot). The 6 chest electrodes are applied to the chest at very specific areas. The chest electrodes provide the V or precordial leads, V<sub>1</sub> through V<sub>6</sub>. To locate the fourth intercostal space and the placement of V<sub>1</sub>, the sternal angle and then the sternal notch, which is about 1 or 2 inches below the sternal angle, are located. When the fingers are moved to the patient's immediate right, the second rib can be palpated. The second intercostal space is the indentation felt just below the second rib. Locating the specific intercostal space is critical for the correct placement of each chest electrode. Errors in diagnosis can occur if electrodes are incorrectly placed. Sometimes, when a patient in the hospital needs to be monitored more closely for ECG changes, the chest electrodes are left in place to ensure the same placement for follow-up 12-lead ECGs.

A standard 12-lead ECG reflects the electrical activity primarily in the left ventricle. Placement of additional electrodes for other leads may be needed to obtain more complete information. For example, in patients with suspected right-sided heart damage, right-sided precordial leads are required to evaluate the right ventricle (see [Fig. 22-2](#)).



**Figure 22-2 •** ECG electrode placement. The standard left precordial leads are V<sub>1</sub>—fourth intercostal space, right sternal border; V<sub>2</sub>—fourth intercostal space, left sternal border; V<sub>3</sub>—diagonally between V<sub>2</sub> and V<sub>4</sub>; V<sub>4</sub>—fifth intercostal space, left

midclavicular line; V<sub>5</sub>—same level as V<sub>4</sub>, anterior axillary line; V<sub>6</sub> (not illustrated)—same level as V<sub>4</sub> and V<sub>5</sub>, midaxillary line. The right precordial leads, placed across the right side of the chest, are the mirror opposite of the left leads. RA, right arm; LA, left arm; RL, right leg; LL, left leg.

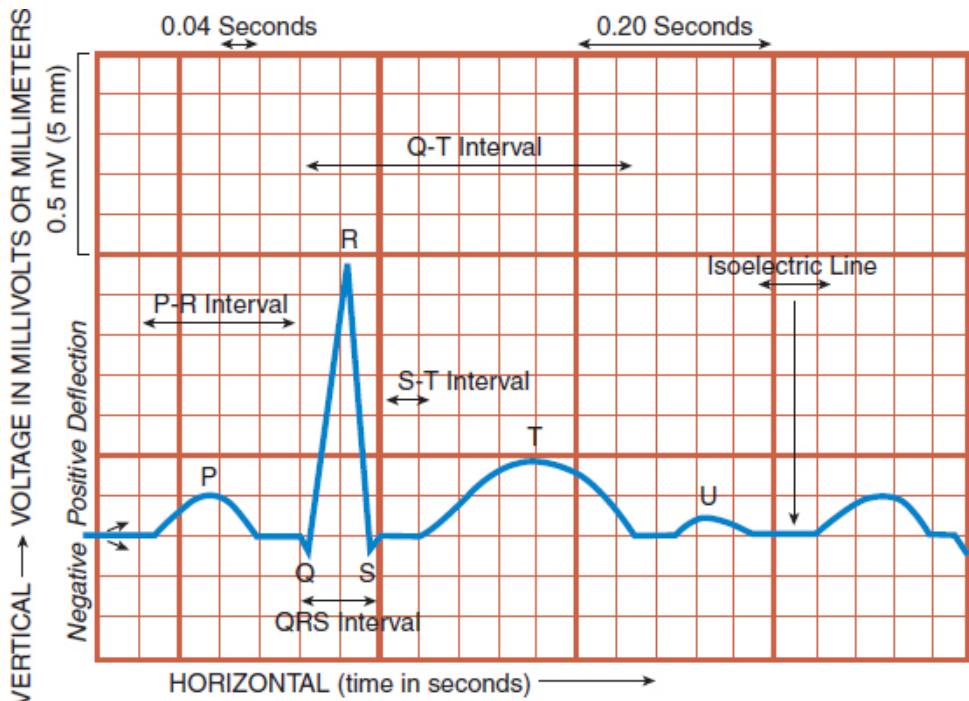
## Components of the Electrocardiogram

The ECG waveform reflects the function of the heart's conduction system in relation to the specific lead. The ECG offers important information about the electrical activity of the heart and is useful in diagnosing arrhythmias. ECG waveforms are printed on graph paper that is divided by vertical and horizontal lines at standard intervals (see Fig. 22-3). Time and rate are measured on the horizontal axis of the graph, and amplitude or voltage is measured on the vertical axis. When an ECG waveform moves toward the top of the paper, it is called a *positive deflection*. When it moves toward the bottom of the paper, it is called a *negative deflection*. When reviewing an ECG, each waveform should be examined and compared with the others.

### Waves, Complexes, and Intervals

The ECG is composed of waveforms (including the P wave, the QRS complex, the T wave, and possibly a U wave) and of segments and intervals (including the PR interval, the ST segment, and the QT interval) (see Fig. 22-3).

The **P wave** represents the electrical impulse starting in the SA node and spreading through the atria. Therefore, the P wave represents atrial depolarization. It is normally 2.5 mm or less in height and 0.11 seconds or less in duration.



**Figure 22-3 •** ECG graph and commonly measured components. Each large box represents 0.20 seconds on the horizontal axis and 5 mm or 0.5 millivolt on the vertical axis. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-16). Philadelphia, PA: Wolters Kluwer.

The **QRS complex** represents ventricular depolarization. Not all QRS complexes have all three waveforms. The Q wave is the first negative deflection after the P wave. The Q wave is normally less than 0.04 seconds in duration and less than 25% of the R-wave amplitude. The R wave is the first positive deflection after the P wave, and the S wave is the first negative deflection after the R wave. When a wave is less than 5 mm in height, small letters (q, r, s) are used; when a wave is taller than 5 mm, capital letters (Q, R, S) are used to label the waves. The QRS complex is normally less than 0.12 seconds in duration.

The **T wave** represents ventricular repolarization (when the cells regain a negative charge; also called the *resting state*). It follows the QRS complex and is usually the same direction (deflection) as the QRS complex. Atrial repolarization also occurs but is not visible on the ECG because it occurs at the same time as ventricular depolarization (i.e., the QRS).

The **U wave** is thought to represent repolarization of the Purkinje fibers; although this wave is rare, it sometimes appears in patients with hypokalemia (low potassium levels), hypertension, or heart disease. If present, the U wave

follows the T wave and is usually smaller than the P wave. If larger in amplitude, it may be mistaken for an extra P wave.

The **PR interval** is measured from the beginning of the P wave to the beginning of the QRS complex and represents the time needed for sinus node stimulation, atrial depolarization, and conduction through the AV node before ventricular depolarization. In adults, the PR interval normally ranges from 0.12 to 0.20 seconds in duration.

The **ST segment**, which represents early ventricular repolarization, lasts from the end of the QRS complex to the beginning of the T wave. The beginning of the ST segment is usually identified by a change in the thickness or angle of the terminal portion of the QRS complex. The end of the ST segment may be more difficult to identify because it merges into the T wave. The ST segment is normally isoelectric (see later discussion of TP interval). It is analyzed to identify whether it is above or below the isoelectric line, which may be, among other signs and symptoms, a sign of cardiac ischemia (see [Chapter 23](#)).

The **QT interval**, which represents the total time for ventricular depolarization and repolarization, is measured from the beginning of the QRS complex to the end of the T wave. The QT interval varies with heart rate, gender, and age; therefore, the measured interval may be corrected ( $QT_c$ ) for these variables through specific calculations. The  $QT_c$  may be automatically calculated by the ECG technology, or a nurse may manually calculate or use a resource that contains a chart of these calculations. The QT interval is usually 0.32 to 0.40 seconds in duration if the heart rate is 65 to 95 bpm. Many medications commonly given in the hospital can cause prolongation of the QT interval ( $QT_c$ ), placing the patient at risk for a lethal ventricular arrhythmia called *torsades de pointes*.

The **TP interval** is measured from the end of the T wave to the beginning of the next P wave—an isoelectric period (see [Fig. 22-3](#)). When no electrical activity is detected, the line on the graph remains flat; this is called the *isoelectric line*. The ST segment is compared with the TP interval to detect ST segment changes. The PR segment is sometimes used to determine the isoelectric line. However, because the PR segment sometimes is altered due to ischemic conditions, the TP interval is the preferred reference for the isoelectric line.

The **PP interval** is measured from the beginning of one P wave to the beginning of the next P wave. The PP interval is used to determine atrial rate and rhythm. The **RR interval** is measured from one QRS complex to the next QRS complex. The RR interval is used to determine ventricular rate and rhythm (see later discussion).

## Analyzing the Electrocardiogram Rhythm Strip

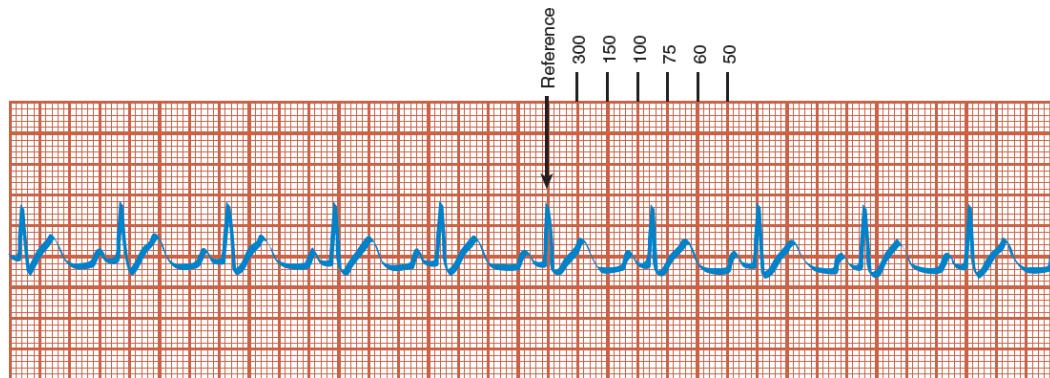
The ECG rhythm strip must be analyzed in a systematic manner to determine the patient's cardiac rate and rhythm, and to detect arrhythmias and conduction disorders, as well as evidence of myocardial ischemia, injury, and infarction.

### Determining Heart Rate from the Electrocardiogram

Heart rate can be obtained from the ECG rhythm strip by several methods. A 1-minute rhythm strip contains 300 large boxes and 1500 small boxes. Therefore, an easy and accurate method of determining heart rate with a regular rhythm is to count the number of small boxes within an RR interval and divide 1500 by that number. If, for example, there are 10 small boxes between two R waves, the heart rate is  $1500/10$ , or 150 bpm; if there are 25 small boxes, the heart rate is  $1500/25$ , or 60 bpm (see Fig. 22-4).

An alternative but less accurate method for estimating heart rate, which is usually used when the rhythm is irregular, is to count the number of RR intervals in 6 seconds and multiply that number by 10. The top of the ECG paper is usually marked at 3-second intervals, which is 15 large boxes horizontally. The RR intervals are counted, rather than QRS complexes, because a computed heart rate based on the latter might be inaccurately high.

The same methods may be used for determining atrial rate, using the PP interval instead of the RR interval.



**Figure 22-4 •** Method for estimating heart rate. The number of small boxes in the RR interval is 17.5; divide this by 1500, and the heart rate is approximately 86 bpm. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-18 (bottom portion)). Philadelphia, PA: Wolters Kluwer.

### Determining Heart Rhythm from the Electrocardiogram

The rhythm is often identified at the same time the rate is determined. **Chart 22-1** provides an example of a method that can be used to analyze the patient's rhythm. The RR interval is used to determine ventricular rhythm and the PP interval to determine atrial rhythm. If the intervals are the same or if the difference between the intervals is less than 0.8 seconds throughout the strip, the rhythm is called *regular*. If the intervals are different, the rhythm is called *irregular*.

Once the rhythm has been analyzed, the findings are compared with and matched to the ECG criteria for arrhythmias to determine a diagnosis. It is important for the nurse not only to identify the arrhythmia, but also to assess the patient to determine the physiologic effect of the arrhythmia and identify possible causes. Treatment of an arrhythmia is based on clinical evaluation of the patient with identification of the arrhythmia's etiology and physiologic effect, not on its presence on ECG alone.

## Chart 22-1

### Interpreting Arrhythmias: Systematic Analysis of the Electrocardiogram

When examining an electrocardiogram (ECG) rhythm strip to learn more about a patient's arrhythmia:

1. Determine the ventricular rate.
2. Determine the ventricular rhythm.
3. Determine the QRS duration.
4. Determine whether the QRS duration is consistent throughout the strip. If not, identify other duration.
5. Identify the QRS shape; if not consistent, then identify other shapes.
6. Identify P waves; is there a P in front of every QRS?
7. Identify the P-wave shape; identify whether it is consistent or not.
8. Determine the atrial rate.
9. Determine the atrial rhythm.
10. Determine each PR interval.
11. Determine if the PR intervals are consistent, irregular but with a pattern to the irregularity, or just irregular.
12. Determine how many P waves for each QRS (P:QRS ratio).

In many cases, the nurse may use a checklist and document the findings next to the appropriate ECG criterion.

Most cardiac monitoring has functionality that includes the ability to continuously monitor the rhythm and alert health care personnel with an auditory and visual alarm when a clinically significant change in the rhythm occurs. However, a high rate of triggered, clinically insignificant alarms may lead to alarm fatigue, which has been linked to nurses ignoring, disabling, or

silencing alarms (Sendelbach & Jepsen, 2018)—putting patients at increased risk of adverse events.



#### **Quality and Safety Nursing Alert**

*It is vital that the nurse assesses the cause(s) of a cardiac monitor's alarm and then adjusts the alarm default settings and individualizes the alarm parameter limits and levels. The assessment should also include an evaluation and discussion with the primary provider to validate that the patient needs to remain on continuous cardiac monitoring.*

## **Normal Sinus Rhythm**

Electrical conduction that begins in the SA node generates a **sinus rhythm**. Normal sinus rhythm occurs when the electrical impulse starts at a regular rate and rhythm in the SA node and travels through the normal conduction pathway. Normal sinus rhythm has the following characteristics (see Fig. 22-5):

*Ventricular and atrial rate:* 60 to 100 bpm in the adult

*Ventricular and atrial rhythm:* Regular

*QRS shape and duration:* Usually normal, but may be regularly abnormal

*P wave:* Normal and consistent shape; always in front of the QRS

*PR interval:* Consistent interval between 0.12 and 0.20 seconds

*P:QRS ratio:* 1:1

Normal sinus rhythm is generally indicative of good cardiovascular health. However, an increase of 10 bpm or more in the resting heart rate increases the risk for sudden cardiac death, atrial fibrillation, heart failure, coronary artery disease, stroke, and cardiovascular disease (Aune, Sen, ó'Hartaigh, et al., 2017).



**Figure 22-5 •** Normal sinus rhythm. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-19A). Philadelphia, PA: Wolters Kluwer.



## Types of Arrhythmias

Arrhythmias include sinus, atrial, junctional, and ventricular arrhythmias and their various subcategories, as well as conduction abnormalities.

### Sinus Node Arrhythmias

Sinus node arrhythmias originate in the SA node; these include sinus bradycardia, sinus tachycardia, and sinus arrhythmia.

#### Sinus Bradycardia

Sinus bradycardia occurs when the SA node creates an impulse at a slower-than-normal rate. Causes include lower metabolic needs (e.g., sleep, athletic training, hypothyroidism), vagal stimulation (e.g., from vomiting, suctioning, severe pain), medications (e.g., calcium channel blockers [e.g., nifedipine, amiodarone], beta-blockers [e.g., metoprolol]), idiopathic sinus node dysfunction, increased intracranial pressure, and coronary artery disease, especially myocardial infarction (MI) of the inferior wall. Unstable and symptomatic bradycardia is frequently due to hypoxemia. Other possible causes include acute altered mental status (e.g., delirium) and acute decompensated heart failure (Fuster et al., 2017). Sinus bradycardia has the following characteristics (see Fig. 22-6):



**Figure 22-6 •** Sinus bradycardia. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-19C). Philadelphia, PA: Wolters Kluwer.

*Ventricular and atrial rate:* Less than 60 bpm in the adult

*Ventricular and atrial rhythm:* Regular

*QRS shape and duration:* Usually normal, but may be regularly abnormal

*P wave:* Normal and consistent shape; always in front of the QRS

*PR interval:* Consistent interval between 0.12 and 0.20 seconds

*P:QRS ratio:* 1:1

All characteristics of sinus bradycardia are the same as those of normal sinus rhythm, except for the rate. The patient is assessed to determine the hemodynamic effect and the possible cause of the arrhythmia. If the decrease in heart rate results from stimulation of the vagus nerve, such as with bearing down during defecation or vomiting, attempts are made to prevent further vagal stimulation. If the bradycardia is caused by a medication such as a beta-blocker, the medication may be withheld. If the slow heart rate causes significant hemodynamic changes resulting in shortness of breath, acute alteration of mental status, angina, hypotension, ST-segment changes, or premature ventricular complexes (PVCs), treatment is directed toward increasing the heart rate. Slow heart rate may be due to sinus node dysfunction (previously known as *sick sinus syndrome*), which has a number of risk factors including increased body mass index, presence of right and left bundle branch block, history of a major cardiovascular event, increased age, and hypertension (Jackson, Rathakrishnan, Campbell, et al., 2017). *Tachy-brady syndrome* is the term used when bradycardia alternates with tachycardia.

### Medical Management

Management depends on the cause and symptoms. Resolving the causative factors may be the only treatment needed. If the bradycardia produces signs and symptoms of clinical instability (e.g., acute alteration in mental status, chest discomfort, or hypotension), 0.5 mg of atropine may be given rapidly as

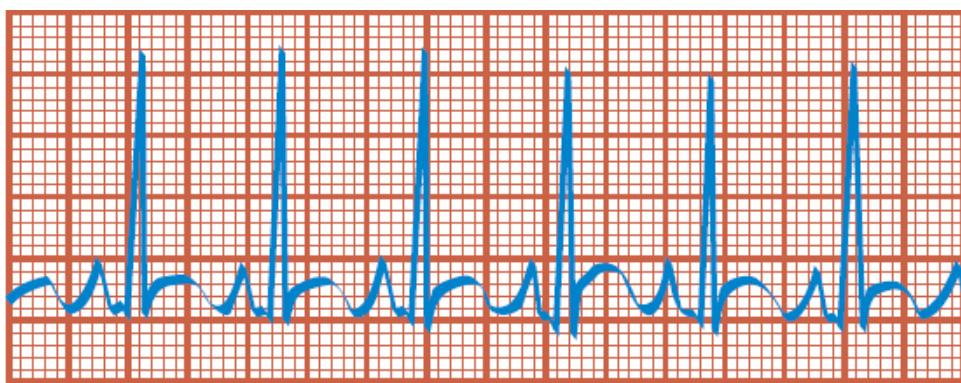
an intravenous (IV) bolus and repeated every 3 to 5 minutes until a maximum dosage of 3 mg is given. Rarely, if the bradycardia is unresponsive to atropine, emergency transcutaneous pacing can be instituted, or medications, such as dopamine, isoproterenol, or epinephrine, are given (Kusumoto, Schoenfeld, Barrett, et al., 2019; see later discussion).

### Sinus Tachycardia

Sinus tachycardia occurs when the sinus node creates an impulse at a faster-than-normal rate. Causes may include the following:

- Physiologic or psychological stress (e.g., acute blood loss, anemia, shock, hypervolemia, hypovolemia, heart failure, pain, hypermetabolic states, fever, exercise, anxiety)
- Medications that stimulate the sympathetic response (e.g., catecholamines, aminophylline, atropine), stimulants (e.g., caffeine, nicotine), and illicit drugs (e.g., amphetamines, cocaine, ecstasy)
- Enhanced automaticity of the SA node and/or excessive sympathetic tone with reduced parasympathetic tone that is out of proportion to physiologic demands, a condition called *inappropriate sinus tachycardia*
- Autonomic dysfunction, which results in a type of sinus tachycardia referred to as postural orthostatic tachycardia syndrome (POTS). POTS is characterized by tachycardia without hypotension, and by presyncopal symptoms such as palpitations, lightheadedness, weakness, and blurred vision, which occur with sudden posture changes

Sinus tachycardia has the following characteristics (see Fig. 22-7):



**Figure 22-7 •** Sinus tachycardia. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-19B). Philadelphia, PA: Wolters Kluwer.

*Ventricular and atrial rate:* Greater than 100 bpm in the adult, but usually less than 120 bpm

*Ventricular and atrial rhythm:* Regular

*QRS shape and duration:* Usually normal, but may be regularly abnormal

*P wave:* Normal and consistent shape; always in front of the QRS, but may be buried in the preceding T wave

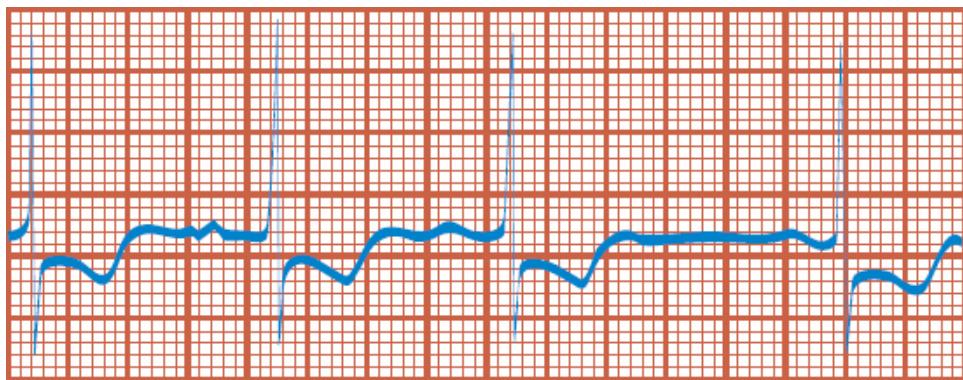
*PR interval:* Consistent interval between 0.12 and 0.20 seconds

*P:QRS ratio:* 1:1

All aspects of sinus tachycardia are the same as those of normal sinus rhythm, except for the rate. Sinus tachycardia does not start or end suddenly (i.e., it is nonparoxysmal). As the heart rate increases, the diastolic filling time decreases, possibly resulting in reduced cardiac output and subsequent symptoms of syncope (fainting) and low blood pressure. If the rapid rate persists and the heart cannot compensate for the decreased ventricular filling, the patient may develop acute pulmonary edema.

## Medical Management

Medical management of sinus tachycardia is determined by the severity of symptoms and directed at identifying and abolishing its cause. Vagal maneuvers, such as carotid sinus massage, gagging, bearing down against a closed glottis (as if having a bowel movement), forceful and sustained coughing, and applying a cold stimulus to the face (such as applying an ice-cold wet towel to the face), or administration of adenosine should be considered to interrupt the tachycardia. If the tachycardia is persistent and causing hemodynamic instability (e.g., acute alteration in mental status, chest discomfort, hypotension), synchronized **cardioversion** (i.e., electrical current given in synchrony with the patient's own QRS complex to stop an arrhythmia) is the treatment of choice, if vagal maneuvers and adenosine are unsuccessful or not feasible (see later discussion). IV beta-blockers (Class II antiarrhythmic) and calcium channel blockers (Class IV antiarrhythmic) (see [Table 22-1](#)) may also be considered in treating hemodynamically stable sinus tachycardia, although synchronized cardioversion may be used if medications are ineffective or contradicted (Page, Joglar, Caldwell, et al., 2016). Catheter ablation (see later discussion) of the SA node may be used in cases of persistent inappropriate sinus tachycardia unresponsive to other treatments. Treatment for POTS often involves a combination of approaches, with treatment targeted at the underlying problem. For example, patients with hypovolemia may be advised to increase their fluid and sodium intake, or use salt tablets if necessary.



**Figure 22-8 •** Sinus arrhythmia. Note irregular RR and PP intervals. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-19D). Philadelphia, PA: Wolters Kluwer.

## Sinus Arrhythmia

Sinus arrhythmia occurs when the sinus node creates an impulse at an irregular rhythm; the rate usually increases with inspiration and decreases with expiration. Nonrespiratory causes include heart disease and valvular disease, but these are rare. Sinus arrhythmia has the following characteristics (see Fig. 22-8):

*Ventricular and atrial rate:* 60 to 100 bpm in the adult

*Ventricular and atrial rhythm:* Irregular

*QRS shape and duration:* Usually normal, but may be regularly abnormal

*P wave:* Normal and consistent shape; always in front of the QRS

*PR interval:* Consistent interval between 0.12 and 0.20 seconds

*P:QRS ratio:* 1:1

## Medical Management

Sinus arrhythmia does not cause any significant hemodynamic effect and therefore is not typically treated.

## Atrial Arrhythmias

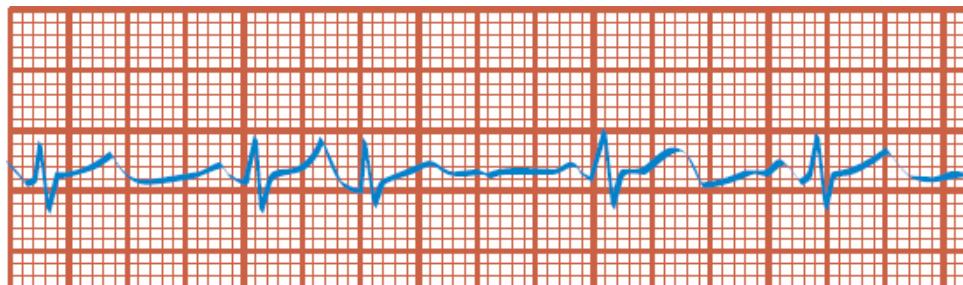
Atrial arrhythmias originate from foci within the atria and not the SA node. These include aberrancies such as premature atrial complexes (PACs) as well as atrial fibrillation and atrial flutter.

### Premature Atrial Complex

A PAC is a single ECG complex that occurs when an electrical impulse starts in the atrium before the next normal impulse of the sinus node. The PAC may be caused by caffeine, alcohol, nicotine, stretched atrial myocardium (e.g., as

in hypervolemia), anxiety, hypokalemia (low potassium level), hypermetabolic states (e.g., with pregnancy), or atrial ischemia, injury, or infarction. PACs are often seen with sinus tachycardia. PACs have the following characteristics (see Fig. 22-9):

*Ventricular and atrial rate:* Depends on the underlying rhythm (e.g., sinus tachycardia)



**Figure 22-9 •** Premature atrial complex (PAC). Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-22A). Philadelphia, PA: Wolters Kluwer.

TABLE 22-1

Summary of Antiarrhythmic Medications<sup>a</sup>

Class <sup>b</sup>	Action	Drug Names	Side Effects	Nursing Interventions
IA	Moderate depression of depolarization; prolongs repolarization Treats and prevents atrial and ventricular arrhythmias	quinidine, procainamide, disopyramide	Decreased cardiac contractility Prolonged QRS, QT Proarrhythmic Hypotension with IV administration Diarrhea with quinidine, constipation with disopyramide Cinchonism with quinidine Lupuslike syndrome with procainamide Anticholinergic effects: dry mouth, urinary hesitancy with disopyramide	Observe for HF. Monitor BP with IV administration. Monitor QRS duration for increase >50% from baseline. Monitor for prolonged QT. Monitor N-acetyl procainamide (NAPA) laboratory values during procainamide therapy. If given for atrial fibrillation, ensure that the patient has been pretreated with a medication to control AV conduction.
IB	Minimal depression of depolarization; shortened repolarization Treats ventricular arrhythmias	lidocaine, mexiletine	CNS changes (e.g., confusion, lethargy) Bradycardia GI distress Tremors	Monitor for CNS changes and tremors. Discuss with the primary provider decreasing lidocaine dose in older adult patients and patients with cardiac/liver dysfunction.
IC	Marked depression of depolarization; little effect on repolarization Treats atrial and ventricular arrhythmias	flecainide, propafenone	Proarrhythmic HF Dizziness, visual disturbances, dyspnea	Decrease dose with renal dysfunction and strict vegetarian diets. Avoid use in patients with structural heart disease (e.g., coronary artery disease and heart failure).
II	Decreases automaticity and conduction Treats atrial and ventricular arrhythmias	acebutolol <sup>c</sup> , atenolol, bisoprolol/HCTZ, esmolol <sup>c</sup> , labetalol, metoprolol, nadolol, propranolol <sup>c</sup> , sotalol (also has class III actions) <sup>c</sup> , timolol	Bradycardia, AV block Decreased contractility Bronchospasm Nausea Asymptomatic and symptomatic hypotension Masks hypoglycemia and thyrotoxicosis CNS disturbances (e.g., confusion, dizziness, fatigue, depression)	Monitor heart rate, PR interval, signs and symptoms of HF, especially in those also taking calcium channel blockers. Monitor blood glucose level in patients with type 2 diabetes. Caution the patient about abrupt withdrawal to avoid tachycardia, hypertension, and myocardial ischemia.
III	Prolongs repolarization Amiodarone treats and prevents ventricular and atrial arrhythmias, especially in patients with ventricular dysfunction. Dofetilide and ibutilide treat and prevent atrial arrhythmias.	amiodarone, dofetilide, dronedarone, ibutilide	Pulmonary toxicity (amiodarone) Corneal microdeposits (amiodarone) Photosensitivity (amiodarone) Bradycardia Hypotension, especially with IV administration Polymorphic ventricular arrhythmias (rare with amiodarone) Nausea and vomiting Potentiates digoxin (amiodarone) See beta-blockers above (sotalol).	Make sure that the patient is sent for baseline pulmonary function tests (amiodarone). Closely monitor the patient. Assess for contraindications prior to administration. Monitor QT duration. Continuous ECG monitoring with initiation of dofetilide and ibutilide. Monitor renal function.
IV	Blocks calcium channel Treats and prevents paroxysmal atrial arrhythmias <sup>b</sup>	verapamil, diltiazem	Bradycardia, AV blocks Hypotension with IV administration HF, peripheral edema Constipation, dizziness, headache, nausea	Monitor heart rate, PR interval. Monitor blood pressure closely with IV administration. Monitor for signs and symptoms of HF. Do not crush sustained-release medications.

<sup>a</sup>Based on Vaughan-Williams classification.<sup>b</sup>There are other calcium channel blockers, but they are not approved or used for arrhythmias.<sup>c</sup>Beta-blocker with labeled use for arrhythmias.

AV, atrioventricular; BP, blood pressure; CNS, central nervous system; ECG, electrocardiogram; GI, gastrointestinal; HCTZ, hydrochlorothiazide; HF, heart failure; IV, intravenous.

Adapted from the American Society of Health System Pharmacists. (2019). *AHFS drug information*. Bethesda, MD: Author; Fuster, V., Harrington, R. A., Narula, J., et al. (Eds.). (2017). *Hurst's the heart* (13th ed.). New York: McGraw-Hill; Al-Khatib, S. M., Stevenson, W. G., Ackerman, M. J., et al. (2018). 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines and the Heart Rhythm Society. *Circulation*, 138(13), e272–e391.

**Ventricular and atrial rhythm:** Irregular due to early P waves, creating a PP interval that is shorter than the others. This is sometimes followed by a longer-than-normal PP interval, but one that is less than twice the normal PP interval. This type of interval is called a *noncompensatory pause*.

**QRS shape and duration:** The QRS that follows the early P wave is usually normal, but it may be abnormal (aberrantly conducted PAC). It may even be absent (blocked PAC).

*P wave:* An early and different P wave may be seen or may be hidden in the T wave; other P waves in the strip are consistent

*PR interval:* The early P wave has a shorter-than-normal PR interval, but still between 0.12 and 0.20 seconds

*P:QRS ratio:* Usually 1:1

PACs are common in normal hearts. The patient may say, “My heart skipped a beat.” A pulse deficit (a difference between the apical and radial pulse rate) may exist.

## Medical Management

If PACs are infrequent; no treatment is necessary. If they are frequent (more than six per minute), this may herald a worsening disease state or the onset of more serious arrhythmias, such as atrial fibrillation. Medical management is directed toward treating the underlying cause (e.g., reduction of caffeine intake, correction of hypokalemia).

## Atrial Fibrillation

Atrial fibrillation is a very common arrhythmia; between 2.7 and 6.1 million Americans are living with atrial fibrillation (Centers for Disease Control and Prevention [CDC], 2017). Atrial fibrillation is a serious public health concern because it is associated with aging, and the older adult population is increasing in the United States (Chen, Chung, Allen, et al., 2018). Atrial fibrillation can result from diverse pathophysiologic etiologies and risks (see [Chart 22-2](#)).

Atrial fibrillation results from abnormal impulse formation that occurs when structural or electrophysiologic abnormalities alter the atrial tissue causing a rapid, disorganized, and uncoordinated twitching of the atrial musculature (January, Wann, Alpert, et al., 2014; January, Wann, Calkins, et al., 2019). Both the extrinsic (central) and intrinsic cardiac autonomic nervous systems (CANS) are thought to play an important role in the initiation and continuance of atrial fibrillation (Qin, Zeng, & Liu, 2019). Separate from the extrinsic (central) nervous system, which includes the brain and spinal cord, the CANS consists of a highly interconnected network of autonomic ganglia and nerve cell bodies embedded within the epicardium, largely within the atrial myocardium and great vessels (pulmonary veins). Hyperactive autonomic ganglia in the CANS are thought to play a critical role in atrial fibrillation, resulting in impulses that are initiated from the pulmonary veins and conducted through to the AV node. The ventricular rate of response depends on the conduction of atrial impulses through the AV node, presence of accessory electrical conduction pathways, and therapeutic effect of medications.

Lack of consistency in describing patterns or types of atrial fibrillation has led to the use of numerous labels, such as **paroxysmal** (i.e., sudden onset with

spontaneous termination), persistent, and permanent. The recommended classification system is noted in [Chart 22-3](#). The use of the term “chronic atrial fibrillation” is no longer included in the classification system, due to a lack of consensus on what constitutes chronicity (January et al., 2014, 2019).

**Chart 22-2**



## RISK FACTORS

### Atrial Fibrillation

- Increasing age
- Hypertension
- Diabetes
- Obesity
- Valvular heart disease
- Heart failure
- Obstructive sleep apnea
- Alcohol abuse
- Hyperthyroidism
- Myocardial infarction
- Smoking
- Exercise
- Cardiothoracic surgery
- Increased pulse pressure
- European ancestry
- Family history

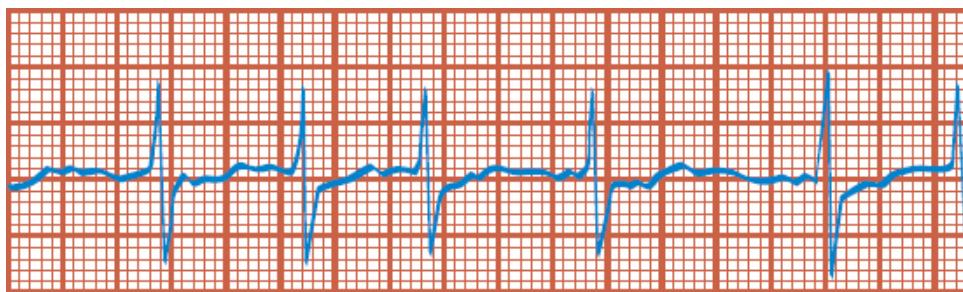
Adapted from January, C. T., Wann, L. S., Alpert, J. S., et al. (2014). 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: A report of the ACC/AHA Task Force on practice guidelines and the Heart Rhythm Society. *Circulation*, 130(23), e199–e267; January, C. T., Wann, L. S., Calkins, H., et al. (2019). 2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society in Collaboration With the Society of Thoracic Surgeons. *Circulation*, 140(2), e125–e151.

**Chart 22-3**

## Atrial Fibrillation Classification System

Type	Description
Paroxysmal	Sudden onset with termination that occurs spontaneously or after an intervention; lasts $\leq$ 7 days, but may recur
Persistent	Continuous, lasting >7 days
Long-standing persistent	Continuous, lasting >12 months
Permanent	Persistent, but decision has been made not to restore or maintain sinus rhythm
Nonvalvular	Absence of moderate-to-severe mitral stenosis or mechanical heart valve

Adapted from January, C. T., Wann, L. S., Alpert, J. S., et al. (2014). 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: A report of the ACC/AHA Task Force on practice guidelines and the Heart Rhythm Society. *Circulation*, 130(23), e199–e267; January, C. T., Wann, L. S., Calkins, H., et al. (2019). 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society in Collaboration With the Society of Thoracic Surgeons. *Circulation*, 140(2), e125–e151.



**Figure 22-10 •** Atrial fibrillation. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-22D). Philadelphia, PA: Wolters Kluwer.

Atrial fibrillation has the following characteristics (Fig. 22-10):

**Ventricular and atrial rate:** Atrial rate is 300 to 600 bpm; ventricular rate is usually 120 to 200 bpm in untreated atrial fibrillation

**Ventricular and atrial rhythm:** Highly irregular

**QRS shape and duration:** Usually normal, but may be abnormal

**P wave:** No discernible P waves; irregular undulating waves that vary in amplitude and shape are seen and referred to as fibrillatory or f waves

*PR interval:* Cannot be measured

*P:QRS ratio:* Many:1

Patients with atrial fibrillation are at increased risk of heart failure, myocardial ischemia, and embolic events such as stroke (January et al., 2014, 2019). A rapid and irregular ventricular response reduces the time for ventricular filling, resulting in a smaller stroke volume. Because atrial fibrillation causes a loss in AV synchrony (the atria and ventricles contract at different times), the atrial kick (the last part of diastole and ventricular filling, which accounts for 25% to 30% of the cardiac output) is also lost. As a consequence, although some patients with atrial fibrillation are asymptomatic, others experience palpitations and clinical manifestations of heart failure (e.g., shortness of breath, hypotension, dyspnea on exertion, fatigue; see [Chapter 25](#)). In addition, a high ventricular rate of response during atrial fibrillation (greater than 80 bpm) can eventually lead to mitral valve dysfunction, mitral regurgitation, intraventricular conduction delays, and dilated ventricular cardiomyopathy.

Patients with atrial fibrillation may exhibit a pulse deficit—a numeric difference between apical and radial pulse rates. The shorter time in diastole reduces the time available for coronary artery perfusion, thereby increasing the risk of myocardial ischemia with the onset of anginal symptoms (see [Chapter 23](#)). Decreasing the ventricular rate may avoid and correct these effects.

The erratic nature of atrial contraction, alterations in ventricular ejection, and atrial myocardial dysfunction promote the formation of thrombi, especially within the left atrium, increasing the risk of an embolic event. The origin of embolisms resulting in stroke for patients with nonvalvular atrial fibrillation is most often the left atrial appendage (LAA) (Schellinger, Tsivgoulis, Steiner, et al., 2018). A therapeutic approach to addressing the role of the LAA in atrial fibrillation, left atrial appendage occlusion (LAAO), is discussed later in this chapter.

## Assessment and Diagnostic Findings

The clinical evaluation of atrial fibrillation should include a history and physical examination that identifies the onset and nature of signs and symptoms, including their frequency, duration and any precipitating factors, and any response to medications. Whether or not the patient has a known history of heart disease or other risks is identified (see [Chart 22-2](#)). A 12-lead ECG is performed to verify the atrial fibrillation rhythm, as well as to identify the presence (or absence) of left ventricular (LV) hypertrophy, bundle branch block, prior myocardial ischemia, or other arrhythmias. The RR, QRS, and QT intervals are analyzed to verify the effectiveness of any prescribed antiarrhythmic medications (January et al., 2014, 2019). A transesophageal echocardiogram (TEE) can identify the presence of valvular heart disease, provide information about LV and right ventricular (RV) size and function, RV

pressures (to identify pulmonary hypertension, which may exist concomitant with atrial fibrillation), LV hypertrophy, and presence of left atrial thrombi (January et al., 2014, 2019).

Blood tests to screen for diseases that are known risks for atrial fibrillation (see [Chart 22-2](#)), including thyroid, renal, and hepatic function, are assessed in the patient with a new onset of atrial fibrillation, as well as when the ventricular rate is difficult to control (January et al., 2014, 2019). Additional tests may include a chest x-ray (to evaluate pulmonary vasculature in a patient suspected of having pulmonary hypertension), exercise stress test (to exclude myocardial ischemia or reproduce exercise-induced atrial fibrillation), Holter or event monitoring (see [Chapter 21](#)), and an EPS (January et al., 2014, 2019; see later discussion).

## Medical Management

Treatment of atrial fibrillation depends on the cause, pattern, and duration of the arrhythmia, the ventricular response rate, as well as the presence of structural or valvular heart disease and other cardiac conditions such as coronary artery disease or heart failure. Strategies for both rhythm control (i.e., conversion to sinus rhythm) and rate control are dependent on shared clinical decision making between the patient and primary provider. In some cases, atrial fibrillation spontaneously converts to sinus rhythm within 24 to 48 hours and without treatment. However, in instances where atrial fibrillation is concomitant with significant other morbid conditions (e.g., severe heart failure), the atrial fibrillation may be classified as “permanent,” meaning that the patient and primary provider have made a joint decision to stop further attempts to restore or maintain sinus rhythm. Therefore, management of atrial fibrillation may not only be different in different patients, but it also may change over time for any one patient.

Medical management revolves around preventing embolic events such as stroke with anticoagulant medications, controlling the ventricular rate of response with antiarrhythmic agents, and treating the arrhythmia as indicated so that it is converted to a sinus rhythm (i.e., *cardioversion*).

## Pharmacologic Therapy

**Antithrombotic Medications.** Antithrombotic drugs may include anticoagulants and antiplatelet drugs. Oral antithrombotic therapy is indicated for most patients with nonvalvular atrial fibrillation (e.g., absence of mechanical heart valve) because it reduces the risk of stroke (January et al., 2014, 2019). Atrial fibrillation guidelines recommend use of a scoring system to assist in assessment of stroke risk. Antithrombotic therapy is then selected based on risk factors outlined in the mnemonic CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> (see [Chart 22-4](#)) with each risk factor assigned points tallied for a total score that indicates an overall risk of stroke (January et al., 2014, 2019).

## Chart 22-4

### Stroke Risk Assessment for the Patient with Atrial Fibrillation: The CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> Scoring System

Risk Factor	Points
C Congestive Heart Failure (left ventricular systolic dysfunction)	1
H Hypertension (BP >130/80 mm Hg)	1
A <sub>2</sub> Age ≥75 years	2
D Diabetes	1
S <sub>2</sub> Prior Stroke/TIA/Thromboembolism	2
V Vascular disease (i.e., prior MI, PAD, or aortic plaque)	1
A Age 65–74 years	1
S <sub>c</sub> Sex category (female gender)	1

MI, myocardial infarction; PAD, peripheral artery disease, TIA; transient ischemic attack.

Adapted from January, C. T., Wann, L. S., Alpert, J. S., et al. (2014). 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: A report of the ACC/AHA Task Force on practice guidelines and the Heart Rhythm Society. *Circulation*, 130(23), e199–e267; January, C. T., Wann, L. S., Calkins, H., et al. (2019). 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society in Collaboration With the Society of Thoracic Surgeons. *Circulation*, 140(2), e125–e151.

According to pharmacologic treatment guidelines (January et al., 2014, 2019):

- Patients with nonvalvular atrial fibrillation with a CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> score of zero may choose the option of no antithrombotic therapy.
- Patients with nonvalvular atrial fibrillation with a CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> score of one may choose no antithrombotic therapy, treatment with an oral anticoagulant, or aspirin.
- Patients with nonvalvular atrial fibrillation with a CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> score of 2 or higher for men and 3 or higher for women may choose warfarin, or a direct thrombin inhibitor (e.g., dabigatran), or a Factor Xa inhibitor (e.g., rivaroxaban, apixaban, edoxaban).

Patients with atrial fibrillation with valvular heart disease or bioprosthetic heart valves may be prescribed warfarin, or a direct-acting oral anticoagulant, or a Factor Xa inhibitor (Malik, Yandrapalli, Aronow, et al., 2019). For

patients with mechanical heart valves, warfarin is recommended (January et al., 2014, 2019). If immediate or short-term anticoagulation is necessary, the patient may be placed on IV or low-molecular-weight heparin until warfarin therapy can be started and the international normalized ratio (INR) level reaches a therapeutic range consistent with antithrombosis, usually defined as an INR between 2.0 and 3.0 (see [Chapter 26](#) for further discussion).

Medication selection for all patients with atrial fibrillation depends upon stroke and bleeding risks as well as patient preferences and values (January et al., 2014, 2019). For instance, treatment with warfarin will require weekly INR testing during initiation of therapy, as well as ongoing monitoring (see [Chapter 26](#) for further discussion). Home monitoring of therapy is an option for some patients. Direct-acting oral anticoagulants and Factor Xa inhibitors require baseline assessment of hemoglobin and hematocrit, as well as liver and renal function, along with INR. Advantages of these medications include fewer drug-drug interactions and dietary limitations, as well as the elimination of frequent INR testing.

**Medications that Control the Heart Rate.** A strategy to control the ventricular rate of response so that the resting heart rate is less than 80 bpm is recommended in order to manage symptoms of atrial fibrillation (January et al., 2014, 2019). To decrease the ventricular rate in patients with paroxysmal, persistent, or permanent atrial fibrillation, a beta-blocker (Class II antiarrhythmic, see [Table 22-1](#)) or non-dihydropyridine calcium channel blocker (Class IV antiarrhythmic, see [Table 22-1](#)) is generally recommended (January et al., 2014, 2019).

**Medications that Convert the Heart Rhythm or Prevent Atrial Fibrillation.** For patients with atrial fibrillation lasting 48 hours or longer, anticoagulation is recommended prior to attempts to restore sinus rhythm, which may be achieved through pharmacologic or electrical cardioversion (January et al., 2014, 2019). In the absence of therapeutic anticoagulation, TEE may be performed prior to cardioversion to identify left atrial thrombus formation, including in the LAA (January et al., 2014, 2019). If no thrombus is identified, cardioversion can proceed.

Medications that may be given to achieve pharmacologic cardioversion to sinus rhythm include flecainide, dofetilide, propafenone, amiodarone, and IV ibutilide (January et al., 2014, 2019). These medications are most effective if given within 7 days of the onset of atrial fibrillation. It is recommended that patients who were prescribed dofetilide be hospitalized so that the QT interval and renal function both may be monitored. Despite a degree of risk, dofetilide is a preferred medication because it is highly effective at converting atrial fibrillation to sinus rhythm, has fewer drug-to-drug interactions, and is better tolerated by patients than other medications. Some patients with recurrent atrial fibrillation may be prescribed flecainide to self-administer at home, an approach referred to as “pill in the pocket” (January et al., 2014, 2019).

Preoperative administration of beta-blockers (see [Table 22-1](#)) has resulted in a significant reduction in atrial fibrillation after cardiac surgery (Burrage, Low, Campbell, et al., 2019). Cholesterol-lowering drugs such as the HMG-CoA reductase inhibitors (also called statins; see [Chapter 23](#), Table 23-1) may also be prescribed to prevent new-onset atrial fibrillation following cardiac surgery (Burrage et al., 2019).

If symptomatic, paroxysmal atrial fibrillation is refractory to at least one Class I or Class III antiarrhythmic medication (see [Table 22-1](#)), and rhythm control is desired, catheter ablation may be indicated (January et al., 2014, 2019; see later discussion).

### **Electrical Cardioversion for Atrial Fibrillation**

Electrical cardioversion is indicated for patients with atrial fibrillation who are hemodynamically unstable (e.g., acute alteration in mental status, chest discomfort, hypotension) and do not respond to medications (January et al., 2014, 2019). Flecainide, propafenone, amiodarone, dofetilide, or sotalol may be given prior to cardioversion to enhance the success of cardioversion and maintain sinus rhythm (January et al., 2014, 2019).



#### **Quality and Safety Nursing Alert**

*The patient with atrial fibrillation is at high risk for thrombus formation. When electrical cardioversion is indicated, the nurse may anticipate that a transesophageal echocardiogram may be performed to evaluate for possible atrial thrombi.*

Because atrial function may be impaired for several weeks after cardioversion, antithrombotic therapy (e.g., warfarin) is indicated for at least 4 weeks after the procedure (January et al., 2014, 2019). Repeated attempts at electrical cardioversion may be made, following administration of an antiarrhythmic medication (see later discussion on Electrical Cardioversion).

### **Cardiac Rhythm Therapies**

Atrial fibrillation that does not respond to medications or electrical cardioversion may be treated by cardiac rhythm therapies, including catheter ablation, maze or mini-maze procedure, or convergent procedure.

#### **Catheter Ablation Therapy**

Catheter **ablation** destroys specific cells that are the cause of a tachyarrhythmia. Catheter ablation is performed most often today for atrial fibrillation, although it may also be useful in treating atrioventricular nodal reentry tachycardia (AVNRT) and recurrent ventricular tachycardia (VT) (see later discussion of these arrhythmias).

Atrial fibrillation is associated with intrinsic cardiac autonomic nervous system activity to the pulmonary veins. Ablation involves a procedure similar to a cardiac catheterization (see [Chapter 21](#)); however, in this instance, a special catheter is advanced at or near the origin of the arrhythmia, where high-frequency, low-energy sound waves are passed through the catheter, causing thermal injury, localized cell destruction, and scarring. The tissue damage is more specific to the arrhythmic tissue, with less trauma to the surrounding cardiac tissue. Ablation may also be accomplished using a special catheter to apply extremely cold temperature to destroy selected cardiac cells, called *cryoablation*. The goal of each of these ablation procedures is to eliminate the arrhythmia, by preventing the ectopic activity arising from the pulmonary veins from reaching the atria, thereby stopping fibrillation (Weber, Sagerer-Gerhardt, & Heinze, 2017).

An EPS (see later discussion) may be performed to induce the arrhythmia prior to the catheter ablation. During the ablation procedure, defibrillation pads, an automatic blood pressure cuff, and a pulse oximeter are used. The patient is usually given moderate sedation (see [Chapter 15](#)) and IV heparin to reduce the risk of periprocedural thromboembolism. Immediately postablation, the patient is monitored for another 30 to 60 minutes and then retested to ensure that the arrhythmia does not recur. Successful ablation is achieved when the arrhythmia cannot be induced. Major risks of catheter ablation include pericardial effusion and tamponade, phrenic nerve injury, stroke, hematoma, retroperitoneal bleeding, pulmonary vein stenosis, and atrioesophageal fistulas (Canpolat, Kocyigit, & Aytemir, 2017).

**Nursing Management.** Postprocedural care on a step-down unit for the patient who has had ablation is similar to the nursing management of a patient who has had a cardiac catheterization (see [Chapter 21](#)); the patient is monitored closely to ensure recovery from sedation. Postprocedural nursing interventions include frequent monitoring for arrhythmias and for signs and symptoms of a stroke and vascular access site complications. Because of the prolonged time required for the procedure as well as the time needed in bed to obtain hemostasis at the vascular access site, it is not unusual for the patient to have back discomfort. In addition to administering any pain medications, the nurse may help to alleviate this pain by placing rolled towels under the patient's knees and waist.

### Maze and Mini-Maze Procedures

The maze procedure is an open heart surgical procedure for refractory atrial fibrillation. Small transmural incisions are made throughout the atria. The resulting formation of scar tissue prevents reentry conduction of the aberrant electrical impulse. Because the procedure requires significant time and cardiopulmonary bypass, its use is reserved only for those patients undergoing cardiac surgery for another reason (e.g., coronary artery bypass; January et al.,

2014, 2019). Some patients may need a permanent pacemaker after this surgery because of subsequent injury to the SA node.

A modification of the maze procedure, minimally invasive maze surgery, or mini-maze, may be performed by making small incisions between the ribs, through which video-guided instruments are inserted. The pulmonary veins are encircled with surgical incisions within the left atrium. This surgery eliminates the need for opening the sternum, heart-lung bypass, and the use of cardioplegia (see [Chapter 23](#) for further discussion of cardiac surgery). This results in a shorter recovery time and a lower risk of infection (January et al., 2014, 2019).

### Convergent Procedure

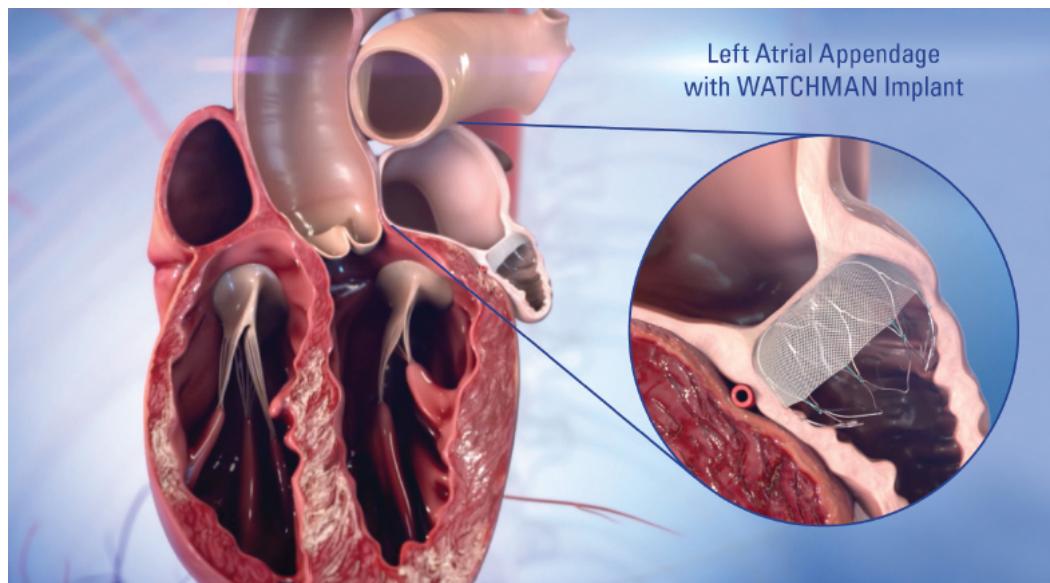
The convergent procedure utilizes a hybrid approach to ablation, requiring the skills of both a cardiothoracic surgeon and an electrophysiologist, a cardiologist with specialized training. This procedure is associated with lower rates of arrhythmia recurrence than catheter ablation, but more complications within 30 days of the procedure (e.g., infections, bleeding) (Jan, Zizek, Gersak, et al., 2018). The surgeon creates a few small incisions in the abdomen so that a special catheter that allows visualization can be inserted through the diaphragm and toward the posterior wall of the heart. The surgeon performs ablation of the epicardial wall in the area around the pulmonary veins and the electrophysiologist performs ablation around the endocardial area of the pulmonary veins. Because of the incisions, the patient usually has a 3-day hospital length of stay (Elrod, 2014). The patient may experience mild dull chest pain caused by the resulting inflammation from the ablation that usually resolves within a few days (Elrod, 2014). This pain is usually alleviated by treatment with acetaminophen as needed. In addition, if the phrenic nerve was affected, the patient may experience shortness of breath that may take days to weeks to resolve (Elrod, 2014).

### Left Atrial Appendage Occlusion

LAAO is an alternative to antithrombotic medications for stroke prevention in patients with nonvalvular atrial fibrillation (Masoudi, Calkins, Kavinsky, et al., 2015). As noted previously, the LAA is the area where the majority of stroke-causing blood clots form in patients with nonvalvular atrial fibrillation. However, concerns about the risk of long-term anticoagulant use and the risk of bleeding can complicate effective management (Ojo, Yandrapalli, Veseli, et al., 2020).

Candidates for LAAO include those patients with increased risk of stroke based on CHA<sub>2</sub>DS<sub>2</sub>-VAS<sub>C</sub> scores of one or higher (see [Chart 22-4](#)) and those patients seeking a nonpharmacologic alternative to treatment (Masoudi et al., 2015). Commonly used is the WATCHMAN, a device typically inserted while the patient is under general anesthesia. Similar to a percutaneous coronary

intervention (PCI) procedure (see Chapter 23), a small incision is made in the femoral area and a catheter is then inserted that guides the device into position. The parachute-shaped device is threaded through to the opening of the LAA, sealing it off and preventing it from releasing clots (see Fig. 22-11).



**Figure 22-11** • The WATCHMAN device in place over the left atrial appendage. ©2021 Boston Scientific Corporation or its affiliates. All rights reserved.

Patients typically stay in the hospital overnight after placement of a WATCHMAN device. The nursing management of patients who received this device is similar to that of patients post cardiac catheterization (see Chapter 21). Patients are prescribed aspirin and warfarin post procedure; approximately 6 weeks post procedure, they should return to the cardiology clinic for a TEE to confirm that the device has effectively occluded the LAA. If LAAO has occurred, then the patient may stop taking warfarin and is prescribed clopidogrel, an antiplatelet medication. After 6 months, the patient may stop taking clopidogrel but must continue taking daily aspirin indefinitely (Carlson & Doshi, 2017).

### Wolff-Parkinson-White Syndrome

In the patient with atrial fibrillation, if the QRS is wide and the ventricular rhythm is very fast and irregular, an accessory pathway should be suspected. An accessory pathway is typically congenital tissue between the atria, bundle of His, AV node, Purkinje fibers, or ventricular myocardium. This anomaly is known as Wolff-Parkinson-White (WPW) syndrome. Electrical cardioversion is the treatment of choice for atrial fibrillation in the presence of WPW syndrome that causes hemodynamic instability. Medications that block AV

conduction (e.g., digoxin, diltiazem, verapamil) should be avoided in WPW because they can increase the ventricular rate. If the patient is hemodynamically stable, procainamide, propafenone, flecainide, or amiodarone are recommended to restore sinus rhythm (January et al., 2014, 2019). Catheter ablation is performed for long-term management (see previous discussion).

## Atrial Flutter

Atrial flutter occurs because of a conduction defect in the atrium and causes a rapid, regular atrial impulse at a rate between 250 and 400 bpm. Because the atrial rate is faster than the AV node can conduct, not all atrial impulses are conducted into the ventricle, causing a therapeutic block at the AV node. This is an important feature of this arrhythmia. If all atrial impulses were conducted to the ventricle, the ventricular rate would also be 250 to 400 bpm, which would result in ventricular fibrillation, a life-threatening arrhythmia. Atrial flutter risk factors mirror those for atrial fibrillation (Fuster et al., 2017; see [Chart 22-2](#)).

Atrial flutter has the following characteristics (see [Fig. 22-12](#)):

*Ventricular and atrial rate:* Atrial rate ranges between 250 and 400 bpm; ventricular rate usually ranges between 75 and 150 bpm

*Ventricular and atrial rhythm:* The atrial rhythm is regular; the ventricular rhythm is usually regular but may be irregular because of a change in the AV conduction

*QRS shape and duration:* Usually normal, but may be abnormal or absent

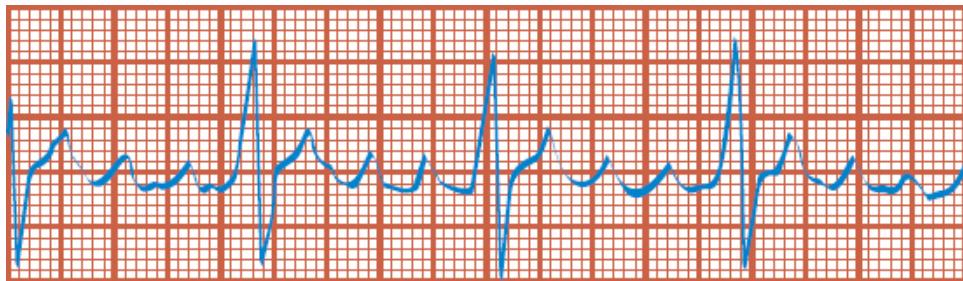
*P wave:* Saw-toothed shape; these waves are referred to as F waves

*PR interval:* Multiple F waves may make it difficult to determine the PR interval

*P:QRS ratio:* 2:1, 3:1, or 4:1

## Medical Management

Atrial flutter can cause serious signs and symptoms, such as chest pain, shortness of breath, and low blood pressure. Medical management involves the use of vagal maneuvers (see previous discussion under Sinus Tachycardia) or a trial administration of adenosine, which causes sympathetic block and slowing of conduction through the AV node. This may terminate the tachycardia; optimally, it will facilitate visualization of flutter waves for diagnostic purposes. Adenosine is given IV by rapid administration, and immediately followed by a 20-mL saline flush and elevation of the arm with the IV line to promote rapid circulation of the medication.



**Figure 22-12 •** Atrial flutter. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-22C). Philadelphia, PA: Wolters Kluwer.



**Figure 22-13 •** Various types of junctional rhythms. **A.** Note that the inverted P wave appears before the normal QRS complex. **B.** Note that the inverted P wave is buried inside the QRS complex. **C.** Note that the inverted P wave follows the QRS complex. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-23A, B, and C). Philadelphia, PA: Wolters Kluwer.

Atrial flutter is treated with antithrombotic therapy, rate control, and rhythm control in the same manner as atrial fibrillation (January et al., 2014, 2019). Electrical cardioversion is often successful in converting atrial flutter to sinus rhythm (see later discussion).

## Junctional Arrhythmias

Junctional arrhythmias originate within AV nodal tissue, and may include premature junctional complexes, junctional rhythms, nonparoxysmal junctional tachycardia, and AV nodal reentry tachycardia.

### Premature Junctional Complex

A premature junctional complex is an impulse that starts in the AV nodal area before the next normal sinus impulse reaches the AV node. Premature junctional complexes are less common than PACs. Causes include digitalis toxicity, heart failure, and coronary artery disease. The ECG criteria for premature junctional complex are the same as for PACs, except for the P wave and the PR interval. The P wave may be absent, may follow the QRS, or may occur before the QRS but with a PR interval of less than 0.12 seconds. This arrhythmia rarely produces significant symptoms. Treatment for frequent premature junctional complexes is the same as for frequent PACs.

### Junctional Rhythm

Junctional or idionodal rhythm occurs when the AV node, instead of the sinus node, becomes the pacemaker of the heart. When the sinus node slows (e.g., from increased vagal tone) or when the impulse cannot be conducted through the AV node (e.g., because of complete heart block), the AV node automatically discharges an impulse. Junctional rhythm not caused by complete heart block has the following characteristics (see [Fig. 22-13](#)):

*Ventricular and atrial rate:* Ventricular rate 40 to 60 bpm; atrial rate also 40 to 60 bpm if P waves are discernible

*Ventricular and atrial rhythm:* Regular

*QRS shape and duration:* Usually normal, but may be abnormal

*P wave:* May be absent, after the QRS complex, or before the QRS; may be inverted, especially in lead II

*PR interval:* If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds

*P:QRS ratio:* 1:1 or 0:1

### Medical Management

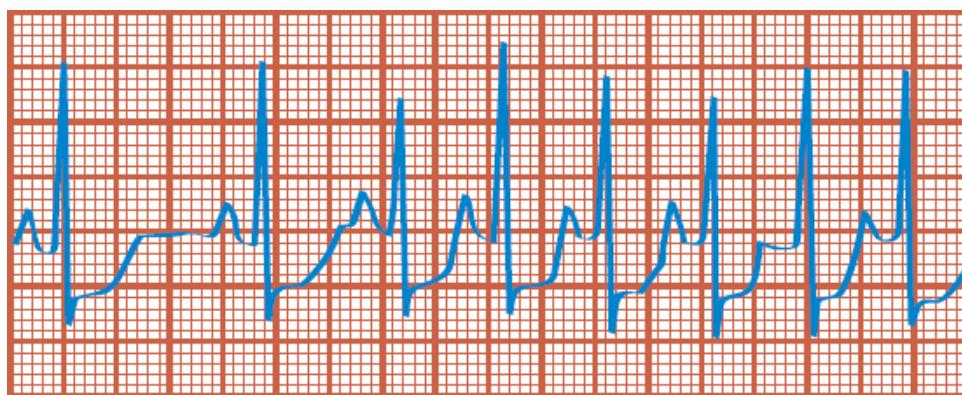
Junctional rhythm may produce signs and symptoms of reduced cardiac output. If this occurs, the treatment is the same as for sinus bradycardia. Emergency pacing may be needed (see later discussion under Pacemaker Therapy).

### Nonparoxysmal Junctional Tachycardia

Junctional tachycardia is caused by enhanced automaticity in the junctional area, resulting in a rhythm similar to junctional rhythm, except at a rate of 70 to 120 bpm. Although this rhythm generally does not have any detrimental hemodynamic effect, it may indicate a serious underlying condition, such as digitalis toxicity, myocardial ischemia, hypokalemia, or chronic obstructive pulmonary disease.

### Atrioventricular Nodal Reentry Tachycardia

AVNRT is a common arrhythmia that occurs when an impulse is conducted to an area in the AV node that causes the impulse to be rerouted back into the same area over and over again at a very fast rate. Each time the impulse is conducted through this area, it is also conducted down into the ventricles, causing a fast ventricular rate. AVNRT that has an abrupt onset and an abrupt cessation with a QRS of normal duration has frequently been called *paroxysmal atrial tachycardia (PAT)* and also *paroxysmal supraventricular tachycardia (PSVT)*. AVNRT also occurs when the duration of the QRS complex is 0.12 seconds or greater and a block in the bundle branch is known to be present. This arrhythmia may last for seconds or several hours. Factors associated with the development of AVNRT include caffeine, nicotine, hypoxemia, and stress. Underlying pathologies include coronary artery disease and cardiomyopathy; however, it occurs more often in females and not in association with underlying structural heart disease. AVNRT has the following characteristics (see Fig. 22-14):



**Figure 22-14 •** Atrioventricular nodal reentry tachycardia (AVNRT), also called paroxysmal atrial tachycardia (PAT), and paroxysmal supraventricular tachycardia (PSVT). Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-22B). Philadelphia, PA: Wolters Kluwer.

*Ventricular and atrial rate:* Atrial rate usually 150 to 250 bpm; ventricular rate usually 120 to 200 bpm

*Ventricular and atrial rhythm:* Regular; sudden onset and termination of the tachycardia

*QRS shape and duration:* Usually normal, but may be abnormal

*P wave:* Usually very difficult to discern

*PR interval:* If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds

*P:QRS ratio:* 1:1, 2:1

Clinical symptoms vary with the rate and duration of the tachycardia and the patient's underlying condition. The tachycardia usually is of short duration, resulting only in palpitations. A fast rate may also reduce cardiac output, resulting in significant signs and symptoms such as restlessness, chest pain, shortness of breath, pallor, hypotension, and loss of consciousness.

## Medical Management

Because AVNRT is generally a benign arrhythmia, the goal of medical management is to alleviate symptoms and improve quality of life. Patients who become significantly symptomatic and require emergency department visits to terminate the rhythm may want to initiate therapy immediately. However, those with minimum symptoms with an AVNRT that terminates spontaneously or with minimal treatment may choose just to be monitored and self-treat.

The aim of therapy is to break the reentry of the impulse. Catheter ablation is the initial treatment of choice and is used to eliminate the area that permits the rerouting of the impulse that causes the tachycardia (Katritsis, 2018; see previous discussion of Atrial Fibrillation: Catheter Ablation Therapy). Vagal maneuvers (see previous discussion under Sinus Tachycardia) may be used to interrupt AVNRT. These techniques increase parasympathetic stimulation, causing slower conduction through the AV node and blocking the reentry of the rerouted impulse. Some patients use some of these methods to terminate the episode on their own. Because of the risk of a cerebral embolic event, carotid sinus massage, which may be performed by physicians, is contraindicated in patients with carotid artery disease.

## Pharmacologic Therapy

If the vagal maneuvers are ineffective, the patient may then receive a bolus of adenosine to correct the rhythm; this is usually effective in terminating AVNRT. Because the effect of adenosine is so short, AVNRT may recur; the first dose may be followed with two additional doses. If the vagal maneuvers and adenosine are ineffective, IV non-dihydropyridine calcium channel blockers (e.g., verapamil), IV beta-blockers or IV digoxin may be considered (Hafeez & Armstrong, 2019). If the patient is unstable or does not respond to the medications, electrical cardioversion is the treatment of choice (see later discussion).

If P waves cannot be identified, the rhythm may be called supraventricular tachycardia (SVT), or PSVT if it has an abrupt onset, until the underlying rhythm and resulting diagnosis is determined. SVT and PSVT indicate only that the rhythm is not VT. SVT could be atrial fibrillation, atrial flutter, or AVNRT, among others. Vagal maneuvers and adenosine may be used to convert the rhythm or at least slow conduction in the AV node to allow visualization of the P waves.

## Ventricular Arrhythmias

Ventricular arrhythmias originate from foci within the ventricles; these may include premature ventricular complexes, VT, ventricular fibrillation, and idioventricular rhythms. Technically, ventricular asystole is characterized by an absence of rhythm formation.

### Premature Ventricular Complex

A PVC is an impulse that starts in a ventricle and is conducted through the ventricles before the next normal sinus impulse. PVCs can occur in healthy people, especially with intake of caffeine, nicotine, or alcohol. PVCs may be caused by cardiac ischemia or infarction, increased workload on the heart (e.g., heart failure and tachycardia), digitalis toxicity, hypoxia, acidosis, or electrolyte imbalances, especially hypokalemia.

In a rhythm referred to as bigeminy, every other complex is a PVC. In trigeminy, every third complex is a PVC, and in quadrigeminy, every fourth complex is a PVC. PVCs have the following characteristics (see Fig. 22-15):

*Ventricular and atrial rate:* Depends on the underlying rhythm (e.g., sinus rhythm)

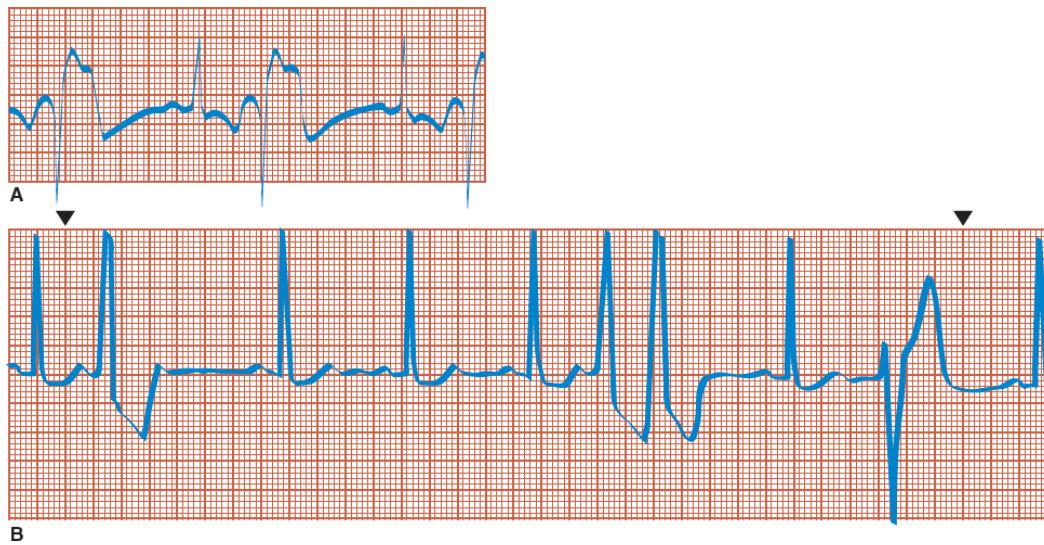
*Ventricular and atrial rhythm:* Irregular due to early QRS, creating one RR interval that is shorter than the others. The PP interval may be regular, indicating that the PVC did not depolarize the sinus node

*QRS shape and duration:* Duration is 0.12 seconds or longer; shape is bizarre and abnormal. When these bizarrely shaped, widened QRS complexes resemble each other, they are called unifocal. When they have at least two different morphologic appearances, they are called multifocal

*P wave:* Visibility of the P wave depends on the timing of the PVC; may be absent (hidden in the QRS or T wave) or in front of the QRS. If the P wave follows the QRS, the shape of the P wave may be different

*PR interval:* If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds

*P:QRS ratio:* 0:1; 1:1



**Figure 22-15 •** Premature ventricular complexes (PVCs). **A.** Ventricular bigeminy that is unifocal; note that every other beat is a PVC, with the same morphologic appearance. **B.** Multifocal PVCs; note that there are at least two different appearing PVCs. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-25B and C). Philadelphia, PA: Wolters Kluwer.

The patient may feel nothing or may say that the heart “skipped a beat.” The effect of a PVC depends on its timing in the cardiac cycle and how much blood was in the ventricles when they contracted. Initial treatment is aimed at correcting the cause.

### Medical Management

PVCs are a common occurrence and may increase in frequency with age (Al-khatib, Stevenson, Ackerman, et al., 2018). PVCs that are frequent and persistent may be treated with amiodarone or beta-blockers, but long-term pharmacotherapy for PVCs is not usually indicated. PVCs are not considered a warning for ensuing VT. However, studies have shown an association of PVCs with adverse outcomes; therefore, patients may need to be evaluated for underlying causes (e.g., ischemic heart disease and LV dysfunction) (Al-khatib et al., 2018).

### Unfolding Patient Stories: Kenneth Bronson • Part 2



Recall from [Chapter 19](#) Kenneth Bronson, who came to the emergency department with difficulty breathing after a week of flu-like symptoms, productive cough, and high fever. He was diagnosed with a right lower lobe pneumonia. Sinus tachycardia with occasional unifocal premature ventricular contractions (PVCs) is seen on the cardiac monitor. What are potential causes for the tachycardia and PVCs that the nurse should investigate when considering his age of 27 years, symptoms experienced over the last week, and the clinical manifestations associated with his diagnosis?

Care for Kenneth and other patients in a realistic virtual environment: [vSim \(\[thepoint.lww.com/vSimMedicalSurgical\]\(http://thepoint.lww.com/vSimMedicalSurgical\)\)](#). Practice documenting these patients' care in DocuCare ([thepoint.lww.com/DocuCareEHR](http://thepoint.lww.com/DocuCareEHR)).



## Ventricular Tachycardia

VT is defined as three or more PVCs in a row, occurring at a rate exceeding 100 bpm. The causes are similar to those of PVC. Patients with larger MIs and lower ejection fractions are at higher risk of lethal VT. VT is an emergency because the patient is nearly always unresponsive and pulseless. VT has the following characteristics (see [Fig. 22-16](#)):

*Ventricular and atrial rate:* Ventricular rate is 100 to 200 bpm; atrial rate depends on the underlying rhythm (e.g., sinus rhythm)

*Ventricular and atrial rhythm:* Usually regular; atrial rhythm may also be regular

*QRS shape and duration:* Duration is 0.12 seconds or more; bizarre, abnormal shape

*P wave:* Very difficult to detect, so the atrial rate and rhythm may be indeterminable

*PR interval:* Very irregular, if P waves are seen

*P:QRS ratio:* Difficult to determine, but if P waves are apparent, there are usually more QRS complexes than P waves

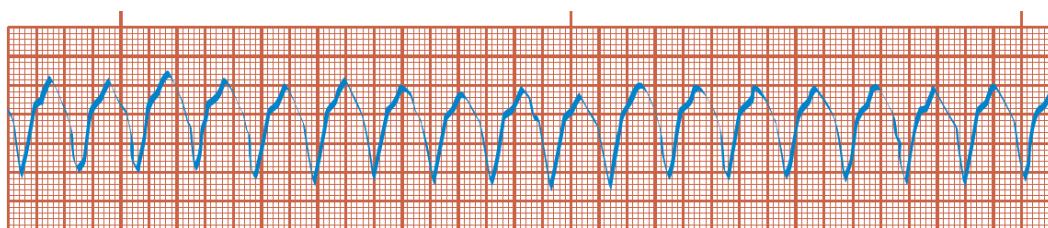
The patient's tolerance or lack of tolerance for this rapid rhythm depends on the ventricular rate and severity of ventricular dysfunction.

## Medical Management

Several factors determine the initial treatment, including the following: identifying the rhythm as monomorphic (having a consistent QRS shape and rate) or polymorphic (having varying QRS shapes and rhythms), determining

the existence of a prolonged QT interval before the initiation of VT, any comorbidities, and ascertaining the patient's heart function (normal or decreased). If the patient is stable, continuing the assessment, especially obtaining a 12-lead ECG, may be the only action necessary.

The patient may need antiarrhythmic medications, antitachycardia pacing, or direct cardioversion or defibrillation. Procainamide, amiodarone, sotalol, and lidocaine are all antiarrhythmic medications that may be considered based upon type of VT (e.g., monomorphic or polymorphic), clinical presentation, and patient comorbidities (e.g., impaired cardiac function, acute MI).



**Figure 22-16 •** Ventricular tachycardia. Reprinted with permission from Huff, J. (2002). *ECG Workout* (4th ed., p. 197). Philadelphia, PA: Lippincott, Williams & Wilkins.

Cardioversion is the treatment of choice for monophasic VT in a patient who is symptomatic. **Defibrillation**, which uses an electrical current given to stop the arrhythmia that is not set to synchronize with the patient's QRS complex, is the treatment of choice for pulseless VT. Any type of VT in a patient who is unconscious and without a pulse is treated in the same manner as ventricular fibrillation: immediate defibrillation is the action of choice (see later discussion on Cardioversion and Defibrillation).

For long-term management, patients with an ejection fraction less than 35% should be considered for an implantable cardioverter defibrillator (ICD) (see later discussion). Those with an ejection fraction greater than 35% may be managed with antiarrhythmic medication.

Torsades de pointes is a polymorphic VT preceded by a prolonged QT interval, which could be congenital or acquired. Common causes include central nervous system disease; certain medications (e.g., ciprofloxacin, erythromycin, haloperidol, lithium, methadone); or low levels of potassium, calcium, or magnesium. Congenital QT prolongation is another cause. Because this rhythm is likely to cause the patient to deteriorate and become pulseless, immediate treatment is required and includes correction of any electrolyte imbalance, such as administration of IV magnesium, and with IV isoproterenol or pacing if associated with bradycardia (Link, Berkow, Kudenchuk, et al., 2015; Soar, Donnino, Maconochie, et al., 2018).



## Ventricular Fibrillation

The most common arrhythmia in patients with cardiac arrest is ventricular fibrillation, which is a rapid, disorganized ventricular rhythm that causes ineffective quivering of the ventricles. No atrial activity is seen on the ECG. The most common cause of ventricular fibrillation is coronary artery disease and resulting acute MI. Other causes include untreated or unsuccessfully treated VT, cardiomyopathy, valvular heart disease, several proarrhythmic medications, acid-base and electrolyte abnormalities, and electrical shock. Another cause is Brugada syndrome, in which the patient (frequently of Asian descent) has a structurally normal heart, few or no risk factors for coronary artery disease, and a family history of sudden cardiac death (Pappone & Santinelli, 2019). Ventricular fibrillation has the following characteristics (see Fig. 22-17):

*Ventricular rate:* Greater than 300 bpm

*Ventricular rhythm:* Extremely irregular, without a specific pattern

*QRS shape and duration:* Irregular, undulating waves with changing amplitudes. There are no recognizable QRS complexes

## Medical Management

Ventricular fibrillation is always characterized by the absence of an audible heartbeat, a palpable pulse, and respirations. Because there is no coordinated cardiac activity, cardiac arrest and death are imminent if the arrhythmia is not corrected. Early defibrillation is critical to survival, with administration of immediate bystander cardiopulmonary resuscitation (CPR) until defibrillation is available. For refractory ventricular fibrillation, administration of amiodarone and epinephrine may facilitate the return of a spontaneous pulse after defibrillation (Link et al., 2015; see Chapter 25 for further discussion on interventions during Cardiac Arrest).

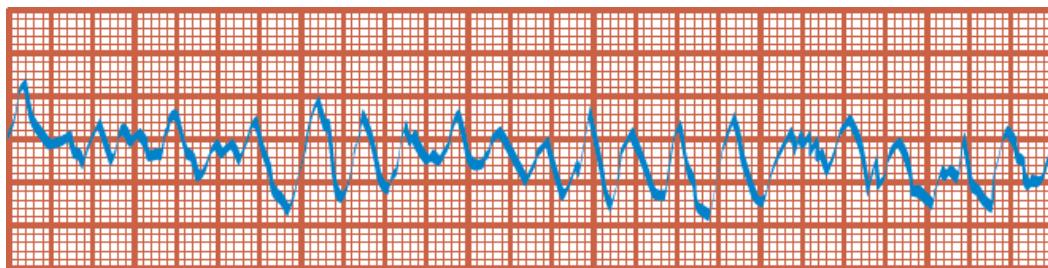
## Idioventricular Rhythm

Idioventricular rhythm, also called *ventricular escape rhythm*, occurs when the impulse starts in the conduction system below the AV node. When the sinus node fails to create an impulse (e.g., from increased vagal tone) or when the impulse is created but cannot be conducted through the AV node (e.g., due to complete AV block), the Purkinje fibers automatically discharge an impulse. When idioventricular rhythm is not caused by AV block, it has the following characteristics (see Fig. 22-18):

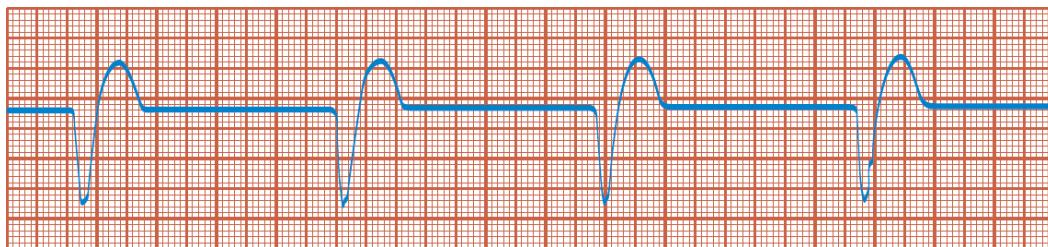
*Ventricular rate:* Between 20 and 40 bpm; if the rate exceeds 40 bpm, the rhythm is known as accelerated idioventricular rhythm

*Ventricular rhythm:* Regular

*QRS shape and duration:* Bizarre, abnormal shape; duration is 0.12 seconds or more



**Figure 22-17 •** Ventricular fibrillation. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical Care Nursing: A Holistic Approach* (11th ed., Fig. 17-27C). Philadelphia, PA: Wolters Kluwer.



**Figure 22-18 •** Idioventricular rhythm.

### Medical Management

Idioventricular rhythm commonly causes the patient to lose consciousness and experience other signs and symptoms of reduced cardiac output. In such cases, the treatment is the same as for asystole and pulseless electrical activity (PEA) (see Chapter 25) if the patient is in cardiac arrest or for bradycardia if the patient is not in cardiac arrest. Interventions include identifying the underlying cause; administering IV epinephrine, atropine, and vasopressor medications; and initiating emergency transcutaneous pacing. In some cases, idioventricular rhythm may cause no symptoms of reduced cardiac output.

### Ventricular Asystole

Commonly called *flatline*, ventricular asystole is characterized by absent QRS complexes confirmed in two different leads, although P waves may be apparent for a short duration. There is no heartbeat, no palpable pulse, and no respiration. Without immediate treatment, ventricular asystole is fatal.

### Medical Management

Ventricular asystole is treated the same as PEA, focusing on high-quality CPR with minimal interruptions and identifying underlying and contributing factors. The key to successful treatment is a rapid assessment to identify a possible cause, which is known as the Hs and Ts: hypoxia, hypovolemia, hydrogen ion (acid–base imbalance), hypo- or hyperglycemia, hypo- or hyperkalemia, hypothermia, trauma, toxins, tamponade (cardiac), tension pneumothorax, or thrombus (coronary or pulmonary) (Link et al., 2015). After the initiation of CPR, intubation and establishment of IV access are the next recommended actions, with no or minimal interruptions in chest compressions (see [Chapter 25](#)).

## Conduction Abnormalities

When assessing the rhythm strip, the underlying rhythm is first identified (e.g., sinus rhythm, sinus arrhythmia). Then, the PR interval is assessed for the possibility of an AV block. AV blocks occur when the conduction of the impulse through the AV node or bundle of His area is decreased or stopped. These blocks can be caused by medications (e.g., digitalis, calcium channel blockers, beta-blockers), Lyme disease, myocardial ischemia and infarction, hypothyroidism, or activities that cause an increase in vagal tone (Kusumoto et al., 2019). If the AV block is caused by increased vagal tone (e.g., long-term athletic training, sleep, coughing, suctioning, pressure above the eyes or on large vessels, anal stimulation), it is commonly accompanied by sinus bradycardia. AV block may be temporary and resolve on its own, or it may be permanent and require permanent pacing.

The clinical signs and symptoms of a heart block vary with the resulting ventricular rate and the severity of any underlying disease processes. Whereas first-degree AV block rarely causes any hemodynamic effect, the other blocks may result in decreased heart rate, causing a decrease in perfusion to vital organs, such as the brain, heart, kidneys, lungs, and skin. A patient with third-degree AV block caused by digitalis toxicity may be stable; another patient with the same rhythm caused by acute MI may be unstable. Health care providers must always keep in mind the need to treat the patient, not the rhythm. The treatment is based on the hemodynamic effect of the rhythm.

### First-Degree Atrioventricular Block

First-degree AV block occurs when all the atrial impulses are conducted through the AV node into the ventricles at a rate slower than normal. This conduction disorder has the following characteristics (see [Fig. 22-19](#)):

*Ventricular and atrial rate:* Depends on the underlying rhythm

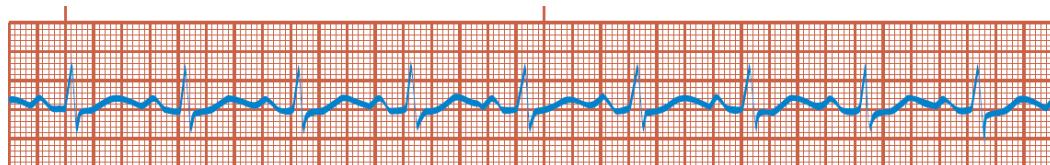
*Ventricular and atrial rhythm:* Depends on the underlying rhythm

*QRS shape and duration:* Usually normal, but may be abnormal

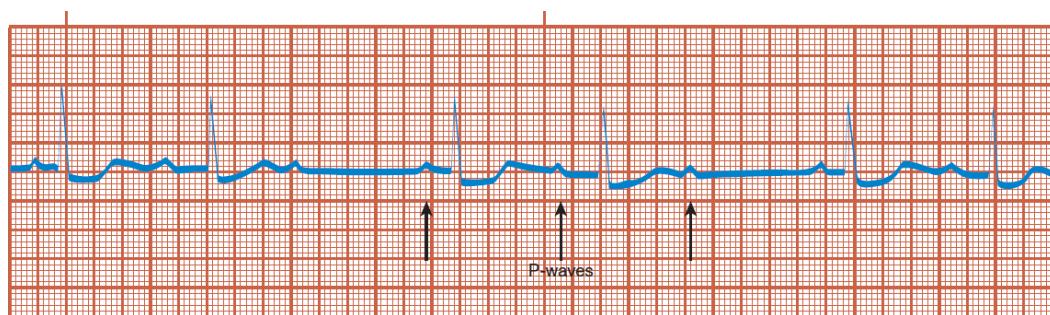
*P wave*: In front of the QRS complex; shows sinus rhythm, regular shape

*PR interval*: Greater than 0.20 seconds; PR interval measurement is constant.

*P:QRS ratio*: 1:1



**Figure 22-19 •** First-degree atrioventricular block. Note that the PR interval is constant but greater than 0.20 seconds. Reprinted with permission from Huff, J. (2002). *ECG Workout* (4th ed., p. 150). Philadelphia, PA: Lippincott, Williams & Wilkins.



**Figure 22-20 •** Second-degree atrioventricular block, type I. Note progressively longer PR durations until there is a nonconducted P wave. Reprinted with permission from Huff, J. (2017). *ECG Workout: Exercises in Arrhythmia Interpretation* (7th ed., Fig. 8-20). Philadelphia, PA: Wolters Kluwer.

### Second-Degree Atrioventricular Block, Type I (Wenckebach)

Second-degree AV block, type I, occurs when there is a repeating pattern in which all but one of a series of atrial impulses are conducted through the AV node into the ventricles (e.g., every four of five atrial impulses are conducted). Each atrial impulse takes a longer time for conduction than the one before, until one impulse is fully blocked. Because the AV node is not depolarized by the blocked atrial impulse, the AV node has time to fully repolarize so that the next atrial impulse can be conducted within the shortest amount of time. Second-degree AV block, type I, has the following characteristics (see Fig. 22-20):

*Ventricular and atrial rate*: Depends on the underlying rhythm, but the ventricular rate is lower than the atrial rate

*Ventricular and atrial rhythm:* The PP interval is regular if the patient has an underlying normal sinus rhythm; the RR interval characteristically reflects a pattern of change. Starting from the RR that is the longest, the RR interval gradually shortens until there is another long RR interval

*QRS shape and duration:* Usually normal, but may be abnormal

*P wave:* In front of the QRS complex; shape depends on underlying rhythm.

*PR interval:* The PR interval becomes longer with each succeeding ECG complex until there is a P wave not followed by a QRS. The changes in the PR interval are repeated between each “dropped” QRS, creating a pattern in the irregular PR interval measurements

*P:QRS ratio:* 3:2, 4:3, 5:4, and so forth

### **Second-Degree Atrioventricular Block, Type II**

Second-degree AV block, type II, occurs when only some of the atrial impulses are conducted through the AV node into the ventricles. Second-degree AV block, type II, has the following characteristics (see Fig. 22-21):

*Ventricular and atrial rate:* Depends on the underlying rhythm, but the ventricular rate is lower than the atrial rate

*Ventricular and atrial rhythm:* The PP interval is regular if the patient has an underlying normal sinus rhythm. The RR interval is usually regular but may be irregular, depending on the P:QRS ratio

*QRS shape and duration:* Usually abnormal, but may be normal

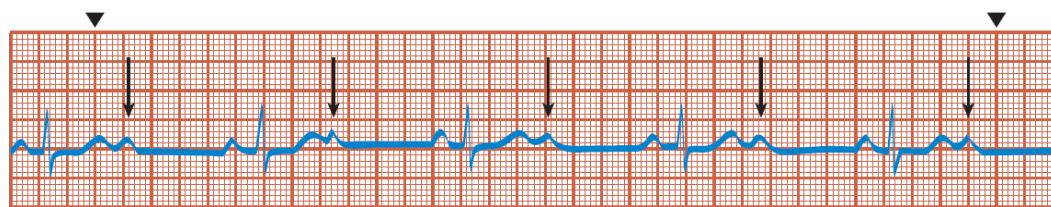
*P wave:* In front of the QRS complex; shape depends on underlying rhythm

*PR interval:* The PR interval is constant for those P waves just before QRS complexes

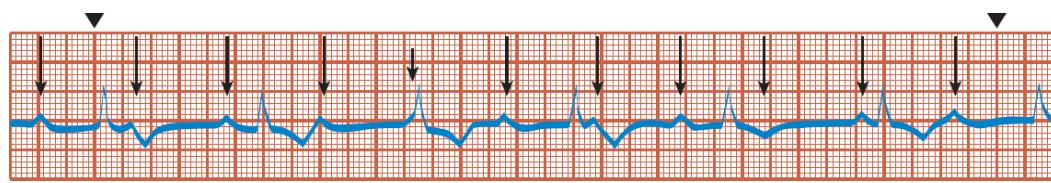
*P:QRS ratio:* 2:1, 3:1, 4:1, 5:1, and so forth

### **Third-Degree Atrioventricular Block**

Third-degree AV block occurs when no atrial impulse is conducted through the AV node into the ventricles. In third-degree AV block, two impulses stimulate the heart: one stimulates the ventricles, represented by the QRS complex, and one stimulates the atria, represented by the P wave. P waves may be seen, but the atrial electrical activity is not conducted down into the ventricles to initiate the QRS complex, the ventricular electrical activity. Having two impulses stimulate the heart results in a condition referred to as AV dissociation, which may also occur during VT. Complete block (third-degree AV block) has the following characteristics (see Fig. 22-22):



**Figure 22-21 •** Second-degree atrioventricular block, type II. Note constant PR interval and presence of more P waves than QRS complexes. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-29C). Philadelphia, PA: Wolters Kluwer.



**Figure 22-22 •** Third-degree atrioventricular block; devoid relationship between P waves and QRS complexes. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 17-29D). Philadelphia, PA: Wolters Kluwer.

*Ventricular and atrial rate:* Depends on the escape rhythm (idionodal or idioventricular) and underlying atrial rhythm, but the ventricular rate is lower than the atrial rate

*Ventricular and atrial rhythm:* The PP interval is regular and the RR interval is regular, but the PP interval is not equal to the RR interval

*QRS shape and duration:* Depends on the escape rhythm; with junctional rhythm, QRS shape and duration are usually normal; with idioventricular rhythm, QRS shape and duration are usually abnormal

*P wave:* Depends on underlying rhythm

*PR interval:* Very irregular

*P:QRS ratio:* More P waves than QRS complexes

## Medical Management of Conduction Abnormalities

Based on the cause of the AV block and the stability of the patient, treatment is directed toward increasing the heart rate to maintain a normal cardiac output. If the patient is stable and has no symptoms, no treatment may be indicated or it may simply consist of decreasing or eliminating the cause (e.g., withholding the medication or treatment). If the causal medication is necessary for treating other conditions and no effective alternative is available, pacemaker implantation may be indicated. The initial treatment of choice is an IV bolus of

atropine, although it is not effective in second-degree AV block, type II, or third-degree AV block. If the patient does not respond to atropine, has advanced AV block, or has had an acute MI, temporary transcutaneous pacing may be started. If the patient has no pulse, treatment is the same as for ventricular asystole (Link et al., 2015; Soar et al., 2018). A permanent pacemaker may be necessary if the block persists (see later discussion).

## NURSING PROCESS

### The Patient with an Arrhythmia

#### Assessment

Major areas of assessment include possible causes of the arrhythmia, contributing factors, and the arrhythmia's effect on the heart's ability to pump an adequate blood volume. When cardiac output is reduced, the amount of oxygen reaching the tissues and vital organs is diminished. This diminished oxygenation produces the signs and symptoms associated with arrhythmias. If these signs and symptoms are severe or if they occur frequently, the patient may experience significant distress and disruption of daily life.

A health history is obtained to identify any previous occurrences of decreased cardiac output, such as syncope, lightheadedness, dizziness, fatigue, chest discomfort, and palpitations. Possible causes of the arrhythmia (e.g., heart disease, chronic obstructive pulmonary disease) need to be identified. All medications, prescribed and over-the-counter (including herbs and nutritional supplements), as well as the route of administration, are reviewed. If a patient is taking an antiarrhythmic medication, assessment for treatment adherence, side effects, adverse reactions, and potential contraindications is necessary. For example, some medications (e.g., digoxin) can cause arrhythmias. Laboratory results are reviewed to assess levels of medications as well as factors that could contribute to the arrhythmia (e.g., anemia). A thorough psychosocial assessment is performed to identify the possible effects of the arrhythmia, the patient's perception and understanding of the arrhythmia and its treatment, and whether anxiety is a significant contributing factor.

The nurse conducts a physical assessment to confirm the data obtained from the history and to observe for signs of diminished cardiac output during the arrhythmic event, especially changes in level of consciousness. The nurse assesses the patient's skin, which may be pale and cool. Signs of fluid retention, such as neck vein distention and crackles and wheezes auscultated in the lungs, may be detected. The rate and rhythm of apical and peripheral pulses are also assessed, and any pulse deficit is noted. The nurse auscultates for extra heart sounds (especially S<sub>3</sub> and S<sub>4</sub>) and for heart murmurs, measures blood pressure, and determines pulse pressures. A declining pulse pressure indicates reduced cardiac output. One assessment may not disclose significant changes in cardiac output; therefore, the nurse compares multiple assessment findings over time, especially those that occur with and without the arrhythmia.

#### Diagnosis

#### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include:

- Impaired cardiac output associated with inadequate ventricular filling or altered heart rate
- Anxiety associated with fear of the unknown outcome of altered health state
- Lack of knowledge about the arrhythmia and its treatment

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Cardiac arrest (see [Chapter 25](#))
- Heart failure (see [Chapter 25](#))
- Thromboembolic event, especially with atrial fibrillation (see [Chapter 26](#))

#### **Planning and Goals**

The major goals for the patient may include eliminating or decreasing the occurrence of the arrhythmia (by decreasing contributory factors) to maintain cardiac output; verbalizing reduction in anxiety; verbalizing an understanding about the arrhythmia, tests used to diagnose the problem, and its treatment; and developing or maintaining self-management skills.

#### **Nursing Interventions**

##### **MONITORING AND MANAGING THE ARRHYTHMIA TO MAINTAIN CARDIAC OUTPUT**

The nurse evaluates the patient's blood pressure, pulse rate and rhythm, rate and depth of respirations, and breath sounds on an ongoing basis to determine the arrhythmia's hemodynamic effect. The nurse also asks the patient about possible symptoms of the arrhythmia (e.g., episodes of lightheadedness, dizziness, or fainting) as part of the ongoing assessment. If a patient with an arrhythmia is hospitalized, the nurse may obtain a 12-lead ECG, continuously monitor the patient, and analyze rhythm strips to track the arrhythmia.

Control of the occurrence or the effect of the arrhythmia, or both, is often achieved with antiarrhythmic medications. The nurse assesses and observes for the benefits and adverse effects of each medication. The nurse, in collaboration with the primary provider, also manages medication administration carefully so that a constant serum level of the medication is maintained. The nurse may also conduct a 6-minute walk test as prescribed, which is used to identify the patient's ventricular rate in response to exercise. The patient is asked to walk for 6 minutes, covering as much distance as possible (Chen, Chen, Lu, et al., 2018). The nurse monitors the patient for symptoms. At the end, the nurse records the distance covered and the pre- and postexercise heart rate as well as the patient's response.

The nurse assesses for factors that contribute to the arrhythmia (e.g., oxygen deficits, acid-base and electrolyte imbalances, caffeine, or

nonadherence to the medication regimen). The nurse also monitors for ECG changes (e.g., widening of the QRS, prolongation of the QT interval, increased heart rate) that increase the risk of an arrhythmic event.

#### **REDUCING ANXIETY**

When the patient experiences episodes of arrhythmia, the nurse stays with the patient and provides assurance of safety and security while maintaining a calm and reassuring attitude. This assists in reducing anxiety (reducing the sympathetic response) and fosters a trusting relationship with the patient. The nurse seeks the patient's view of the events and discusses the emotional response to the arrhythmia, encouraging verbalization of feelings and fears, providing supportive or empathetic statements, and assisting the patient to recognize feelings of anxiety, anger, or sadness. The nurse emphasizes successes with the patient to promote a sense of self-management of the arrhythmia. For example, if a patient is experiencing episodes of arrhythmia and a medication is given that begins to reduce the incidence of the arrhythmia, the nurse communicates that information to the patient and explores the patient's response to this information. In addition, the nurse can help the patient develop a system to identify possible causative, influencing, and alleviating factors (e.g., keeping a diary). The nursing goal is to maximize the patient's control and to make the episode less threatening.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** When educating patients about arrhythmias, the nurse first assesses the patient's understanding, clarifies misinformation, and then shares needed information in terms that are understandable and in a manner that is not frightening or threatening. The nurse clearly explains the etiology of the arrhythmia and treatment options to the patient and family. If necessary, the nurse explains the importance of maintaining therapeutic serum levels of antiarrhythmic medications so that the patient understands why medications should be taken regularly each day and the importance of regular blood testing. If the medication has the potential to alter the heart rate, the patient should be taught how to take their pulse before each dose and to notify the primary provider if the pulse is abnormal. In addition, the relationship between an arrhythmia and cardiac output is explained so that the patient recognizes symptoms of the arrhythmia and the rationale for the treatment regimen. If the patient is prescribed an anticoagulant medication, patient education points about taking anticoagulant medications are summarized in [Chapter 26, Chart 26-10](#). The patient and family need to be educated about measures to take to decrease the risk of recurrence of the arrhythmia. If the patient has a potentially lethal arrhythmia, the nurse establishes with the

patient and family a plan of action to take in case of an emergency and, if appropriate, encourages a family member to obtain CPR training.

The patient and family should also be educated about potential risks of the arrhythmia and their signs and symptoms. For example, the patient with atrial fibrillation should be educated about the possibility of an embolic event.

**Continuing and Transitional Care.** A referral for home, community-based, or transitional care usually is not necessary for the patient with an arrhythmia unless the patient is hemodynamically unstable and has significant symptoms of decreased cardiac output. Home, community-based, or transitional care may be warranted if the patient has significant comorbidities, socioeconomic issues, or limited self-management skills that could increase the risk of nonadherence to the therapeutic regimen. A referral may also be indicated if the patient has had an electronic device implanted recently.

### Evaluation

Expected patient outcomes may include:

1. Maintains cardiac output
  - a. Demonstrates heart rate, blood pressure, respiratory rate, and level of consciousness within normal ranges
  - b. Demonstrates no or decreased episodes of arrhythmia
2. Experiences reduced anxiety
  - a. Expresses a positive attitude about living with the arrhythmia
  - b. Expresses confidence in ability to take appropriate actions in an emergency
3. Expresses understanding of the arrhythmia and its treatment
  - a. Explains the arrhythmia and its effects
  - b. Describes the medication regimen and its rationale
  - c. Explains the need to maintain a therapeutic serum level of the medication
  - d. Describes a plan to eliminate or limit factors that contribute to the arrhythmia
  - e. States actions to take in the event of an emergency

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## ADJUNCT MODALITIES AND MANAGEMENT

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Arrhythmia treatments depend on whether the disorder is acute or chronic, as well as on the cause of the arrhythmia and its actual or potential hemodynamic effects.

Acute arrhythmias may be treated with medications or with external electrical therapy (emergency defibrillation, cardioversion, or pacing). Many antiarrhythmic medications are used to treat atrial and ventricular tachyarrhythmias (see [Table 22-1](#)). The choice of medication depends on the specific arrhythmia and its duration, the presence of structural heart disease (e.g., heart failure), and the patient's response to previous treatment. The nurse is responsible for monitoring and documenting the patient's responses to the medication and for ensuring that the patient has the knowledge and ability to manage the medication regimen.

If medications alone are ineffective in eliminating or decreasing the arrhythmia, certain adjunct mechanical therapies are available. The most common therapies are elective cardioversion and defibrillation for acute tachyarrhythmia, and implantable electronic devices for bradycardias (pacemakers) and chronic tachyarrhythmias (ICDs). Surgical treatments, although less common, are also available. The nurse is responsible for assessing the patient's understanding of and response to mechanical therapy, as well as the patient's self-management abilities. The nurse explains that the purpose of the device is to help the patient lead a life that is as active and productive as possible.

## Cardioversion and Defibrillation

Cardioversion and defibrillation are used to treat tachyarrhythmias by delivering an electrical current that depolarizes a critical mass of myocardial cells. When the cells repolarize, the SA node is usually able to recapture its role as the heart's pacemaker.



### Concept Mastery Alert

*One major difference between cardioversion and defibrillation is the timing of the delivery of electrical current. In cardioversion, the delivery of the electrical current is synchronized with the patient's electrical events; in defibrillation, the delivery of the current is immediate and unsynchronized.*

The same type of device, called a *defibrillator*, is used for both cardioversion and defibrillation. The electrical voltage required to defibrillate the heart is usually greater than that required for cardioversion and may cause more myocardial damage. Only biphasic types of defibrillators are now manufactured; these deliver an electrical charge from one paddle that then automatically redirects its charge back to the originating paddle. Because the delivery of the electrical charge varies among devices, the manufacturer's

recommended dose should be followed for the first and subsequent defibrillations (Link et al., 2015; Soar et al., 2018).



**Figure 22-23 •** Standard paddle placement for defibrillation.

The electrical current may be delivered externally through the skin with the use of paddles or with conductor pads. The paddles or pads may be placed on the front of the chest (standard placement) (see Fig. 22-23), or one pad may be placed on the front of the chest and the other pad placed under the patient's back just left of the spine (anteroposterior placement) (see Fig. 22-24).

Defibrillator multifunction conductor pads contain a conductive medium and are connected to the defibrillator to allow for hands-off defibrillation. This method reduces the risk of touching the patient during the procedure and increases electrical safety. Automated external defibrillators (AEDs), which are now found in many public areas, use this type of delivery for the electrical current.

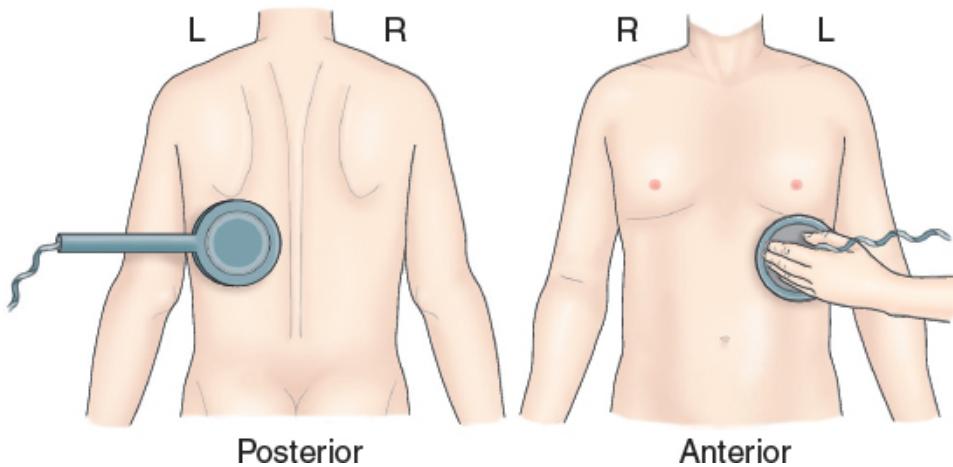


#### **Quality and Safety Nursing Alert**

*When using paddles, the appropriate conductant is applied between the paddles and the patient's skin. Any other type of conductant, such as ultrasound gel, should not be substituted.*

Whether using pads or paddles, the nurse must observe two safety measures. First, good contact must be maintained between the pads or paddles and the patient's skin (with a conductive medium between them) to prevent electrical current from leaking through the air (arcng) when the defibrillator is

discharged. Second, no one is to be in contact with the patient or with anything that is touching the patient when the defibrillator is discharged, to minimize the chance that electrical current is conducted to anyone other than the patient. [Chart 22-5](#) provides a review of nursing responsibility when a patient is cardioverted or defibrillated.



**Figure 22-24 •** Anteroposterior paddle placement for defibrillation.

### Chart 22-5

## Assisting with External Defibrillation or Cardioversion

When assisting with external defibrillation or cardioversion, the nurse should remember these key points:

- Multifunction conductor pads or paddles are used, with a conducting medium between the paddles and the skin in the proper locations. The conducting medium is available as a sheet, gel, or paste. Gels or pastes with poor electrical conductivity (e.g., ultrasound gel) should not be used.
- Paddles or pads should be placed so that they do not touch the patient's clothing or bed linen and are not near medication patches or in the direct flow of oxygen.
- Women with large breasts should have the left pad or paddle placed underneath or lateral to the left breast.
- During cardioversion, the monitor leads must be attached to the patient in order to set the defibrillator to the synchronized mode ("in sync"). If defibrillating, the defibrillator must *not* be in the synchronized mode (most machines default to the "not-sync" mode).
- When using paddles, 20–25 lb of pressure must be used in order to ensure good skin contact.
- When using a manual discharge device, it must not be charged until it is ready to shock; then thumbs and fingers must be kept off the discharge buttons until paddles or pads are on the chest and ready to deliver the electrical charge.
- When it is time to defibrillate, whomever is delivering the charge should announce, "charging to (number of joules)" prior to discharging.
- "Clear!" must be called three times before discharging: As "Clear" is called the first time, the discharger must visually check that they are not touching the patient, bed, or equipment; as "Clear" is called the second time, the discharger must visually check that no one else is touching the bed, the patient, or equipment, including the endotracheal tube or adjuncts; and as "Clear" is called the third time, the discharger must perform a final visual check to ensure that everyone is clear of the patient and anything touching the patient.
- The delivered energy and resulting rhythm are recorded.
- Cardiopulmonary resuscitation (CPR) is immediately resumed after the defibrillation charge is delivered, if appropriate, starting with chest compressions.
- If CPR is necessary, after five cycles (about 2 minutes) of CPR, the cardiac rhythm is checked again and another shock is delivered, if warranted. A vasoactive or antiarrhythmic medication is given as soon as possible after the rhythm check to facilitate a positive response to defibrillation.
- After the event is complete, the skin under the pads or paddles is inspected for burns; if any are detected, the primary provider or a wound care nurse is consulted about appropriate treatment.

- The defibrillator is plugged back into an outlet, and supplies are restocked as needed.

Adapted from Link, M. S., Berkow, L. C., Kudenchuk, P. J., et al. (2015). Part 7: Adult advanced cardiovascular life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 132(18 supp 2), S444–S464; Soar, J., Donnino, M. W., Maconochie, I., et al. (2018). 2018 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations summary. *Resuscitation*, 133, 194–206.

## Electrical Cardioversion

Electrical cardioversion involves the delivery of a “timed” electrical current to terminate a tachyarrhythmia. In cardioversion, the defibrillator is set to synchronize with the ECG on a cardiac monitor so that the electrical impulse discharges during ventricular depolarization (QRS complex). The synchronization prevents the discharge from occurring during the vulnerable period of repolarization (T wave), which could result in VT or ventricular fibrillation. The ECG monitor connected to the external defibrillator usually displays a mark or line that indicates sensing of a QRS complex. Sometimes the lead and the electrodes must be changed for the monitor to recognize the patient’s QRS complex. When the synchronizer is on, no electrical current is delivered if the defibrillator does not discern a QRS complex. Therefore, it is important to ensure that the patient is connected to the monitor and to select a lead (not “paddles”) that has the most appropriate sensing of the QRS. Because there may be a short delay until recognition of the QRS, the discharge buttons of an external manual defibrillator must be held down until the shock has been delivered. In most monitors, the synchronization mode must be reactivated if the initial cardioversion was ineffective and another cardioversion is needed (i.e., the device defaults to unsynchronized defibrillation mode).

If the cardioversion is elective and the arrhythmia has lasted longer than 48 hours, anticoagulation for a few weeks before cardioversion may be indicated (January et al., 2014, 2019). Digoxin is usually withheld for 48 hours before cardioversion to ensure the resumption of sinus rhythm with normal conduction. The patient is instructed not to eat or drink for at least 4 hours before the procedure. Gel-covered paddles or conductor pads are positioned anteroposteriorly (front and back) for cardioversion. Before cardioversion, the patient receives moderate sedation IV as well as an analgesic medication or anesthesia. Respiration is then supported with supplemental oxygen delivered by a bag-valve mask device with suction equipment readily available. Although patients rarely require intubation, equipment is nearby in case it is needed. The amount of voltage used varies from 50 to 360 joules, depending

on the defibrillator's technology, the type and duration of the arrhythmia, and the size and hemodynamic status of the patient (Link et al., 2015; Soar et al., 2018).

Indications of a successful response are conversion to sinus rhythm, adequate peripheral pulses, and adequate blood pressure. Because of the sedation, airway patency must be maintained and the patient's state of consciousness assessed. Vital signs and oxygen saturation are monitored and recorded until the patient is stable and recovered from sedation and analgesic medications or anesthesia. ECG monitoring is required during and after cardioversion (Link et al., 2015; Soar et al., 2018).

## Defibrillation

Defibrillation is used in emergency situations as the treatment of choice for ventricular fibrillation and pulseless VT, the most common cause of abrupt loss of cardiac function and sudden cardiac death. Defibrillation is not used for patients who are conscious or have a pulse. The energy setting for the initial and subsequent shocks using a monophasic defibrillator should be set at 360 joules (Link et al., 2015; Soar et al., 2018). The energy setting for the initial shock using a biphasic defibrillator may be set at 150 to 200 joules, with the same or an increasing dose with subsequent shocks (Link et al., 2015; Soar et al., 2018). The sooner defibrillation is used, the better the survival rate (Hedge & Gnugnoli, 2019). Several studies have demonstrated that early defibrillation performed by lay people in a community setting can increase the survival rate (Hedge & Gnugnoli, 2019). If immediate CPR is provided and defibrillation is performed within 5 minutes, more adults in ventricular fibrillation may survive with intact neurologic function (Link et al., 2015; Soar et al., 2018). The availability and the use of AEDs in public places can shorten the interval from collapse to rhythm recognition and defibrillation, which can significantly improve survival out of the hospital (Hedge & Gnugnoli, 2019).

Epinephrine is given after initial unsuccessful defibrillation to make it easier to convert the arrhythmia to a normal rhythm with the next defibrillation. This medication may also increase cerebral and coronary artery blood flow. Antiarrhythmic medications such as amiodarone, lidocaine, or magnesium may be given if ventricular arrhythmia persists (see [Table 22-1](#)). This treatment with continuous CPR, medication administration, and defibrillation continues until a stable rhythm resumes or until it is determined that the patient cannot be revived.

## Electrophysiology Studies

An EPS is an invasive procedure used to evaluate and treat various chronic arrhythmias that have caused cardiac arrest or significant symptoms. It also is indicated for patients with symptoms that suggest an arrhythmia that has gone undetected and undiagnosed by other methods. Because an EPS is invasive, it is performed in the hospital and may require that the patient be admitted. An EPS is used to do the following:

- Identify the impulse formation and propagation through the cardiac electrical conduction system
- Assess the function or dysfunction of the SA and AV nodal areas
- Identify the location (called *mapping*) and mechanism of the arrhythmogenic foci (the exact site where the arrhythmia originates)
- Assess the effectiveness of antiarrhythmic medications and devices for the patient with an arrhythmia
- Treat certain arrhythmias through the destruction of the causative cells (ablation)

An EPS procedure is a type of cardiac catheterization that is performed in a specially equipped cardiac catheterization laboratory by an electrophysiologist, assisted by other EPS laboratory personnel. The patient is conscious but lightly sedated. Usually, one or more catheters are inserted into the groin, neck, or antecubital fossa. The electrodes are positioned within the heart at specific locations—for instance, in the right atrium near the sinus node, in the coronary sinus, near the tricuspid valve, and at the apex of the right ventricle. The number and placement of electrodes depend on the type of study being conducted. These electrodes allow the electrical signal to be recorded from within the heart (intracardiogram).

The electrodes also allow the electrophysiologist to introduce a pacing stimulus to the intracardiac area at a precisely timed interval and rate, thereby stimulating the area (programmed stimulation). An area of the heart may be paced at a rate much faster than the normal rate of **automaticity**, the rate at which impulses are spontaneously formed (e.g., in the sinus node). This allows the pacemaker to become an artificial focus of automaticity and to assume control (overdrive suppression). Then, the pacemaker is stopped suddenly, and the time it takes for the sinus node to resume control is assessed. A prolonged time indicates dysfunction of the sinus node.

One of the main purposes of programmed stimulation is to assess the ability of the area surrounding the electrode to cause a reentry arrhythmia. One or a series of premature impulses is delivered to an area in an attempt to cause the tachyarrhythmia. Because the precise location of the suspected area and the specific timing of the pacing needed are unknown, the electrophysiologist uses many different techniques to cause the arrhythmia during the study. If the arrhythmia can be reproduced by programmed stimulation, it is called *inducible*. Once an arrhythmia is induced, a treatment plan is determined and

implemented. If, on the follow-up EPS, the tachyarrhythmia cannot be induced, then the treatment is determined to be effective. Different medications may be given and combined with cardiac implantable electronic devices to determine the most effective treatment to suppress the arrhythmia.

Patient care, patient education, and associated complications of an EPS are similar to those associated with cardiac catheterization (see [Chapter 21](#)). The study is usually about 2 hours in length; however, if the electrophysiologist conducts not only a diagnostic procedure but also treatment, the study can take up to 6 hours. During the procedure, patients benefit from a calm, reassuring approach.

Patients who are to undergo an EPS may be anxious about the procedure and its outcome. A detailed discussion involving the patient, the family, and the electrophysiologist usually occurs to ensure that the patient can give informed consent and to reduce the patient's anxiety about the procedure. Before the procedure, the nurse educates the patient about the EPS and its usual duration, the environment where the procedure is performed, and what to expect. Although an EPS is not painful, it does cause discomfort and can be tiring. It may also cause feelings that were experienced when the arrhythmia occurred in the past. In addition, patients are educated about what will be expected of them (e.g., lying very still during the procedure, reporting symptoms or concerns).

The patient should also know that the arrhythmia may occur during the procedure. It often stops on its own; if it does not, treatment is given to restore the patient's normal rhythm. The arrhythmia may have to be terminated using cardioversion or defibrillation, but this is performed under more controlled circumstances than if performed in an emergency.

Postprocedural care is similar to that for cardiac catheterization, including restriction of activity to promote hemostasis at the insertion site (see [Chapter 21](#)). To identify any complications and to ensure healing, the patient's vital signs and the appearance of the insertion site are assessed frequently. Because an artery is not always used, there is a lower incidence of vascular complications than with other catheterization procedures (Zipes, Libby, Bonow, et al., 2018).

## Pacemaker Therapy

A pacemaker is an electronic device that provides electrical stimuli to the heart muscle. Pacemakers are usually used when a patient has a permanent or temporary slower-than-normal impulse formation, or a symptomatic AV or ventricular conduction disturbance. They may also be used to control some tachyarrhythmias that do not respond to medication. Biventricular (both ventricles) pacing, also called **cardiac resynchronization therapy (CRT)**,

may be used to treat advanced heart failure. Pacemaker technology also may be used in conjunction with an ICD.

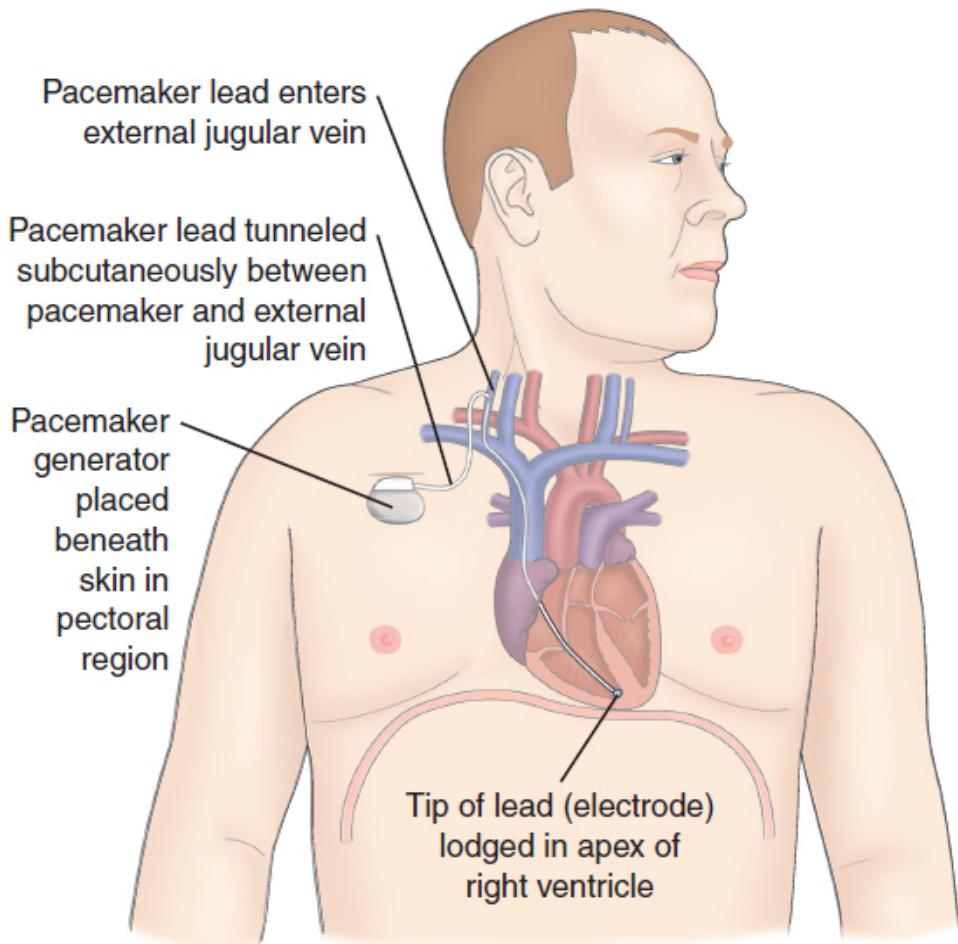
Pacemakers can be permanent or temporary. Temporary pacemakers are used to support patients until they improve or receive a permanent pacemaker (e.g., after acute MI or during open heart surgery). Temporary pacemakers are used only in hospital settings.

## Pacemaker Design and Types

Pacemakers consist of two components: an electronic pulse generator and pacemaker electrodes, which are located on leads or wires. The generator contains the circuitry and batteries that determine the rate (measured in beats per minute) and the strength or output (measured in milliamperes [mA]) of the electrical stimulus delivered to the heart. The generator also has circuitry that can detect the intracardiac electrical activity to cause an appropriate response; this component of pacing is called *sensitivity* and is measured in millivolts (mV). Sensitivity is set at the level that the intracardiac electrical activity must exceed to be sensed by the device. Leads, which carry the impulse created by the generator to the heart, can be threaded by fluoroscopy through a major vein into the heart, usually the right atrium and ventricle (endocardial leads), or they can be lightly sutured onto the outside of the heart and brought through the chest wall during open heart surgery (epicardial wires). These epicardial pacemakers are typically temporary and are removed by a gentle tug a few days after surgery. The endocardial leads may be temporarily placed with catheters through a vein (usually the femoral, subclavian, or internal jugular vein [transvenous wires]), usually guided by fluoroscopy. The leads may also be part of a specialized pulmonary artery catheter (see [Chapter 21](#)). However, obtaining a pulmonary artery wedge pressure may cause the leads to move out of pacing position. The endocardial and epicardial wires are connected to a temporary generator, which is about the size of a cellular phone. The energy source for a temporary generator is a common household battery. Monitoring for pacemaker malfunctioning and battery failure is a nursing responsibility.

The endocardial leads also may be placed permanently, passed into the heart through the subclavian, axillary, or cephalic vein, and connected to a permanent generator. These types of pacemakers are sometimes also called transvenous pacemakers. Most current leads have a fixation mechanism (e.g., a screw) at the end of the lead that allows precise positioning and avoidance of dislodgement. The permanent generator, which often weighs less than 1 ounce and is the size of a pack of chewing gum, is usually implanted in a subcutaneous pocket created in the pectoral region, below the clavicle in men, or behind the breast in women (see [Fig. 22-25](#)). This procedure usually takes about 1 hour, and it is performed in a cardiac catheterization laboratory using a

local anesthetic and moderate sedation. Close monitoring of the respiratory status is needed until the patient is fully awake.



**Figure 22-25 •** Implanted transvenous pacing lead (with electrode) and pacemaker generator.

Leadless pacemakers, a newer type of permanent pacemaker, are 90% smaller than transvenous pacemakers. They feature a self-contained, single-unit pulse generator and electrode that is inserted transvenously directly into the right ventricle (Groner & Grippe, 2019; Hayes, 2018).

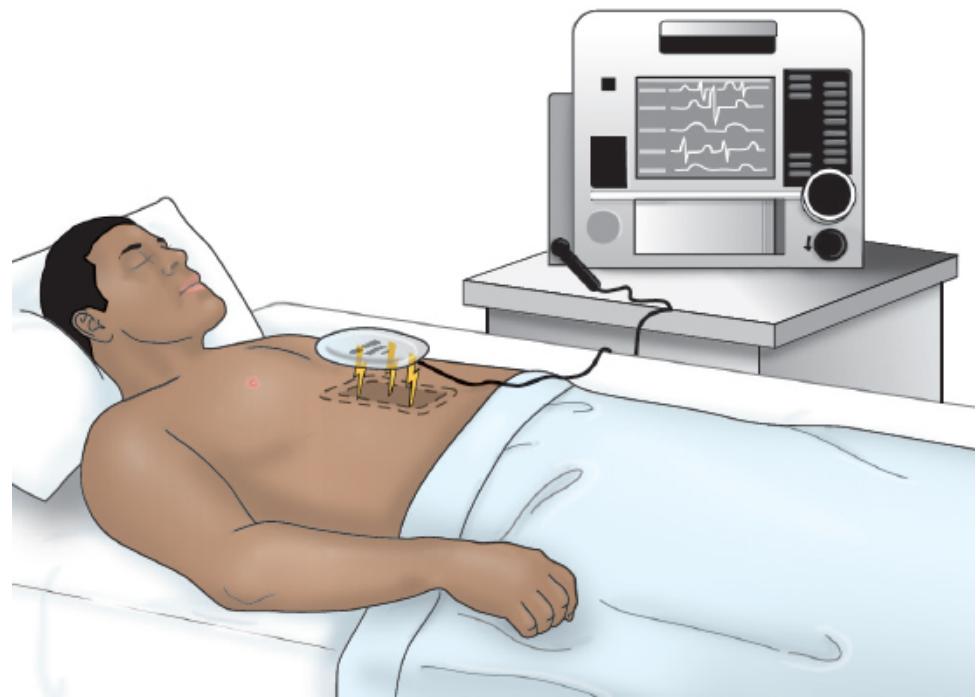
Permanent pacemaker generators are insulated to protect against body moisture and warmth and have filters that protect them from electrical interference from most household devices, motors, and appliances. Lithium cells are most commonly used; they last approximately 5 to 15 years, depending on the type of pacemaker, how it is programmed, and how often it is used. Most pacemakers have an elective replacement indicator (ERI), which is a signal that indicates when the battery is approaching depletion. The pacemaker continues to function for several months after the appearance of ERI to ensure that there is adequate time for a battery replacement. Although

some batteries are rechargeable, most are not. Because the battery is permanently sealed in the pacemaker, the entire generator must be replaced. To replace a failing generator of a transvenous pacemaker, the leads are disconnected, the old generator is removed, and a new generator is reconnected to the existing leads and reimplanted in the already existing subcutaneous pocket. Sometimes the leads are also replaced. When leadless pacemaker batteries signal that they must be replaced, a new system is simply implanted and the old battery is then disabled (Groner & Grippe, 2019). Battery replacement of both systems is usually performed using a local anesthetic. The patient usually can be discharged from the hospital the day of the procedure.

If a patient suddenly develops a bradycardia, is symptomatic but has a pulse, and is unresponsive to atropine, emergency pacing may be started with transcutaneous pacing, which most defibrillators are now equipped to perform. Some AEDs are able to do both defibrillation and transcutaneous pacing. Large pacing ECG electrodes (sometimes the same conductive pads used for cardioversion and defibrillation) are placed on the patient's chest and back. The electrodes are connected to the defibrillator, which is the temporary pacemaker generator (see [Fig. 22-26](#)). Because the impulse must travel through the patient's skin and tissue before reaching the heart, transcutaneous pacing can cause significant discomfort (burning sensation and involuntary muscle contraction) and is intended to be used only in emergencies for short periods of time. This type of pacing necessitates hospitalization. If the patient is alert, sedation and analgesia may be given. After transcutaneous pacing, the skin under the electrode should be inspected for erythema and burns. Transcutaneous pacing is not indicated for pulseless bradycardia (Link et al., 2015; Soar et al., 2018).

## Pacemaker Generator Functions

Because of the sophistication and wide use of pacemakers, a universal code has been adopted to provide a means of safe communication about their function. The coding is referred to as the NASPE-BPEG code because it is sanctioned by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group. The complete code consists of five letters; the fourth and fifth letters are used only with permanent pacemakers (Bernstein, Daubert, Fletcher, et al., 2002; Mulpuru, Madhavan, McLeod, et al., 2017; Tracy, Epstein, Darbar, et al., 2012; see [Chart 22-6](#)).



**Figure 22-26 •** Transcutaneous pacemaker with electrode pads connected to the anterior and posterior chest walls.

### Chart 22-6

## **North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group Code (NASPE-BPEG Code) for Pacemaker Generator Function**

- The first letter of the code identifies the chamber or chambers being paced (i.e., the chamber containing a pacing electrode). The letter characters for this code are A (atrium), V (ventricle), or D (dual, meaning both A and V).
- The second letter identifies the chamber or chambers being sensed by the pacemaker generator. Information from the electrode within the chamber is sent to the generator for interpretation and action by the generator. The letter characters are A (atrium), V (ventricle), D (dual), and O (indicating that the sensing function is turned off).
- The third letter of the code describes the type of response that will be made by the pacemaker to what is sensed. The letter characters used to describe this response are I (inhibited), T (triggered), D (dual— inhibited and triggered), and O (none). Inhibited response means that the response of the pacemaker is controlled by the activity of the patient's heart—that is, when the patient's heart beats, the pacemaker does not function, but when the heart does not beat, the pacemaker does function. In contrast, a triggered response means that the pacemaker responds (paces the heart) when it senses intrinsic heart activity.
- The fourth letter of the code is related to a permanent generator's ability to vary the heart rate. This ability is available in most current pacemakers. The possible letters are O, indicating no rate responsiveness, or R, indicating that the generator has rate modulation (i.e., the pacemaker has the ability to automatically adjust the pacing rate from moment to moment based on parameters, such as QT interval, physical activity, acid-base changes, body temperature, rate and depth of respirations, or oxygen saturation). A pacemaker with rate-responsive ability is capable of improving cardiac output during times of increased cardiac demand, such as exercise and decreasing the incidence of atrial fibrillation. All contemporary pacemakers have some type of sensor system that enables them to provide rate-adaptive pacing.
- The fifth letter of the code has two different indications: (1) that the permanent generator has multisite pacing capability with the letters A (atrium), V (ventricle), D (dual), and O (none); or (2) that the pacemaker has an antitachycardia function.
- Commonly, only the first three letters are used for a pacing code. An example of an NASPE-BPEG code is DVI:
  - D:** Both the atrium and the ventricle have a pacing electrode in place.
  - V:** The pacemaker is sensing the activity of the ventricle only.
  - I:** The pacemaker's stimulating effect is inhibited by ventricular activity—in other words, it does not create an impulse when the pacemaker

senses that the patient's ventricle is active.

Adapted from Bernstein, A. D., Daubert, J-C., Fletcher, R. D., et al. (2002). The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group. *Pacing and Clinical Electrophysiology*, 25(2), 260–264; Gillis, A. M., Russo, A. M., Ellenbogen, K. A., et al. (2012). HRS/ACCF expert consensus statement on pacemaker device and mode selection. *Journal of American College of Cardiology*, 60(7), 682–703; Mulpuru, S. K., Madhavan, M., McLeod, C. J., et al. (2017). Cardiac pacemakers: Function, troubleshooting, and management: Part 1 of a 2-Part Series. *Journal of the American College of Cardiology*, 69(2), 189–210.



**Figure 22-27 •** Pacemaker capture. **A.** Atrial pacemaker; note that each vertical pacemaker spike is followed by a P wave. **B.** Ventricular pacemaker; note that each vertical pacemaker spike is followed by a QRS complex. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (11th ed., Fig. 18-34A and B). Philadelphia, PA: Wolters Kluwer.

The pacemaker paces the atrium and then the ventricle when no ventricular activity is sensed for a period of time (the time is individually programmed into the pacemaker for each patient). A straight vertical line usually can be seen on the ECG when pacing is initiated. The line that represents pacing is called a *pacemaker spike*. The appropriate ECG complex should immediately follow the pacing spike; therefore, a P wave should follow an atrial pacing spike (see Fig. 22-27A) and a QRS complex should follow a ventricular pacing spike (see Fig. 22-27B). Because the impulse starts in a different place than the patient's normal rhythm, the QRS complex or P wave that responds to pacing looks different from the patient's normal ECG complex. *Capture* is a term used to denote that the appropriate complex followed the pacing spike.

The type of pacemaker generator and the settings selected depend on the patient's arrhythmia, underlying cardiac function, and age. Pacemakers are

generally set to sense and respond to intrinsic activity, which is called *on-demand pacing*. If the pacemaker is set to pace but not to sense, it is called a *fixed* or *asynchronous pacemaker*; this is written in pacing code as AOO or VOO. The pacemaker paces at a constant rate, independent of the patient's intrinsic rhythm. VOO pacing may indicate battery failure.

VVI (V, paces the ventricle; V, senses ventricular activity; I, paces only if the ventricles do not depolarize) pacing causes loss of AV synchrony and atrial kick, which may cause a decrease in cardiac output and an increase in atrial distention and venous congestion. Pacemaker syndrome, causing symptoms such as chest discomfort, shortness of breath, fatigue, activity intolerance, and orthostatic hypotension, is most common with VVI pacing (Mulpuru et al., 2017; Tracy et al., 2012). Atrial pacing and dual-chamber (right atrial and RV) pacing have been found to reduce the incidence of atrial fibrillation, ventricular dysfunction, and heart failure (Mulpuru et al., 2017; Tracy et al., 2012).

Single-chamber atrial pacing (AAI) or dual-chamber pacing (DDD) is recommended over VVI in patients with sinus node dysfunction, the most common cause of bradycardias requiring a pacemaker, and a functioning AV node (Mulpuru et al., 2017; Tracy et al., 2012). AAI pacing ensures synchrony between atrial and ventricular stimulation (and therefore contraction), as long as the patient has no conduction disturbances in the AV node. Dual-chamber pacemakers are recommended as the treatment for patients with AV conduction disturbances (Mulpuru et al., 2017; Tracy et al., 2012).

Synchronized biventricular pacing, also called *cardiac resynchronization therapy (CRT)*, is associated with improved mortality rates in patients with heart failure and in patients with left bundle branch block (Agrawal, 2018). Synchronized biventricular pacing features three leads: one for the right atrium; one for the right ventricle; and one for the left ventricle, usually placed in the left lateral wall. This therapy improves cardiac function, resulting in decreased heart failure symptoms and an improved quality of life. Biventricular pacing may be used with an ICD (Agrawal, 2018).

## Complications of Pacemaker Use

Complications associated with pacemakers relate to their presence within the body and improper functioning (see [Chart 22-7](#)). In the initial hours after a temporary or permanent pacemaker is inserted, the most common complication is dislodgment of the pacing electrode. Minimizing patient activity can help prevent this complication. If a temporary electrode is in place, the extremity through which the catheter has been advanced is immobilized. With a permanent pacemaker, the patient is instructed initially to restrict activity on the side of the implantation.

Leadless pacemakers are associated with fewer complications than transvenous pacemakers, including fewer infections, hematomas, lead dislodgement, and lead fracture. However, they provide only single-chamber RV pacing and do not feature concomitant defibrillator capabilities, limiting their usefulness (Groner & Grippe, 2019; Hayes, 2018).

The ECG is monitored very carefully to detect pacemaker malfunction. Improper pacemaker function, which can arise from failure in one or more components of the pacing system, is outlined in [Table 22-2](#). The following data should be noted on the patient's record: model of pacemaker, type of generator, date and time of insertion, location of pulse generator, stimulation threshold, and pacer settings (e.g., rate, energy output [mA], sensitivity [mV], and duration of interval between atrial and ventricular impulses [AV delay]). This information is important for identifying normal pacemaker function and diagnosing pacemaker malfunction.

### Chart 22-7

### Potential Complications from Insertion of a Pacemaker

- Local infection at the entry site of the leads for temporary pacing, or at the subcutaneous site for permanent generator placement. Prophylactic antibiotic and antibiotic irrigation of the subcutaneous pocket prior to generator placement has decreased the rate of infection to a minimal rate.
- Pneumothorax or hemothorax. The risk is reduced if cephalic vein cut down, contrast venography, or ultrasound guidance is utilized.
- Bleeding and hematoma at the lead entry sites for temporary pacing, or at the subcutaneous site for permanent generator placement. This usually can be managed with cold compresses and discontinuation of antiplatelet and antithrombotic medications.
- Ventricular ectopy and tachycardia from irritation of the ventricular wall by the endocardial electrode.
- Movement or dislocation of the lead placed transvenously (perforation of the myocardium).
- Phrenic nerve, diaphragmatic (hiccupping may be a sign), or skeletal muscle stimulation if the lead is dislocated or if the delivered energy (mA) is set high. The occurrence of this complication is avoided by testing during device implantation.
- Cardiac perforation resulting in pericardial effusion and, rarely, cardiac tamponade, which may occur at the time of implantation or months later. This condition can be recognized by the change in QRS complex morphology, diaphragmatic stimulation, or hemodynamic instability.
- Twiddler syndrome may occur when the patient manipulates the generator, causing lead dislodgement or fracture of the lead.
- Pacemaker syndrome (hemodynamic instability caused by ventricular pacing and the loss of AV synchrony).

Adapted from Link, M. S., Berkow, L. C., Kudenchuk, P. J., et al. (2015). Part 7: Adult advanced cardiovascular life support: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 132(18 suppl 2), S444–S464; Mulpuru, S. K., Madhavan, M., McLeod, C. J., et al. (2017). Cardiac pacemakers: Function, troubleshooting, and management: Part 1 of a 2-Part Series. *Journal of the American College of Cardiology*, 69(2), 189–210; Soar, J., Donnino, M. W., Maconochie, I., et al. (2018). 2018 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations summary. *Resuscitation*, 133, 194–206; Tracy, C. M., Epstein, A. E., Darbar, D., et al. (2012). ACCF/AHA/HRS focused update of the 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, 60(14), 1297–1313.

A patient experiencing pacemaker malfunction may develop bradycardia as well as signs and symptoms of decreased cardiac output (e.g., diaphoresis, orthostatic hypotension, syncope). The degree to which these symptoms become apparent depends on the severity of the malfunction, the patient's level of dependency on the pacemaker, and the patient's underlying condition. Pacemaker malfunction is diagnosed by analyzing the ECG. Manipulating the electrodes, changing the generator's settings, or replacing the pacemaker generator or leads (or both) may be necessary.

**TABLE 22-2** Assessing Pacemaker Malfunction

Problem	Possible Cause	Nursing Considerations
Loss of capture—complex does <i>not</i> follow pacing spike	Inadequate stimulus Lead dislodgement Lead wire fracture Catheter malposition Battery depletion Electronic insulation break Medication change Myocardial ischemia	Check security of all connections; increase milliamperage. Reposition extremity; turn patient to left side. Change battery. Change generator.
Undersensing—pacing spike occurs at preset interval despite the patient's intrinsic rhythm	Sensitivity too high Electrical interference (e.g., by a magnet) Faulty generator	Decrease sensitivity. Eliminate interference. Replace generator.
Oversensing—loss of pacing artifact; pacing does not occur at preset interval despite the lack of intrinsic rhythm	Sensitivity too low Electrical interference Battery depletion Change in medication	Increase sensitivity. Eliminate interference. Change battery.
Loss of pacing—total absence of pacing spikes	Oversensing Battery depletion Loose or disconnected wires Perforation	Change battery. Check security of all connections. Apply magnet over permanent generator. Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Contact physician.
Change in pacing QRS shape	Septal perforation	Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Contact physician.
Rhythmic diaphragmatic or chest wall twitching or hiccupping	Output too high Myocardial wall perforation	Decrease milliamperage. Turn pacer off. Contact physician at once. Monitor closely for decreased cardiac output.

ECG, electrocardiogram.

Adapted from Mulpuru, S. K., Madhavan, M., McLeod, C. J., et al. (2017). Cardiac pacemakers: Function, troubleshooting, and management: Part 1 of a 2-Part Series. *Journal of the American College of Cardiology*, 69(2), 189–210.

Inhibition of permanent pacemakers or reversion to asynchronous fixed rate pacing can occur with exposure to strong electromagnetic fields (electromagnetic interference [EMI]). However, pacemaker technology allows patients to safely use most household electronic appliances and devices (e.g., microwave ovens, electric tools). Gas-powered engines should be turned off before working on them. Objects that contain magnets (e.g., the earpiece of a phone, large stereo speakers, jewelry) should not be near the generator for longer than a few seconds. Patients are advised to place digital cellular phones at least 6 to 12 inches away from (or on the side opposite of) the pacemaker generator and not to carry them in a shirt pocket. Large electromagnetic fields, such as those produced by magnetic resonance imaging (MRI), radio and television transmitter towers and lines, transmission power lines (not the distribution lines that bring electricity into a home), and electrical substations may cause EMI. Patients should be cautioned to avoid such situations or to simply move farther away from the area if they experience dizziness or a feeling of rapid or irregular heartbeats (palpitations). Welding and the use of a chain saw should be avoided. If such tools are used, precautionary steps such as limiting the welding current to a 60- to 130-ampere range or using electric rather than gasoline-powered chain saws are advised.

In addition, the metal of the pacemaker generator may trigger store and library antitheft devices as well as airport and building security alarms; however, these alarm systems generally do not interfere with the pacemaker function. Patients should walk through them quickly and avoid standing in or near these devices for prolonged periods of time. The handheld screening devices used in airports may interfere with the pacemaker. Patients should be advised to ask security personnel to perform a hand search instead of using the handheld screening device. Patients also should be educated to wear or carry medical identification to alert personnel to the presence of the pacemaker.

## Pacemaker Surveillance

Remote monitoring technology is now routinely embedded in the pacemaker so that it replaces the need for frequent in-person follow-up cardiologist visits; this is also associated with improved survival (Costa, Yeung, Gilbert, et al., 2018). Remote monitoring systems in use include transtelephonic monitoring (use of an analog phone with transmission through a landline), inductive monitoring (use of a wand with transmission through a landline or a cellular connection), and remote wandless monitoring (use of wandless transmitter through radiofrequency) (Madhavan, Mulpuru, McLeod, et al., 2017). Remote monitoring allows pertinent information, such as ECG data, to be transmitted to the primary provider at the cardiology clinic. In addition, the pacemaker rate and other data concerning pacemaker function (e.g., generator setting, battery status, sensing function, lead integrity, pacing data, such as number of pacing

events) are obtained and evaluated by the cardiologist. This simplifies the diagnosis of a failing generator, reassures the patient, and improves management when the patient is physically remote from pacemaker testing facilities. A follow-up schedule is variable, and is dependent upon the patient's needs and the pacemaker in use; the system is setup for maintenance checks on an annual basis.

## Implantable Cardioverter Defibrillator

The **implantable cardioverter defibrillator (ICD)** is an electronic device that detects and terminates life-threatening episodes of tachycardia or fibrillation, especially those that are ventricular in origin. Patients at high risk of VT or ventricular fibrillation and who would benefit from an ICD are those who have survived sudden cardiac death, which usually is caused by ventricular fibrillation, or have experienced spontaneous, symptomatic VT (syncope secondary to VT) not due to a reversible cause (called a *secondary prevention intervention*). Patients with coronary artery disease who are 40 days postacute MI with moderate to severe LV dysfunction (ejection fraction less than or equal to 35%) are at risk of sudden cardiac death and therefore an ICD is indicated (called a *primary prevention intervention*). An ICD implantation is also recommended in patients who have been diagnosed with nonischemic dilated cardiomyopathy for at least 9 months and have functional NYHA Class II or III heart failure (Wilkoff, Fauchier, Stiles, et al., 2015) (see [Chapter 25, Table 25-1](#)) (Al-khatib et al., 2018; Ganz, 2019).



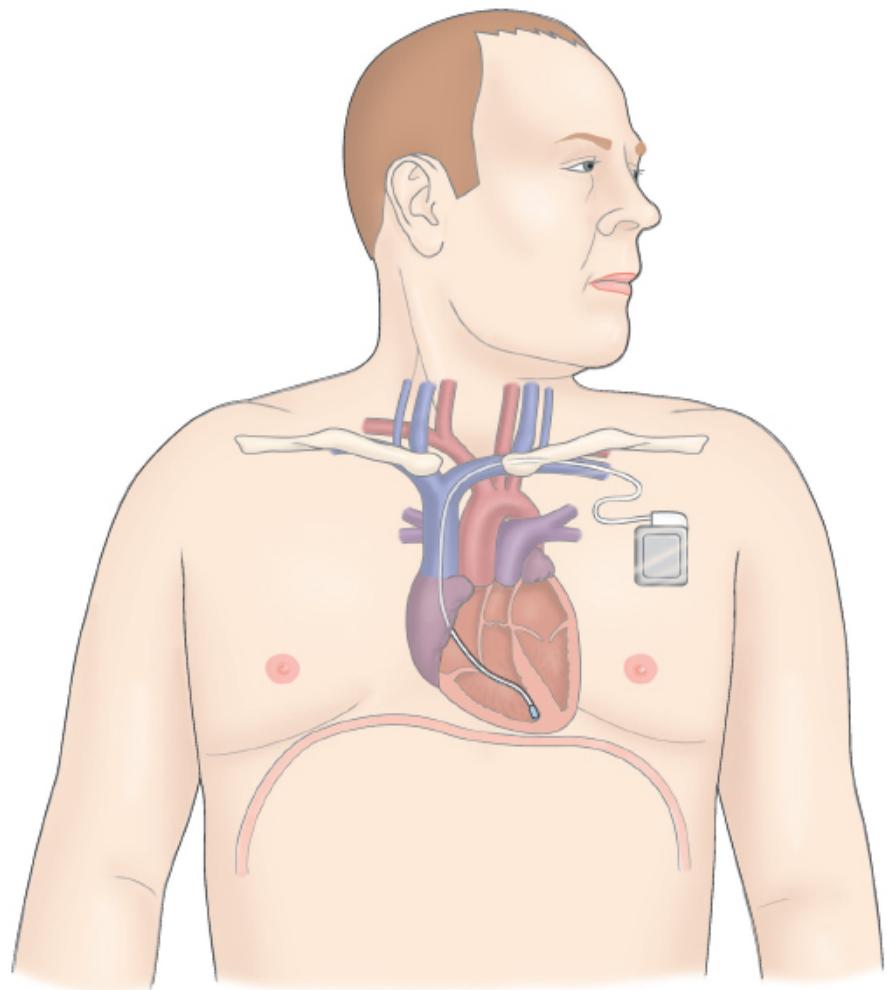
**Figure 22-28 •** The wearable cardioverter defibrillator vest.  
Courtesy of ZOLL LifeVest.

Because there may be a waiting period for ICD implantation, especially those postacute MI, patients who are at risk for sudden cardiac death may be prescribed a wearable vestlike automated defibrillator, which works just like an AED in that a shock is delivered less than a minute after a life-threatening rhythm is detected (National Heart, Lung, and Blood Institute [NHLBI], 2019; see Fig. 22-28). Prior to the delivery of the shock, the vest vibrates and issues an alarm to announce that a shock is imminent. The vest weighs about a pound, is worn under the patient's clothing, and is attached to a monitor with a battery that is worn in a holster or on a shoulder strap. The monitor automatically downloads information once a day, usually in the middle of the night. The vest must be worn at all times, even if the patient is admitted to the hospital and placed on an ECG monitor, and removed only when showering or bathing. The battery needs to be changed every day. Education is provided to

the patient by the device manufacturer. However, the nurse should assess the patient's understanding of the education provided and explore any issues that may prevent the patient from wearing it.

An ICD has a generator about the size of a pack of chewing gum that is implanted in a subcutaneous pocket, usually in the upper chest wall. An ICD also has at least an RV lead that is implanted transvenously and can sense intrinsic electrical activity and deliver an electrical impulse. The implantation procedure, postimplantation care, and length of hospital stay are much like those for insertion of a pacemaker (see [Fig. 22-29](#)).

ICDs are designed to respond to two criteria: a rate that exceeds a predetermined level and a change in the isoelectric line segments. When an arrhythmia occurs, rate sensors require a set duration of time to sense the arrhythmia. Then, the device automatically charges; after a second "look" confirms the arrhythmia, it delivers the programmed charge through the lead to the heart. The time from arrhythmia detection to electrical discharge depends on the charging time, which depends on the programmed energy level (Zipes et al., 2018). However, in an ICD that has the capability of providing atrial therapies, the device can be programmed to be activated by the patient, giving the patient time to activate the charge at a time and place of their choosing. The life of the lithium battery is about 9 years but varies depending on the use of the ICD. ICD surveillance is similar to that of the pacemaker; however, it includes stored endocardial ECGs as well as information about the number and frequency of shocks that have been delivered.



**Figure 22-29 •** The implantable cardioverter defibrillator consists of a generator and a sensing/pacing/defibrillating electrode.

Antiarrhythmic medication may be given with this technology to minimize the occurrence of a tachyarrhythmia and to reduce the frequency of ICD discharge.

Several types of devices are available. ICD, the generic name, is used as the abbreviation for these various devices. Each device offers a different delivery sequence, but all are capable of delivering high-energy (high-intensity) defibrillation to treat a tachycardia (atrial or ventricular).

Some ICDs can respond with (1) antitachycardia pacing, in which the device delivers electrical impulses at a fast rate in an attempt to disrupt the tachycardia, (2) low-energy (low-intensity) cardioversion, or (3) defibrillation; others may use all three techniques. Pacing is used to terminate tachycardias caused by a conduction disturbance called *reentry*, which is repetitive restimulation of the heart by the same impulse. An impulse or a series of impulses is delivered to the heart by the device at a fast rate to collide with and stop the heart's reentry conduction impulses, and therefore to stop the

tachycardia. Typically, ICDs also have pacemaker capability if the patient develops bradycardia, which sometimes occurs after treatment of the tachycardia. Usually, the mode is VVI.

The subcutaneous ICD is a more recently introduced therapeutic alternative to the conventional ICD. The main advantage to subcutaneous defibrillators is that the complications associated with vascular access are avoided. Patients without pacing indications, or antitachycardia pacing, or cardiac resynchronization are best candidates for this technology (Sideris, Archontakis, Gatzoulis, et al., 2017).

Which device is used and how it is programmed depend on the patient's arrhythmia(s). The device may be programmed differently for different arrhythmias (e.g., ventricular fibrillation, VT with a fast ventricular rate, and VT with a slow ventricular rate). As with pacemakers, there is an NASPE-BPEG code for communicating the functions of the ICDs (Bernstein et al., 2002; Wilkoff et al., 2015). The first letter represents the chamber or chambers shocked (O, none; A, atrium; V, ventricle; D, both atrium and ventricle). The second letter represents the chamber that can be antitachycardia paced (O, A, V, D, meaning the same as the first letter). The third letter indicates the method used by the generator to detect a tachycardia (E, electrogram; H, hemodynamics). The last letter represents the chambers that have antibradycardia pacing (O, A, V, D, meaning the same as the first and second letters of the ICD code).

Complications of ICD implantation are similar to those associated with pacemaker insertion. The primary potential complication is surgery-related infection; its risk increases with battery or lead replacement. A few complications are associated with the technical aspects of the equipment, like those of pacemakers, such as premature battery depletion and dislodged or fractured leads. Inappropriate delivery of ICD therapy, usually due to oversensing or atrial and sinus tachycardias with a rapid ventricular rate response, is the most frequent long-term complication. This requires reprogramming of the device.

## Nursing Management

After a permanent electronic device (pacemaker or ICD) is inserted, the patient's heart rate and rhythm are monitored by ECG. The device's settings are noted and compared with the ECG recordings to assess the device's function. For example, pacemaker malfunction is detected by examining the pacemaker spike and its relationship to the surrounding ECG complexes. In addition, cardiac output and hemodynamic stability are assessed to identify the patient's response to pacing and the adequacy of pacing. The appearance or increasing frequency of arrhythmia is observed and reported to the primary provider. If the patient has an ICD implanted and develops VT or ventricular

fibrillation, the ECG should be recorded to note the time between the onset of the arrhythmia and the onset of the device's shock or antitachycardia pacing.

The incision site where the generator was implanted is observed for bleeding, hematoma formation, or infection, which may be evidenced by swelling, unusual tenderness, drainage, and increased warmth. The patient may complain of continuous throbbing or pain. These symptoms are reported to the primary provider.

A chest x-ray is usually taken after the procedure and prior to discharge to document the position of leads in addition to ensuring that the procedure did not cause a pneumothorax. It is necessary to assess the function of the device throughout its lifetime and especially after changes in the patient's medication regimen. For example, antiarrhythmic agents, beta-blockers, and diuretics may increase the pacing threshold, whereas corticosteroids and alpha-adrenergic agents may decrease the pacing threshold; the opposite effect occurs when the patient is taken off these medications.

The patient is also assessed for anxiety, depression, or anger, which may be symptoms of ineffective coping with the implantation. In addition, the level of knowledge and education needs of the patient and family and the history of adherence to the therapeutic regimen should be identified. It is especially important to include the family when providing education and support.

In the peri- and postoperative phases, the nurse carefully observes the patient's responses to the device and provides the patient and family with further education as needed. The nurse also assists the patient and family in addressing concerns and in making decisions about self-care and lifestyle changes necessitated by the arrhythmia and resulting device implantation.

## Preventing Infection

The nurse changes the dressing as needed and inspects the insertion site for redness, swelling, soreness, or any unusual drainage. Any change in wound appearance, an increase in the patient's temperature, or an increase in the patient's white blood count should be reported to the primary provider.

## Promoting Effective Coping

The patient treated with an electronic device experiences not only lifestyle and physical changes but also emotional changes (Palese, Cracina, Purino, et al., 2019; see [Chart 22-8](#) Nursing Research Profile: Experiences of Patients Shocked by an Implantable Cardioverter Defibrillator). At different times during the healing process, the patient may feel angry, depressed, fearful, anxious, or a combination of these emotions. Although each patient uses individual coping strategies (e.g., humor, prayer, communication with a significant other) to manage emotional distress, some strategies may work better than others. Signs that may indicate ineffective coping include social

isolation, increased or prolonged irritability or depression, and difficulty in relationships.

To promote effective coping strategies, the nurse must recognize both the patient's and family's perceptions of the situation and their resulting emotional state and assist them to explore their reactions and feelings. Because of the unpredictable and possibly painful ICD discharge, patients with ICDs are most vulnerable to feelings of helplessness, leading to depression. The nurse can help the patient identify positive methods to deal with the actual or perceived limitations and manage any lifestyle changes needed. The nurse may help the patient identify changes (e.g., loss of ability to participate in contact sports), the emotional responses to the change (e.g., anger), and how the patient responds to that emotion (e.g., quickly becomes angry when talking with spouse). The nurse reassures the patient that these responses are normal and helps the patient identify realistic goals (e.g., develop interest in another activity) and develop a plan to attain these goals. The patient and family should be encouraged to talk about their experiences and emotions with each other and the health care team. The nurse may refer the patient and family to a hospital, community, or online support group. The nurse may also encourage the use of spiritual resources. Based on the patient's interest, the nurse also may educate the patient about easy-to-use stress reduction techniques (e.g., deep-breathing exercises, relaxation) to facilitate coping. Instructing the patient about the ICD may help the patient cope with changes that occur as a result of device implantation.

Chart 22-8



### NURSING RESEARCH PROFILE

## **Experiences of Patients Shocked by an Implantable Cardioverter Defibrillator**

Palese, A., Cracina, A., Purino, M., et al. (2019). The experiences of patients electrically shocked by an implantable cardioverter defibrillator: Findings from a descriptive qualitative study. *Nursing in Critical Care*. Retrieved on 7/9/2019 at: doi.org/10.1111/nicc.12424

### **Purpose**

Insertion rates for implantable cardioverter defibrillators (ICDs) have steadily risen. ICDs may be surgically implanted as a secondary preventive measure for survivors of ventricular arrhythmias or sudden cardiac death and as primary prevention for patients with preexisting select cardiac conditions. The ICD is proven to be effective in lowering mortality rates in sudden cardiac death; however, little has been explored regarding the patients' experiences as lived before, during, and after shocks from an ICD.

### **Design**

This study used a descriptive qualitative design. A convenience sample of adult patients with ICDs who reported at least one shock during their first follow-up visit post ICD implantation were eligible to participate. All patients were followed by a cardiology department in an academic hospital located in northern Italy. A total of 50 participants consented to enroll in this study. Each participant went through semi-structured face-to-face audiorecorded interviews. Content analysis methodology was used to analyze the data.

### **Findings**

The content analysis revealed four themes: *feeling surprised versus feeling altered by the changes inside me*; *living an intense, monodimensional experience versus living a multidimensional storm experience*; *facing the event alone versus being supported*; and *living a drama versus being used to it*. Most participants reported that they received shocks in their homes or in other community-based settings, many with family members present, and that they then presented to their local emergency department. Most participants could not recall the moments preceding the shocks, but did report feeling vague physical symptoms.

### **Nursing Implications**

Findings from this study suggest that ICD shocks can have a profound effect on patients' physical and emotional health. Nurses need to be vigilant to recognize patients' educational needs. Furthermore, nurses need to tailor appropriate, supportive interventions to improve patients' experiences living with an ICD. In particular, nurses can assist patients with ICDs in identifying pre-shock prodromal physical symptoms so that the ICD may be reprogrammed if indicated, or so that the doses of antiarrhythmic medications might be better titrated to prevent arrhythmias. Moreover, most

participants in this study received shocks outside the health care setting, and many of them were shocked in the presence of family members. Tailoring education programs to include significant others in identifying prodromal symptoms pre-shock and in seeking appropriate treatment post-shock may improve the quality of life of family members and patients with ICDs.

### Promoting Home, Community-Based, and Transitional Care

After device insertion, the patient's hospital stay is typically short (e.g., may be 1 day or less), and follow-up in an outpatient clinic, office, or device clinic is common. The patient's anxiety and feelings of vulnerability may interfere with the ability to learn information provided. The nurse needs to include caregivers in the education and provide printed materials for use by the patient and caregiver. The nurse establishes priorities for learning with the patient and caregiver. Education includes the importance of periodic device monitoring, promoting safety, surgical site care, and avoiding EMI (see [Chart 22-9](#)). In addition, the educational plan should include information about activities that are safe and those that may be dangerous. The nurse discusses with the patient and family what they have to do when a shock is delivered. The nurse may facilitate CPR training for the family.

Chart 22-9



### HOME CARE CHECKLIST

## Educating the Patient with an Implantable Cardiac Device

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of device implantation on physiologic functioning, ADLs, IADLs, self-image and roles, relationships (including sexuality), and spirituality.
- State changes in lifestyle (e.g., diet, activity, mobility/driving restrictions) necessary to maintain health.
- State the name, dose, side effects, frequency, and schedule for all medications.
- For patients with pacemakers, check pulse daily. Report *immediately* any sudden slowing or increasing of the pulse rate. This may indicate pacemaker malfunction.
- Avoid infection at the insertion site of the device:
- Leave the incision uncovered and observe it daily for redness, increased swelling, and heat.
- Take temperature at same time each day; report any increase.
- Avoid wearing tight, restrictive clothing that may cause friction over the insertion site.
- Initially avoid soaking in the tub and lotion, creams, or powders in the area of the device.
- Adhere to activity restrictions:
- Restrict movement of arm until incision heals; do not raise arm above head for 2 wks.
- Avoid heavy lifting for a few weeks.
- Discuss safety of activities (e.g., driving) with a primary provider.
- Recognize that although it may take up to 2–3 wks to resume normal activities, physical activity does not usually have to be curtailed, with the exception of contact sports.
- Electromagnetic interference: Understand the importance of the following:
- Avoid large magnetic fields, such as those created by MRI, large motors, arc welding, and electrical substations. Magnetic fields may deactivate the device, negating its effect on an arrhythmia.
- At security gates at airports, government buildings, or other secured areas, show identification card and request a hand (not handheld device) search. Obtain and carry a letter from the primary provider about this requirement.
- Some electrical and small motor devices, as well as products that contain magnets (e.g., cellular phones), may interfere with the functioning of the cardiac device if the electrical device is placed very close to it. Avoid leaning directly over large electrical devices or motors, or ensure that contact is of brief duration; place cellular phone on opposite side of cardiac device.

- Household appliances (e.g., microwave ovens) should not cause any concern.
- Describe precautions and safety measures to be used:
- Describe what to do if symptoms occur, and notify the primary provider if any discharges seem unusual.
- Maintain a log that records discharges of an ICD. Record events that precipitate the sensation of shock. This provides important data for the primary provider to use in readjusting the medical regimen.
- Encourage family members to attend a cardiopulmonary resuscitation class.
- Call 911 for emergency assistance if feeling of dizziness occurs.
- Wear medical identification (e.g., MedicAlert) that includes primary provider information.
- Avoid frightening family or friends with unexpected shocks from an ICD, which will not harm them. Inform family and friends that in the event they are in contact with the patient when a shock is delivered, they may also feel the shock. It is especially important to warn sexual partners that this may occur.
- Carry medical identification with the primary provider's name, type and model number of the device, manufacturer's name, and hospital where device was inserted.
- Identify community resources for peer and caregiver/family support.
- Adhere to appointments for follow-up care that are scheduled to monitor the electronic performance of the cardiac device. This is especially important during the first month after implantation and near the end of the battery life. Remember to take the log of ICD discharges to review with the primary provider.
- Identify the need for health promotion, disease prevention, and screening activities.

ADL, activities of daily living; IADL, instrumental activities of daily living; ICD, implantable cardioverter defibrillator.

## CRITICAL THINKING EXERCISES

**1  ebp** Your uncle, who has a history of alcohol abuse, calls to tell you that his primary provider told him that he has atrial fibrillation. He says that he has been prescribed warfarin and that he really doesn't want to take it because he heard that it is an ingredient in "rat poison." What is the strength of the evidence that supports your uncle's continued adherence to warfarin therapy? What are the risks involved should he decide that he will not take this drug as prescribed?

**2  pq** You work as a staff nurse on a surgical gynecology unit. A 55-year-old woman assigned to your care is one day post total abdominal hysterectomy. On your initial rounds, the patient is resting comfortably in bed with moderate pain, heart rate of 65 bpm, blood pressure of 120/70 mm Hg, and respiratory rate of 20 breaths per minute. Four hours later, you enter the patient's room and she is sleeping with a heart rate of 98 bpm, blood pressure of 110/60 mm Hg, and respiratory rate of 16 breaths per minute. What are the possible causes of this increase in heart rate? What are your assessment priorities?

**3  ipc** It is the start of the 12-hour day shift on the cardiovascular unit where you work. A 45-year-old patient who was admitted last evening with a MI is now suddenly experiencing frequent PVCs and a four-beat run of ventricular tachycardia. The patient has remained hemodynamically stable during these episodes. You are preparing for the morning huddle with team members that include unlicensed assistive personnel and attending health care providers. How will you facilitate this interprofessional discussion concerning your patient's PVCs and ventricular tachycardia?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference, consensus statement, or guideline.

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## Resources

- American Association of Critical-Care Nurses, [www.aacn.org](http://www.aacn.org)  
American Association of Heart Failure Nurses (AAHFN), [www.aahfn.org](http://www.aahfn.org)  
American College of Cardiology (ACC), [www.acc.org](http://www.acc.org)  
American Heart Association, National Center, [www.heart.org](http://www.heart.org)  
Heart Rhythm Society, [www.hrsonline.org](http://www.hrsonline.org)  
National Institutes of Health, National Heart, Lung, Blood Institute, Health Information Center, [www.nhlbi.nih.gov](http://www.nhlbi.nih.gov)

# 23 Management of Patients with Coronary Vascular Disorders

## LEARNING OUTCOMES

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

1. Describe the pathophysiology, clinical manifestations, and treatment of coronary vascular disorders including coronary atherosclerosis, angina pectoris, and myocardial infarction.
2. Use the nursing process as a framework for care of the patient with angina pectoris, with acute coronary syndrome, or who has undergone cardiac surgery.
3. Describe percutaneous coronary interventional and coronary artery revascularization procedures.
4. Identify the nursing care of a patient who has had a percutaneous coronary interventional procedure for treatment of coronary artery disease.

## NURSING CONCEPT

Perfusion

## GLOSSARY

**acute coronary syndrome (ACS):** signs and symptoms that indicate unstable angina or acute myocardial infarction

**angina pectoris:** chest pain brought about by myocardial ischemia

**atheroma:** fibrous cap composed of smooth muscle cells that forms over lipid deposits within arterial vessels and protrudes into the lumen of the vessel, narrowing the lumen and obstructing blood flow; also called *plaque*

**atherosclerosis:** abnormal accumulation of lipid deposits and fibrous tissue within arterial walls and the lumen

**contractility:** ability of the cardiac muscle to shorten in response to an electrical impulse

**coronary artery bypass graft (CABG):** a surgical procedure in which a blood vessel from another part of the body is grafted onto the occluded coronary artery below the occlusion in such a way that blood flow bypasses the blockage

**high-density lipoprotein (HDL):** a protein-bound lipid that transports cholesterol to the liver for excretion in the bile; composed of a higher proportion of protein to lipid than low-density lipoprotein; exerts a beneficial effect on the arterial wall

**ischemia:** insufficient tissue oxygenation

**low-density lipoprotein (LDL):** a protein-bound lipid that transports cholesterol to tissues in the body; composed of a lower proportion of protein to lipid than high-density lipoprotein; exerts a harmful effect on the arterial wall

**metabolic syndrome:** a cluster of metabolic abnormalities including insulin resistance, obesity, dyslipidemia, and hypertension that increase the risk of cardiovascular disease

**myocardial infarction (MI):** death of heart tissue caused by lack of oxygenated blood flow

**percutaneous coronary intervention (PCI):** a procedure in which a catheter is placed in a coronary artery, and one of several methods is employed to reduce blockage within the artery

**percutaneous transluminal coronary angioplasty (PTCA):** a type of percutaneous coronary intervention in which a balloon is inflated within a coronary artery to break an atheroma and open the vessel lumen, improving coronary artery blood flow

**stent:** a metal mesh that provides structural support to a coronary vessel, preventing its closure

**sudden cardiac death:** abrupt cessation of effective heart activity

**thrombolytic:** a pharmacologic agent that breaks down blood clots; alternatively referred to as a fibrinolytic

**troponin:** a cardiac muscle biomarker; measurement is used as an indicator of heart muscle injury

Cardiovascular disease is the leading cause of death in the United States for men and women of all racial and ethnic groups (Arnett, Blumenthal, Albert, et al., 2019).

Research related to the identification of and treatment for cardiovascular disease includes all segments of the population affected by cardiac conditions, including women, children, and people of diverse racial and ethnic backgrounds. The results of ongoing research are used by nurses to identify specific prevention and treatment strategies in these populations.

## Coronary Artery Disease



Coronary artery disease (CAD) is the most prevalent type of cardiovascular disease in adults. For this reason, nurses must recognize various manifestations of coronary artery conditions and evidence-based methods for assessing, preventing, and treating these disorders.

### Coronary Atherosclerosis

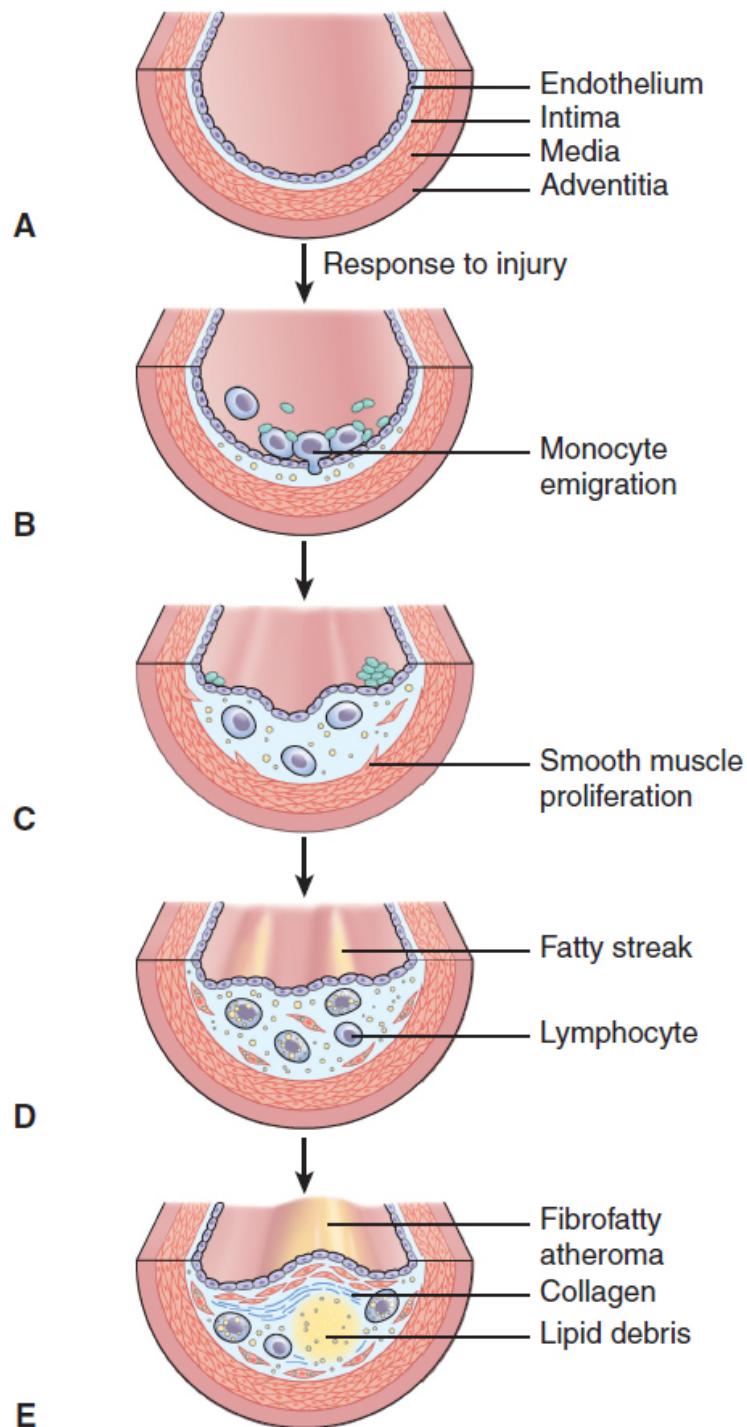
The most common cause of cardiovascular disease in the United States is **atherosclerosis**, an abnormal accumulation of lipid, or fatty substances, and fibrous tissue in the lining of arterial blood vessel walls. These substances block and narrow the coronary vessels in a way that reduces blood flow to the myocardium. Atherosclerosis involves a repetitious inflammatory response to injury of the artery wall and subsequent alteration in the structural and biochemical properties of the arterial walls. New information that relates to the development of atherosclerosis has increased the understanding of treatment and prevention of this progressive and potentially life-threatening process.

### Pathophysiology

The inflammatory response involved with the development of atherosclerosis begins with injury to the vascular endothelium and progresses over many years (Norris, 2019). The injury may be initiated by smoking or tobacco use, hypertension, hyperlipidemia, and other factors. The endothelium undergoes changes and stops producing the normal antithrombotic and vasodilating agents. The presence of inflammation attracts inflammatory cells, such as macrophages. The macrophages ingest lipids, becoming “foam cells” that transport the lipids into the arterial wall. Some of the lipid is deposited on the arterial wall, forming fatty streaks. Activated macrophages also release biochemical substances that can further damage the endothelium by contributing to the oxidation of low-density lipoprotein (LDL). The oxidized LDL is toxic to the endothelial cells and fuels progression of the atherosclerotic process (Norris, 2019).

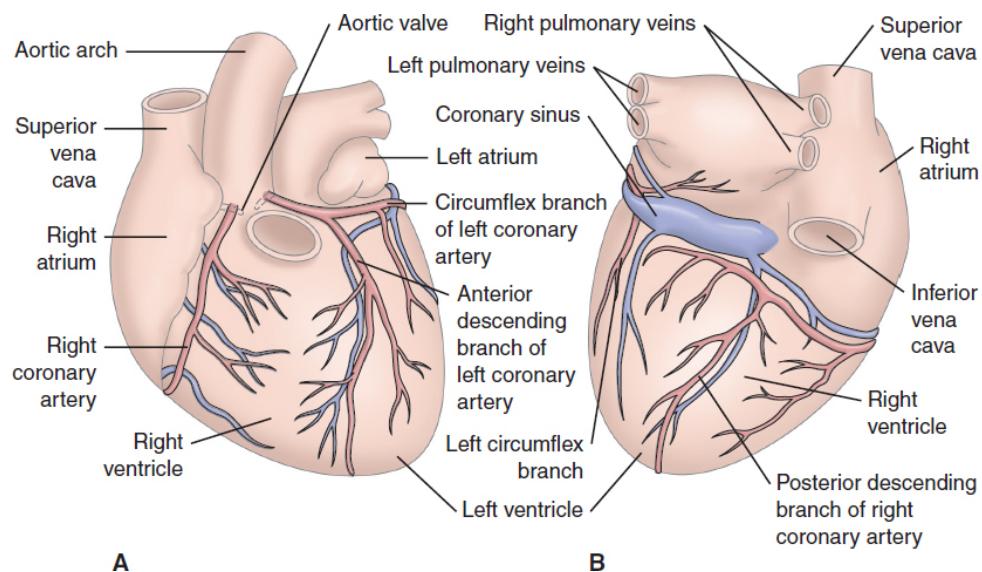
Following the transport of lipid into the arterial wall, smooth muscle cells proliferate and form a fibrous cap over a core filled with lipid and inflammatory infiltrate. These deposits, called **atheromas**, or plaques, protrude into the lumen of the vessel, narrowing it and obstructing blood flow (see Fig. 23-1). Plaque may be stable or unstable, depending on the degree of inflammation and thickness of the fibrous cap. If the fibrous cap over the plaque is thick and the lipid pool remains

relatively stable, it can resist the stress of blood flow and vessel movement. If the cap is thin and inflammation is ongoing, the lesion becomes what is called *vulnerable plaque*. At this point, the lipid core may grow, causing the fibrous plaque to rupture. A ruptured plaque attracts platelets and causes thrombus formation. A thrombus may then obstruct blood flow, leading to acute coronary syndrome (ACS), which may result in an acute **myocardial infarction (MI)**. When an MI occurs, a portion of the heart muscle no longer receives blood flow and becomes necrotic.



**Figure 23-1 • A, B.** Atherosclerosis begins as monocytes and lipids enter the intima of an injured vessel. Smooth muscle cells proliferate within the vessel wall (**C**), contributing to the development of fatty accumulations and atheroma (**D**). As the plaque enlarges, the vessel narrows and blood flow decreases (**E**). The plaque may rupture and a thrombus might form, obstructing blood flow.

The anatomic structure of the coronary arteries makes them particularly susceptible to atherosclerosis. As Figure 23-2 shows, the three major coronary arteries have multiple branches. Atherosclerotic lesions most often form where the vessels branch and with turbulent blood flow, suggesting a hemodynamic component is involved in their formation (Norris, 2019). Although heart disease is most often caused by atherosclerosis of the coronary arteries, other phenomena may also decrease blood flow to the heart. Examples include vasospasm (sudden constriction or narrowing) of a coronary artery and profound hypotension.



**Figure 23-2 •** The coronary arteries supply the heart muscle with oxygenated blood, adjusting the flow according to metabolic needs. **A.** Anterior view of the heart. **B.** Posterior view of heart.

## Clinical Manifestations

CAD produces symptoms and complications according to the location and degree of narrowing of the arterial lumen, thrombus formation, and obstruction of blood flow to the myocardium. This impediment to blood flow is usually progressive, causing an inadequate blood supply that deprives the cardiac muscle cells of oxygen needed for their survival. The condition is known as **ischemia**. **Angina pectoris** refers to chest pain that is brought about by myocardial ischemia. Angina pectoris usually is caused by significant coronary atherosclerosis. If the decrease in blood supply is great enough, of long enough duration, or both, irreversible damage and death of myocardial cells may result. Over time, irreversibly damaged myocardium undergoes degeneration and is replaced by scar tissue, causing various degrees of myocardial dysfunction. Significant myocardial damage may result in persistently low cardiac output and heart failure where the heart cannot support the body's needs for blood. A decrease in blood supply from CAD may cause the heart to abruptly stop beating; this is known as **sudden cardiac death** (see Chapter 25 for further discussion on CPR).

The most common manifestation of myocardial ischemia is the onset of chest pain. However, the classic epidemiologic study of the people in Framingham,

Massachusetts, showed that nearly 15% of men and women who had coronary events, which included unstable angina, MIs, or sudden cardiac death events, were totally asymptomatic prior to the coronary event (Kannel, 1986). Patients with myocardial ischemia may present to an emergency department (ED) or clinic with a variety of symptoms other than chest pain. Some complain of epigastric distress and pain that radiates to the jaw or left arm. Patients who are older or have a history of diabetes or heart failure may report shortness of breath. Many women have been found to have atypical symptoms, including indigestion, nausea, palpitations, and numbness (Davis, 2017). Prodromal symptoms may occur (e.g., angina a few hours to days before the acute episode), or a major cardiac event may be the first indication of coronary atherosclerosis.

## Risk Factors

Epidemiologic studies point to several factors that increase the probability that a person will develop heart disease. Major risk factors are listed in [Chart 23-1](#). Although many people with CAD have one or more risk factors, some do not have classic risk factors. Elevated **low-density lipoprotein (LDL)**, also known as bad cholesterol, is a well-known risk factor and the primary target of cholesterol-lowering therapy. People at the highest risk for having a cardiac event are those with known CAD or those with diabetes, peripheral arterial disease, abdominal aortic aneurysm, or carotid artery disease. The latter diseases are referred to as CAD risk equivalents, because patients with these diseases have the same risk for a cardiac event as patients with CAD. The likelihood of having a cardiac event is also affected by factors, such as age, gender, systolic blood pressure, smoking history, level of total cholesterol, and level of **high-density lipoprotein (HDL)**, also known as good cholesterol. The Framingham Risk Calculator is a tool commonly used to estimate the risk for having a cardiac event within the next 10 years (Grundy, Stone, Bailey, et al., 2018). This tool is designed for adults 20 years and older. The calculation is performed using the individual's risk factor data, including age, gender, total cholesterol, HDL, smoking status, systolic blood pressure, and need for antihypertensive medication.

In addition, a cluster of metabolic abnormalities known as **metabolic syndrome** has emerged as a major risk factor for cardiovascular disease (Grundy et al., 2018). A diagnosis of this syndrome is made when a patient has three of the following five risk factors:

- Enlarged waist circumference (greater than 35.4 inches in males, greater than 31.4 inches in females)
- Elevated triglycerides (greater than or equal to 175 mg/dL, or currently on drug treatment for elevated triglycerides)
- Reduced HDL (less than 40 mg/dL in males, less than 50 mg/dL in females, or currently on drug treatment for reduced HDL)
- Hypertension (systolic blood pressure greater than or equal to 130 mm Hg and/or diastolic blood pressure greater than or equal to 80 mm Hg on an average of two to three measurements obtained on two to three separate occasions, or currently on antihypertensive drug treatment for a history of hypertension)

Chart 23-1



## RISK FACTORS

### Coronary Artery Disease

A nonmodifiable risk factor is a circumstance over which a person has no control. A modifiable risk factor is one over which a person may exercise control, such as by changing a lifestyle or personal habit or by using medication. A risk factor may operate independently or in tandem with other risk factors. The more risk factors a person has, the greater the likelihood of coronary artery disease (CAD). Those at risk are advised to seek regular medical examinations and to engage in heart-healthy behavior (a deliberate effort to reduce the number and extent of risks).

#### Nonmodifiable Risk Factors

Family history of CAD (first-degree relative with cardiovascular disease at 55 years of age or younger for men and at 65 years of age or younger for women)

Increasing age (more than 45 years for men; more than 55 years for women)

Gender (men develop CAD at an earlier age than women)

Race (higher incidence of heart disease in African Americans than in Caucasians)

History of premature menopause (before age 40) and history of pregnancy-associated disorders such as preeclampsia

Primary hypercholesterolemia (a genetic condition resulting in elevated LDL)

#### Modifiable Risk Factors

Hyperlipidemia

Tobacco use

Hypertension

Diabetes

Metabolic syndrome

Obesity

Physical inactivity

Chronic inflammatory conditions (e.g., rheumatoid arthritis, lupus, HIV/AIDS)

Chronic kidney disease

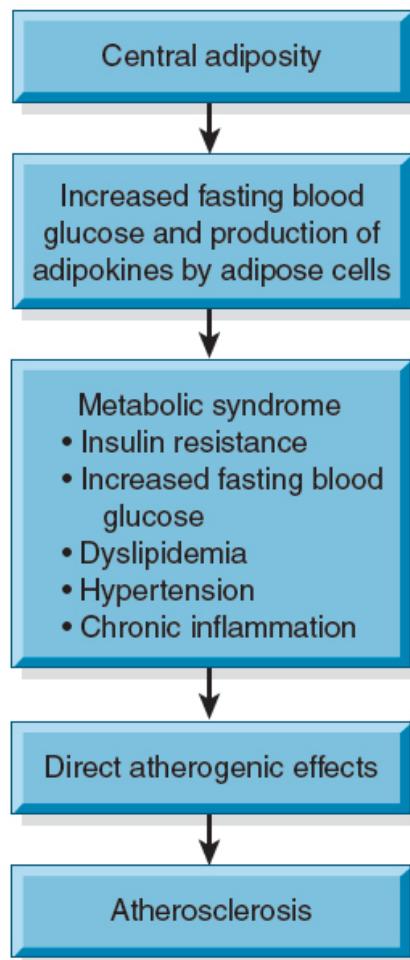
Adapted from Arnett, D. K., Blumenthal, R. S., Albert, M. A., et al. (2019). ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease. *Journal of the American College of Cardiology*, 74(10), e177–e232.

- Elevated fasting glucose (greater than or equal to 100 mg/dL on two separate occasions, or current drug treatment for elevated glucose)

Many people with type 2 diabetes fit this clinical picture. Theories suggest that in patients with obesity, excessive adipose tissue may secrete mediators that lead to metabolic changes. Adipokines (adipose tissue cytokines), free fatty acids, and other substances are known to modify insulin action and contribute to atherogenic changes in the cardiovascular system (see [Fig. 23-3](#)).

C-reactive protein (CRP) is known to be an inflammatory marker for cardiovascular risk, including acute coronary events and stroke. The liver produces CRP in response to a stimulus such as tissue injury, and high levels of this protein may occur in people with diabetes and those who are likely to have an acute coronary event (Norris, 2019). To determine overall cardiovascular risk, clinicians may view high sensitivity C-reactive protein (hs-CRP) test results together with other screening tools such as measurements of lipid levels.

## Physiology/Pathophysiology



**Figure 23-3 •** Pathophysiology of cardiovascular disease in metabolic syndrome. Central adiposity plays a major role in the development of metabolic syndrome. Adipokines released from fat cells along with other hormones and metabolites are thought to contribute to the development of metabolic abnormalities. The eventual effect of these processes is the promotion of atherosclerosis.

## Prevention

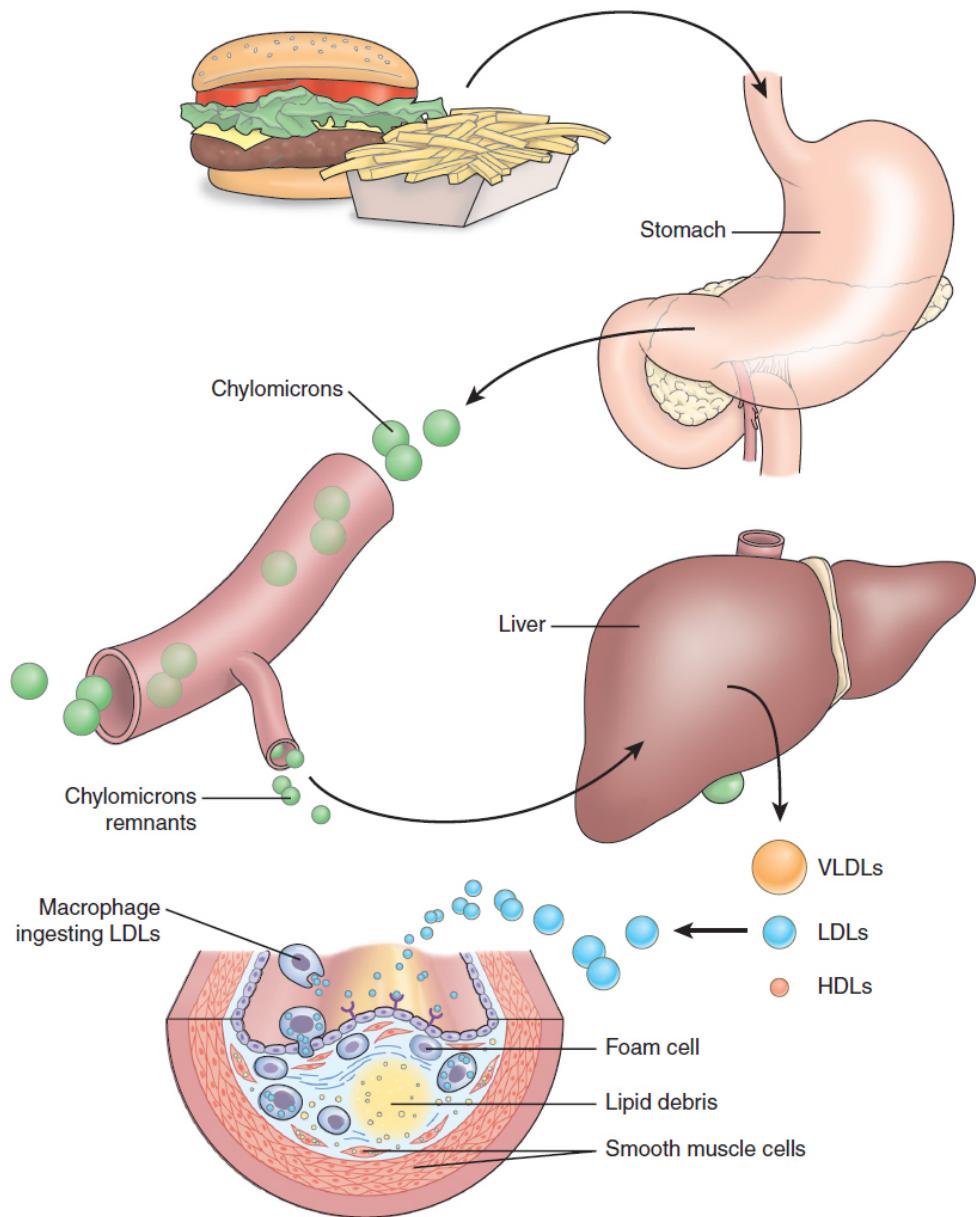
Four modifiable risk factors—cholesterol abnormalities, tobacco use, hypertension, and diabetes—are established risk factors for CAD and its complications. As a result, they receive much attention in health promotion programs.

### Controlling Cholesterol Abnormalities

The association of a high blood cholesterol level with heart disease is well established, and the metabolism of fats is known to be an important contributor to the development of heart disease. Fats, which are insoluble in water, are encased in

water-soluble lipoproteins that allow them to be transported within the circulatory system. The various lipoproteins are categorized by their protein content, which is measured in density. The density increases when more protein is present. Four elements of fat metabolism—total cholesterol, LDL, HDL, and triglycerides—are known to affect the development of heart disease. Cholesterol is processed by the gastrointestinal (GI) tract into lipoprotein globules called *chylomicrons*. These are reprocessed by the liver as lipoproteins (see Fig. 23-4). This is a physiologic process necessary for the formation of lipoprotein-based cell membranes and other important metabolic processes. When an excess of LDL is produced, LDL particles adhere to receptors in the arterial endothelium. Here, macrophages ingest them, contributing to plaque formation.

## Physiology/Pathophysiology



**Figure 23-4 • Lipoproteins and the development of atherosclerosis.** As dietary cholesterol and saturated fat are processed by the gastrointestinal tract, chylomicrons enter the blood. They are broken down into chylomicron remnants in the capillaries. The liver processes them into lipoproteins. When these are released into the circulation, excess low-density lipoproteins (LDLs) adhere to receptors on the intimal wall. Macrophages also ingest LDLs and transport them into the vessel wall, beginning the process of plaque formation. HDLs, high-density lipoproteins; VLDLs, very-low-density lipoproteins.

The American College of Cardiology and the American Heart Association (ACC/AHA) have developed clinical practice guidelines on the treatment of blood cholesterol to reduce cardiovascular risk in adults (Grundy et al., 2018). These guidelines address primary prevention (preventing the occurrence of CAD) and secondary prevention (preventing the progression of CAD). All adults 20 years and older should have a fasting lipid profile (total cholesterol, LDL, HDL, and triglycerides) performed at least once every 5 years, and more often if the profile is abnormal. Patients who have had an acute event (e.g., MI), a percutaneous coronary intervention (PCI), or a coronary artery bypass graft (CABG) require assessment of their LDL cholesterol level within a few months of the event or procedure, because LDL levels may be low immediately after the acute event or procedure. Subsequently, lipids should be monitored every 4 to 12 weeks until the desired level is achieved and then every 3 to 12 months as needed (Grundy et al., 2018). A fasting lipid profile should demonstrate the following values (Stone, Robinson, Lichtenstein, et al., 2014):

- LDL cholesterol less than 100 mg/dL (less than 70 mg/dL for very high risk patients)
- Total cholesterol less than 200 mg/dL
- HDL cholesterol greater than 40 mg/dL for males and greater than 50 mg/dL for females
- Triglyceride less than 150 mg/dL

LDL is the target of current therapy because of its strong association with advancing CAD. The total cholesterol level is also a clear predictor of coronary events. HDL is known as good cholesterol because it transports other lipoproteins such as LDL to the liver, where they can be degraded and excreted. Because of this, a high HDL level is a strong negative risk factor for heart disease (i.e., it protects against heart disease).

Triglyceride is made up of fatty acids and is transported through the blood by a lipoprotein. Although an elevated triglyceride level (more than 200 mg/dL) may be genetic in origin, it also can be caused by obesity, physical inactivity, excessive alcohol intake, high-carbohydrate diets, diabetes, kidney disease, and certain medications, such as oral contraceptives and corticosteroids.



### Concept Mastery Alert

It is important to remember the different types of cholesterol and the role of each as a risk factor for heart disease. HDL is the “good cholesterol,” and higher levels are better; LDL is the “bad cholesterol,” and lower levels are better.

## Dietary Measures

Adults who need to lower LDL (and blood pressure) should consider the AHA’s diet recommendations or the Mediterranean diet, which are reported to reduce mortality from cardiovascular disease (Franquesa, Pujol-Busquets, García-Fernández, et al., 2019). Both eating plans provide similar key elements: an emphasis on plant foods (fruits, vegetables, whole-grain breads or other forms of cereals, beans, nuts, and seeds), minimally processed foods, seasonally fresh foods, inclusion of fish, and minimal intake of red meat. Individuals needing to lower LDL and blood pressure

should also limit the intake of sweets and sugar sweetened beverages (Arnett et al., 2019). Adopting a strict vegetarian diet can significantly reduce blood lipids, blood glucose, body mass index, and blood pressure; however, this type of intensive dietary program may not be acceptable to all patients who need to modify risk factors. Referral to a dietitian can help patients in following a diet that is appropriate.

Many resources are available to assist people in controlling their cholesterol levels. The National Heart, Lung, and Blood Institute (NHLBI) and its National Cholesterol Education Program (NCEP), the AHA, and the American Diabetes Association (ADA), as well as CAD support groups and reliable Internet sources, are a few examples of the available resources (see Resources section at the end of this chapter). Cookbooks and recipes that include the nutritional contents of foods can be included as resources for patients. Dietary control has been made easier because food manufacturers are required to provide nutritional data on product labels. The label information of interest to a person attempting to eat a heart-healthy diet is as follows: serving size (expressed in household measures), amount of total fat per serving, amount of saturated fat and trans fat per serving, amount of cholesterol per serving, and amount of fiber per serving.

## Physical Activity

Management of an elevated triglyceride level focuses on weight reduction and increased physical activity. Regular, moderate physical activity increases HDL levels and reduces triglyceride levels, decreasing the incidence of coronary events and reducing overall mortality risk. The goal for most adults is to engage in moderate-intensity aerobic activity of at least 150 minutes per week or vigorous-intensity aerobic activity of at least 75 minutes per week, or an equivalent combination (Arnett et al., 2019). In addition, adults should engage in muscle-strengthening activities on 2 or more days each week that work all major muscle groups. The nurse helps the patient to set realistic goals for physical activity. For example, inactive patients can start with activity that lasts 3 minutes, such as parking farther from a building to increase daily walking time. Patients should be instructed to engage in an activity or variety of activities that interest them to maintain motivation. They should also be taught to exercise to an intensity that does not preclude their ability to talk; if they cannot have a conversation while exercising, they should slow down or switch to a less intensive activity. When the weather is hot and humid, patients should exercise during the early morning, or indoors, and wear loose-fitting clothing. When the weather is cold, they should layer clothing and wear a hat. Patients should stop any activity if chest pain, unexpected shortness of breath, dizziness, lightheadedness, or nausea occurs.

## Medications

If diet alone cannot normalize serum cholesterol levels, medications can have a synergistic effect with the prescribed diet and control cholesterol levels (see [Table 23-1](#)). Lipid-lowering medications can reduce CAD mortality in patients with elevated lipid levels and in at-risk patients with normal lipid levels (Grundy et al., 2018). The various types of lipid-lowering agents affect the lipid components somewhat differently; these types include 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) (or statins), fibrin acids (or fibrates), bile acid sequestrants (or resins),

cholesterol absorption inhibitors, and proprotein convertase subtilisin-kexin type 9 (PCSK9) agents. Because of their high cost, PCSK9 agents are prescribed on a limited basis, but may be considered for those at high cardiovascular risk or who have familial hypercholesterolemia (Grundy et al., 2018).

Before starting statin therapy, the provider and patient should discuss risk factors, adherence to a healthy lifestyle, benefits of risk-reduction, the potential of adverse effects and drug–drug interactions, as well as patient preferences for an individualized treatment plan (Grundy et al., 2018).

### Promoting Cessation of Tobacco Use

Tobacco use contributes to the development and severity of CAD in at least three ways:

- Nicotinic acid in tobacco triggers the release of catecholamines, which raise the heart rate and blood pressure (Frandsen & Pennington, 2021). Nicotinic acid can also cause the coronary arteries to constrict. These effects lead to an increased risk of CAD and sudden cardiac death.
- Tobacco use can increase the oxidation of LDL, damaging the vascular endothelium (Lee, Ong, Zhou, et al., 2019). This increases platelet adhesion and leads to a higher probability of thrombus formation.
- Inhalation of smoke increases the blood carbon monoxide level and decreases the supply of oxygen to the myocardium (Frandsen & Pennington, 2021). Hemoglobin, the oxygen-carrying component of blood, combines more readily with carbon monoxide than with oxygen. Myocardial ischemia and reduced contractility can result.

**TABLE 23-1**

## Select Medications that Affect Lipoprotein Metabolism

Medications	Therapeutic Effects	Considerations
<b>HMG-CoA Reductase Inhibitors (Statins)</b>		
Atorvastatin	↓ Total cholesterol	Frequently given as initial therapy for significantly elevated cholesterol and LDL levels
Simvastatin	↓ LDL	Myalgia and arthralgia are common adverse effects
Rosuvastatin	↑ HDL ↓ TGs Inhibit enzyme involved in lipid synthesis (HMG-CoA) Favorable effects on vascular endothelium, including anti-inflammatory and antithrombotic effects	Myopathy and possible rhabdomyolysis are potential serious effects Monitor liver function tests Contraindicated in liver disease Check for drug interactions Indication for use now includes ACS and stroke Administer in evening
<b>Fibric Acids (Fibrates)</b>		
Fenofibrate	↑ HDL	Adverse effects include diarrhea, flatulence, rash, myalgia
Gemfibrozil	↓ TGs ↓ Synthesis of TGs and other lipids	Serious adverse effects include pancreatitis, hepatotoxicity, and rhabdomyolysis Contraindicated in severe kidney and liver disease Use with caution in patients who are also taking statins
<b>Bile Acid Sequestrants</b>		
Cholestyramine	↓ LDL	Most often used as adjunct therapy when statins alone have not been effective in controlling lipid levels
Colestipol	Slight ↑ HDL	Side effects include constipation, abdominal pain, GI bleeding
Colesevelam	Oxidize cholesterol into bile acids, which ↓ fat absorption	May decrease absorption of other drugs Taken before meals
<b>Cholesterol Absorption Inhibitor</b>		
Ezetimibe	↓ LDL Inhibits absorption of cholesterol in small intestine	Better tolerated than bile acid sequestrants Used in combination with other agents, such as statins Side effects include abdominal pain, arthralgia, myalgia Contraindicated in liver disease
<b>Proprotein Convertase Subtilisin-Kexin Type 9 (PCSK9) Agents</b>		
Alirocumab	Prolongs receptor activity to promote clearance of cholesterol	Only administered by subcutaneous injection via a pen device, one or two times per month as prescribed
Evolocumab	↓ LDL ↓ risk of MI and stroke ↓ need for stent or CABG	Side effects include rhinitis, sore throat, flulike symptoms, muscle pain, diarrhea, and redness, pain, or bruising at injection site

↓ decrease, ↑ increase; ACS, acute coronary syndrome; CABG, coronary artery bypass graft; GI, gastrointestinal; HDL, high-density lipoprotein; HMG-CoA, 3-hydroxy-3-methylglutaryl coenzyme A; LDL, low-density lipoprotein; MI, myocardial infarction; TGs, triglycerides.

Adapted from Frandsen, G., & Pennington, S. S. (2021). *Abrams' clinical drug therapy: Rationales for nursing practice* (12th ed.). Philadelphia, PA: Wolters Kluwer Health.

A person at increased risk for heart disease is encouraged to stop tobacco use through any means possible: educational programs, counseling, consistent motivation and reinforcement messages, support groups, and medications. Some people have found complementary therapies (e.g., acupuncture, guided imagery, hypnosis) to be helpful. People who stop smoking reduce their risk of heart disease within the first year, and the risk continues to decline as long as they refrain from smoking (Benjamin, Muntner, Alonso, et al., 2019).

The use of medications such as the nicotine patch, nicotine lozenges, nicotine gum, varenicline, or bupropion may assist with stopping the use of tobacco (Barua, Rigotti, Benowitz, et al., 2018). Products containing nicotine have some of the same effects as smoking: catecholamine release (increasing heart rate and blood pressure) and increased platelet adhesion. These medications should be used for a short time and at the lowest effective doses.

Exposure to others' smoke (passive or secondhand smoke) increases the risk for CAD by 25% to 30% and for stroke by 20% to 30% (Benjamin et al., 2019). Other forms of tobacco use are becoming increasingly common today. Use of electronic nicotine delivery systems (ENDS) including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars has increased, particularly among adolescents and young adults. Specifically, e-cigarette use, which entails inhalation of a vaporized liquid that includes nicotine, solvents, and flavoring ("vaping"), has risen significantly in these groups. Use of cigarillos and other mass market cigars, hookahs, and water pipes are also on the rise. Short-term exposure to water pipe smoking is associated with an increase in systolic blood pressure and heart rate, but long-term effects remain unclear. The cardiovascular risks associated with e-cigarette use are not yet known (Benjamin et al., 2019).

## Managing Hypertension

Hypertension is defined as systolic blood pressure measurements of greater than 130 mm Hg and/or diastolic blood pressure levels greater than 80 mm Hg. A single reading is not adequate to make a diagnosis. Averaging two or three measurements obtained on two to three different occasions will provide a more accurate measurement (Whelton, Carey, Aronow, et al., 2018). The risk of cardiovascular disease increases as blood pressure increases, and current guidelines support treating hypertension with a goal of keeping the blood pressure under 130/80 for all adults (Whelton et al., 2018). Long-standing elevated blood pressure may result in increased stiffness of the vessel walls, leading to vessel injury and a resulting inflammatory response within the intima. Inflammatory mediators then lead to the release of growth-promoting factors that cause vessel hypertrophy and hyperresponsiveness. These changes result in acceleration and aggravation of atherosclerosis. Hypertension also increases the work of the left ventricle, which must pump harder to eject blood

into the arteries. Over time, the increased workload causes the heart to enlarge and thicken (i.e., hypertrophy) and may eventually lead to heart failure.

Early detection of high blood pressure and adherence to a therapeutic regimen can prevent the serious consequences associated with untreated elevated blood pressure, including CAD. Intensive management of hypertension lowers the risk of cardiovascular events, including heart attack and stroke, and lowers the risk of death (Whelton et al., 2018; see [Chapter 27](#) for a detailed discussion of hypertension).

## Controlling Diabetes

Diabetes is known to accelerate the development of heart disease. Hyperglycemia fosters dyslipidemia, increased platelet aggregation, and altered red blood cell function, which can lead to thrombus formation. These metabolic alterations may impair endothelial cell-dependent vasodilation and smooth muscle function, promoting the development of atherosclerosis. Treatment with insulin, metformin, and other therapeutic interventions that lower plasma glucose levels can lead to improved endothelial function and patient outcomes. See [Chapter 46](#) for a detailed discussion of diabetes.

## Gender

Heart disease has long been recognized as a cause of morbidity and mortality in men, but it has not always been as readily recognized in women. Cardiovascular events in women occur an average of 10 years later in life than they do in men (Wada, Miyauchi, & Daida, 2019). Women tend to have a higher incidence of complications from cardiovascular disease and a higher mortality. In addition, women tend to not recognize the symptoms of CAD as early as men, and they wait longer to report their symptoms and seek medical assistance (Wada et al., 2019).

The age difference between women and men who were newly diagnosed with CAD was traditionally thought to be related to estrogen. Menopause is now recognized as a milestone in the aging process, during which risk factors tend to accumulate. Cardiovascular disease may be well developed by the time of menopause, and although hormone therapy (HT) (formerly referred to as hormone replacement therapy) for menopausal women was once promoted as preventive therapy for CAD, research does not support HT as an effective means of prevention. HT decreases menopausal symptoms and the risk of osteoporosis-related bone fractures; however, it also has been associated with an increased incidence of CAD, breast cancer, deep vein thrombosis, stroke, and pulmonary embolism. Current guidelines do not recommend HT for primary or secondary prevention of CAD (Wada et al., 2019; see [Chapter 21](#) for further discussion).

In the past, women who possibly had coronary vascular events were less likely than men to be referred for coronary artery diagnostic procedures such as heart catheterization or treatment with invasive interventions (e.g., PCI). However, as a result of better education of health care professionals and the general public, gender differences now have less influence on diagnosis and treatment (Wada et al., 2019).

## Unfolding Patient Stories: Carl Shapiro • Part 1



Carl Shapiro, who has a family history of atherosclerotic cardiovascular disease, is diagnosed with hypertension and hyperlipidemia during a routine visit to his primary provider. He is overweight, smokes a half pack of cigarettes per day, and describes his job as stressful. What questions can the nurse ask Carl Shapiro to help develop a plan for patient education? What topics are important for the nurse to address, and how can the information be presented? (Carl Shapiro's story continues in Chapter 67.)

Care for Carl and other patients in a realistic virtual environment: **vSim** ([thepoint.lww.com/vSimMedicalSurgical](http://thepoint.lww.com/vSimMedicalSurgical)). Practice documenting these patients' care in DocuCare ([thepoint.lww.com/DocuCareEHR](http://thepoint.lww.com/DocuCareEHR)).

## Angina Pectoris

Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest. The cause is insufficient coronary blood flow, resulting in a decreased oxygen supply when there is increased myocardial demand for oxygen in response to physical exertion or emotional stress. In other words, the need for oxygen exceeds the supply.

## Pathophysiology

Angina is usually caused by atherosclerotic disease and most often is associated with a significant obstruction of at least one major coronary artery. Normally, the myocardium extracts a large amount of oxygen from the coronary circulation to meet its continuous demands. When demand increases, flow through the coronary arteries needs to be increased. When there is a blockage in a coronary artery, flow cannot be increased and ischemia results. The types of angina are listed in [Chart 23-2](#). Several factors are associated with typical anginal pain:

- Physical exertion, which precipitates an attack by increasing myocardial oxygen demand
- Exposure to cold, which causes vasoconstriction and elevated blood pressure, with increased oxygen demand
- Eating a heavy meal, which increases the blood flow to the mesenteric area for digestion, thereby reducing the blood supply available to the heart muscle; in a severely compromised heart, shunting of blood for digestion can be sufficient to induce anginal pain

## Chart 23-2

### Types of Angina

- **Stable angina:** predictable and consistent pain that occurs on exertion and is relieved by rest and/or nitroglycerin
- **Unstable angina** (also called *preinfarction angina* or *crescendo angina*): symptoms increase in frequency and severity; may not be relieved with rest or nitroglycerin
- **Intractable or refractory angina:** severe incapacitating chest pain
- **Variant angina** (also called *Prinzmetal's angina*): pain at rest with reversible ST-segment elevation; thought to be caused by coronary artery vasospasm
- **Silent ischemia:** objective evidence of ischemia (such as electrocardiographic changes with a stress test), but patient reports no pain

- Stress or any emotion-provoking situation, causing the release of catecholamines, which increases blood pressure, heart rate, and myocardial workload

Unstable angina is not closely associated with these listed factors. It may occur at rest (see later discussion).

## Clinical Manifestations

Ischemia of the heart muscle may produce pain or other symptoms, varying from mild indigestion to a choking or heavy sensation in the upper chest. The severity ranges from discomfort to agonizing pain. The pain may be accompanied by severe apprehension and a feeling of impending death. It is often felt deep in the chest behind the sternum (retrosternal area). Typically, the pain or discomfort is poorly localized and may radiate to the neck, jaw, shoulders, and inner aspects of the upper arms, usually the left arm. The patient often feels tightness or a heavy choking or strangling sensation that has a viselike, insistent quality. The patient with diabetes may not have severe pain with angina because autonomic neuropathy can blunt nociceptor transmission, dulling the perception of pain (Norris, 2019).

A feeling of weakness or numbness in the arms, wrists, and hands, as well as shortness of breath, pallor, diaphoresis, dizziness or lightheadedness, and nausea and vomiting, may accompany the pain. An important characteristic of angina is that it subsides with rest or administration of nitroglycerin. In many patients, anginal symptoms follow a stable, predictable pattern.

Unstable angina is characterized by attacks that increase in frequency and severity and are not relieved by rest and administration of nitroglycerin. Patients with unstable angina require medical intervention.



## Gerontologic Considerations

The older adult with angina may not exhibit a typical pain profile because of the diminished pain transmission that can occur with aging. Often the presenting

symptom in older adults is dyspnea. Sometimes there are no symptoms (“silent” CAD), making recognition and diagnosis a clinical challenge. Older patients should be encouraged to recognize their chest pain–like symptom (e.g., weakness) as an indication that they should rest or take prescribed medications. Pharmacologic stress testing and cardiac catheterization may be used to diagnose CAD in older patients. Medications used to manage angina are given cautiously in older adults because they are associated with an increased risk of adverse reactions (Frandsen & Pennington, 2021). Invasive procedures (e.g., PCI) that were once considered too risky in older adults are now being performed successfully, and many older adults benefit from symptom relief and longer survival (Lattuca, Kerneis, & Zeitouni, 2019).

## Assessment and Diagnostic Findings

The diagnosis of angina begins with the patient’s history related to the clinical manifestations of ischemia. A 12-lead electrocardiogram (ECG) may show changes indicative of ischemia, such as T-wave inversion, ST-segment elevation, or the development of an abnormal Q wave (Norris, 2019). Laboratory studies are performed; these generally include cardiac biomarker testing to rule out ACS (see later discussion). The patient may undergo an exercise or pharmacologic stress test in which the heart is monitored continuously by an ECG, echocardiogram, or both. The patient may also be referred for a nuclear scan or invasive procedure (e.g., cardiac catheterization, coronary angiography).

## Medical Management

The objectives of the medical management of angina are to decrease the oxygen demand of the myocardium and to increase the oxygen supply. Medically, these objectives are met through pharmacologic therapy and control of risk factors. Alternatively, reperfusion procedures may be used to restore the blood supply to the myocardium. These include PCI procedures (e.g., percutaneous transluminal coronary angioplasty [PTCA] and intracoronary stents) and CABG (see later discussion).

### Pharmacologic Therapy

Table 23-2 summarizes drug therapy.

#### Nitroglycerin

Nitrates are a standard treatment for angina pectoris. Nitroglycerin is a potent vasodilator that improves blood flow to the heart muscle and relieves pain. Nitroglycerin dilates primarily the veins and, to a lesser extent, the arteries. Dilation of the veins causes venous pooling of blood throughout the body. As a result, less blood returns to the heart, and filling pressure (preload) is reduced. If the patient is hypovolemic (does not have adequate circulating blood volume), the decrease in filling pressure can cause a significant decrease in cardiac output and blood pressure (Frandsen & Pennington, 2021).

Nitrates also relax the systemic arteriolar bed, lowering blood pressure and decreasing afterload. These effects decrease myocardial oxygen requirements,

bringing about a more favorable balance between supply and demand.

Nitroglycerin may be given by several routes: sublingual tablet or spray, oral capsule, topical agent, and intravenous (IV) administration. Sublingual nitroglycerin is generally placed under the tongue or in the cheek (buccal pouch) and ideally alleviates the pain of ischemia within 3 minutes. [Chart 23-3](#) provides more information on self-administration of sublingual nitroglycerin. Oral preparations and topical patches are used to provide sustained effects. A regimen in which the patches are applied in the morning and removed at bedtime allows for a nitrate-free period to prevent the development of tolerance.

**TABLE 23-2**



Select Medications Used to Treat Stable Angina

Medications	Major Indications
<b>Nitrates</b>	
Nitroglycerin	Short- and long-term reduction of myocardial oxygen consumption through selective vasodilation
<b>Beta-Adrenergic Blocking Agents (Beta-Blockers)</b>	
Metoprolol	Reduction of myocardial oxygen consumption by blocking beta-adrenergic stimulation of the heart
Atenolol	
<b>Calcium Ion Antagonists (Calcium Channel Blockers)</b>	
Amlodipine	Negative inotropic effects; indicated in patients not responsive to beta-blockers; used as primary treatment for vasospasm
Diltiazem	
<b>Antiplatelet Medications</b>	
Aspirin	Prevention of platelet aggregation
Clopidogrel	
Prasugrel	
Ticagrelor	
<b>Anticoagulants</b>	
Heparin (unfractionated)	Prevention of thrombus formation
Low-molecular-weight heparins:	
Enoxaparin	
Dalteparin	

Adapted from Rousan, T. A., Mathew, S. T., & Thadani, U. (2017). Drug therapy for stable angina pectoris. *Drugs*, 77(3), 265–284.

A continuous or intermittent IV infusion of nitroglycerin may be given to the hospitalized patient with recurring signs and symptoms of ischemia or after a revascularization procedure. The rate of infusion is titrated to the patient's pain level and blood pressure. It usually is not given if the systolic blood pressure is less than 90 mm Hg. Generally, after the patient is symptom-free, the nitroglycerin may be switched to an oral or topical preparation within 24 hours. A common adverse effect of nitroglycerin is headache, which may limit the use of this drug in some patients.

### Beta-Adrenergic Blocking Agents

Beta-blockers such as metoprolol reduce myocardial oxygen consumption by blocking beta-adrenergic sympathetic stimulation to the heart. The result is a reduction in heart rate, slowed conduction of impulses through the conduction system, decreased blood pressure, and reduced myocardial **contractility** (force of contraction). Because of these effects, beta-blockers balance the myocardial oxygen needs (demands) and the amount of oxygen available (supply). This helps control chest pain and delays the onset of ischemia during work or exercise. Beta-blockers reduce the incidence of recurrent angina, infarction, and cardiac mortality. The dose can be titrated to achieve a resting heart rate of 50 to 60 bpm (Frandsen & Pennington, 2021).

Cardiac side effects and possible contraindications include hypotension, bradycardia, advanced atrioventricular block, and acute heart failure. If a beta-blocker is given IV for an acute cardiac event, the ECG, blood pressure, and heart rate are monitored closely after the medication has been given. Side effects include depressed mood, fatigue, decreased libido, and dizziness. Patients taking beta-blockers are cautioned not to stop taking them abruptly, because angina may worsen, and MI may develop. Beta-blocker therapy should be decreased gradually over several days before being discontinued. Patients with diabetes who take beta-blockers are instructed to monitor their blood glucose levels as prescribed because beta-blockers can mask signs of hypoglycemia. Beta-blockers that are not cardioselective also affect the beta-adrenergic receptors in the bronchioles, causing bronchoconstriction, and therefore are contraindicated in patients with significant chronic pulmonary disorders, such as asthma.

### Chart 23-3 PHARMACOLOGY

### **Self-Administration of Nitroglycerin**

Most patients with angina pectoris self-administer nitroglycerin on an as-needed basis. A key nursing role in such cases is educating patients about the medication and how to take it. Sublingual nitroglycerin comes in tablet and spray forms.

- Instruct the patient to make sure that the mouth is moist, the tongue is still, and saliva is not swallowed until the nitroglycerin tablet dissolves. If the pain is severe, the patient can crush the tablet between the teeth to hasten sublingual absorption.
- Advise the patient to carry the medication at all times as a precaution. However, because nitroglycerin is very unstable, it should be carried securely in its original container (e.g., capped dark glass bottle); tablets should never be removed and stored in metal or plastic pillboxes.
- Explain that nitroglycerin is volatile and is inactivated by heat, moisture, air, light, and time. Instruct the patient to renew the nitroglycerin supply every 6 months.
- Inform the patient that the medication should be taken in anticipation of any activity that may produce pain. Because nitroglycerin increases tolerance for exercise and stress when taken prophylactically (i.e., before angina-producing activity, such as exercise, stair-climbing, or sexual intercourse), it is best taken before pain develops.
- Recommend that the patient note how long it takes for the nitroglycerin to relieve the discomfort. Advise the patient that if pain persists after taking three sublingual tablets at 5-minute intervals, emergency medical services should be called.
- Discuss possible side effects of nitroglycerin, including flushing, throbbing headache, hypotension, and tachycardia.
- Advise the patient to sit down for a few minutes when taking nitroglycerin to avoid hypotension and syncope.

Adapted from Comerford, K. C., & Durkin, M. T. (Eds.) (2020). *Nursing2020 Drug Handbook*. Philadelphia, PA: Wolters Kluwer.

### **Calcium Channel Blocking Agents**

Calcium channel blockers have a variety of effects on the ischemic myocardium. These agents decrease sinoatrial node automaticity and atrioventricular node conduction, resulting in a slower heart rate and a decrease in the strength of myocardial contraction (negative inotropic effect). These effects decrease the workload of the heart. Calcium channel blockers also increase myocardial oxygen supply by dilating the smooth muscle wall of the coronary arterioles; they decrease myocardial oxygen demand by reducing systemic arterial pressure and the workload of the left ventricle (Frandsen & Pennington, 2021). The calcium channel blockers most commonly used are amlodipine and diltiazem. In addition to their use to treat angina, they are commonly prescribed for hypertension. Hypotension may occur after the administration of any of the calcium channel blockers, particularly when administered IV. Other side effects may include atrioventricular block, bradycardia, and constipation.

## Antiplatelet and Anticoagulant Medications

Antiplatelet medications are given to prevent platelet aggregation and subsequent thrombosis, which impedes blood flow through the coronary arteries.

### Aspirin

Aspirin prevents platelet aggregation and reduces the incidence of MI and death in patients with CAD (Frandsen & Pennington, 2021). A 162- to 325-mg dose of aspirin should be given to the patient with a new diagnosis of angina and then continued with 81 to 325 mg daily. Patients should be advised to continue aspirin even if they concurrently take other analgesics such as acetaminophen. Because aspirin may cause GI upset and bleeding, the use of histamine-2 ( $H_2$ ) blockers (e.g., famotidine) or proton pump inhibitors (e.g., omeprazole) should be considered concomitant with continued aspirin therapy (Ibanez, James, Agewall, et al., 2018).

### Adenosine Diphosphate Receptor Antagonists (P2Y<sub>12</sub>)

These medications act on different pathways than aspirin to block platelet activation. However, unlike aspirin, these agents may take a few days to achieve antiplatelet effects. Clopidogrel is commonly prescribed in addition to aspirin in patients at high risk for MI. Newer oral agents such as prasugrel and ticagrelor may be used in place of clopidogrel during coronary events and interventions (Frandsen & Pennington, 2021). Both carry the risk of bleeding from the GI tract or other sites.

### Heparin

Unfractionated IV heparin prevents the formation of new blood clots (i.e., it is an anticoagulant). Treating patients with unstable angina with heparin reduces the occurrence of MI. If the patient's signs and symptoms indicate a significant risk for a cardiac event, the patient is hospitalized and may be given an IV bolus of heparin and started on a continuous infusion. The dose of heparin given is based on the results of the activated partial thromboplastin time (aPTT). Heparin therapy is usually considered therapeutic when the aPTT is 2 to 2.5 times the normal aPTT value.

A subcutaneous injection of low-molecular-weight heparin (LMWH; enoxaparin or dalteparin) may be used instead of IV unfractionated heparin to treat patients with unstable angina or non-ST-segment elevation myocardial infarction (NSTEMI) (Frandsen & Pennington, 2021). LMWH provides effective and stable anticoagulation, potentially reducing the risk of rebound ischemic events, and eliminating the need to monitor aPTT results. LMWHs may be beneficial before and during PCIs as well as for ACS.

Because unfractionated heparin and LMWH increase the risk of bleeding, the patient is monitored for signs and symptoms of external and internal bleeding, such as low blood pressure, increased heart rate, and decreased serum hemoglobin and hematocrit. The patient receiving heparin is placed on bleeding precautions, which include:

- Applying pressure to the site of any needle puncture for a longer time than usual
- Avoiding intramuscular (IM) injections
- Avoiding tissue injury and bruising from trauma or use of constrictive devices (e.g., continuous use of an automatic blood pressure cuff)

A decrease in platelet count or evidence of thrombosis may indicate heparin-induced thrombocytopenia (HIT), an antibody-mediated reaction to heparin that may result in thrombosis. Patients who have received heparin within the past 3 months and those who have been receiving unfractionated heparin for 4 to 14 days are at high risk for HIT (Frandsen & Pennington, 2021). As an alternative to LMWH and unfractionated heparin, argatroban, a direct antithrombotic agent might be prescribed (Frandsen & Pennington, 2021; see [Chapter 29](#) for further discussion of HIT).

### Glycoprotein IIb/IIIa Agents

IV administration of glycoprotein (GP) IIb/IIIa agents, such as abciximab or eptifibatide, is indicated for hospitalized patients with unstable angina and as adjunct therapy for PCI. These agents prevent platelet aggregation by blocking the GP IIb/IIIa receptors on the platelets, preventing adhesion of fibrinogen and other factors that crosslink platelets to each other and thus form intracoronary clots (Urden, Stacy, & Lough, 2019). As with heparin, bleeding is the major side effect, and bleeding precautions should be initiated.

### Oxygen Administration

Oxygen therapy is usually initiated at the onset of chest pain in an attempt to increase the amount of oxygen delivered to the myocardium and to decrease pain. The therapeutic effectiveness of oxygen is determined by observing the rate and rhythm of respirations and the color of skin and mucous membranes. Blood oxygen saturation is monitored by pulse oximetry; the normal oxygen saturation ( $\text{SpO}_2$ ) level is  $>95\%$  on room air (Urden et al., 2019).

## NURSING PROCESS

### The Patient with Angina Pectoris



#### Assessment

The nurse gathers information about the patient's symptoms and activities, especially those that precede and precipitate attacks of angina pectoris. Appropriate questions are listed in [Chart 23-4](#). The answers to these questions form the basis for designing an effective program of treatment and prevention. In addition to assessing angina pectoris or its equivalent, the nurse also assesses the patient's risk factors for CAD, the patient's response to angina, the patient's and family's understanding of the diagnosis, and adherence to the current treatment plan.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include:

- Risk for impaired cardiac function
- Anxiety associated with cardiac symptoms and possible death
- Lack of knowledge about the underlying disease and methods for avoiding complications
- Able to perform self care

**Chart 23-4**



### ASSESSMENT

#### Assessing Angina

Ask the following:

- "Where is the pain (or prodromal symptoms)? Can you point to it?"
- "Can you feel the pain anywhere else?"
- "How would you describe the pain?"
- "Is it like the pain you had before?"
- "Can you rate the pain on a 0–10 scale, with 10 being the most pain?"
- "When did the pain begin?"
- "How long does it last?"
- "What brings on the pain?"
- "What helps the pain go away?"
- "Do you have any other symptoms with the pain?"

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- ACS and/or MI (described later in this chapter)
- Arrhythmias and cardiac arrest (see Chapters 22 and 25)
- Heart failure (see [Chapter 25](#))

- Cardiogenic shock (see [Chapter 11](#))

### Planning and Goals

Major patient goals include immediate and appropriate treatment when angina occurs, prevention of angina, reduction of anxiety, awareness of the disease process and understanding of the prescribed care, adherence to the self-care program, and absence of complications.

### Nursing Interventions

#### TREATING ANGINA

If the patient reports pain (or cardiac ischemia is suggested by prodromal symptoms, which may include sensations of indigestion or nausea, choking, heaviness, weakness or numbness in the upper extremities, dyspnea, or dizziness), the nurse takes immediate action. The patient experiencing angina is directed to stop all activities and sit or rest in bed in a semi-Fowler position to reduce the oxygen requirements of the ischemic myocardium. The nurse assesses the patient's angina, asking questions to determine whether the angina is the same as the patient typically experiences. A change may indicate a worsening of the disease or a different cause. The nurse then continues to assess the patient, measuring vital signs and observing for signs of respiratory distress. If the patient is in the hospital, a 12-lead ECG is usually obtained and assessed for ST-segment and T-wave changes. If the patient has been placed on cardiac monitoring with continuous ST-segment monitoring, the ST segment is assessed for changes.

Nitroglycerin is given sublingually, and the patient's response is assessed (relief of chest pain and effect on blood pressure and heart rate). If the chest pain is unchanged or is lessened but still present, nitroglycerin administration is repeated up to three doses. Each time blood pressure, heart rate, and the ST segment (if the patient is on a monitor with ST-segment monitoring capability) are assessed. The nurse administers oxygen therapy if the patient's respiratory rate is increased or if the oxygen saturation level is decreased. Oxygen is usually given at 2 L/min by nasal cannula, even without evidence of desaturation, although there is no current evidence of a positive effect on patient outcome. If the pain is significant and continues after these interventions, the patient is further evaluated for acute MI and may be transferred to a higher-acuity nursing unit (Ibanez et al., 2018).

#### REDUCING ANXIETY

Patients with angina often fear loss of their roles within society and the family. They may also fear that the pain (or the prodromal symptoms) may lead to an MI or death. Exploring the implications that the diagnosis has for the patient and providing information about the illness, its treatment, and methods of preventing its progression are important nursing interventions. Various stress reduction methods, such as guided imagery or music therapy, should be explored with the patient (Meghani, 2017). Addressing the spiritual needs of the patient and family may also assist in allaying anxieties and fears.

#### PREVENTING PAIN

The nurse reviews the assessment findings, identifies the level of activity that causes the patient's pain or prodromal symptoms, and plans the patient's activities

accordingly. If the patient has pain frequently or with minimal activity, the nurse alternates the patient's activities with rest periods. Balancing activity and rest is an important aspect of the educational plan for the patient and family.

#### PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



**Educating Patients About Self-Care.** The program for educating the patient with angina is designed so that the patient and family understand the illness, identify the symptoms of myocardial ischemia, state the actions to take when symptoms develop, and discuss methods to prevent chest pain and the advancement of CAD. The goals of education are to reduce the frequency and severity of anginal attacks, to delay the progress of the underlying disease if possible, and to prevent complications. The factors outlined in [Chart 23-5](#) are important in educating the patient with angina pectoris.

The self-care program is prepared in collaboration with the patient and family or friends. Activities should be planned to minimize the occurrence of anginal episodes. The patient needs to understand that any pain unrelieved within 15 minutes by the usual methods, including nitroglycerin (see [Chart 23-3](#)), should be treated at the closest ED; the patient should call 911 for assistance.

**Continuing and Transitional Care.** For patient with disability or special needs, arrangements are made for transitional, home, or community care when appropriate. A home health or transitional care nurse can assist the patient with scheduling and keeping follow-up appointments. The patient may need reminders about follow-up monitoring, including periodic laboratory testing. In addition, the home health nurse may monitor the patient's adherence to dietary restrictions and to prescribed antianginal medications, including nitroglycerin. If the patient has severe anginal symptoms, the nurse may assess the home environment and recommend modifications that diminish the occurrence of anginal episodes. For instance, if a patient cannot climb stairs without experiencing ischemia, the home health nurse may help the patient plan daily activities that minimize stair-climbing.

Chart 23-5



#### HOME CARE CHECKLIST

## Managing Angina Pectoris

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of angina pectoris on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State changes in lifestyle (e.g., diet, activity) or home environment necessary to maintain health.
- Follow a diet low in saturated fat, high in fiber, and, if indicated, lower in calories.
- Reduce the probability of an episode of anginal pain by balancing rest with regular daily activities that do not produce chest discomfort, shortness of breath, or undue fatigue.
- Follow the prescribed exercise regimen.
  - Recognize that temperature extremes (particularly cold) may induce anginal pain; therefore, avoid exercise in temperature extremes.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Take medications, especially aspirin and beta-blockers, as prescribed.
- Carry nitroglycerin at all times; state when and how to use it; identify its side effects.
- Avoid using medications or any over-the-counter substances (e.g., diet pills, nasal decongestants) that can increase the heart rate and blood pressure without first discussing with the primary provider.
- Use appropriate resources for support during emotionally stressful times (e.g., counselor, nurse, clergy, primary provider).
- Stop smoking and the use of other forms of tobacco and avoid secondhand smoke (because smoking increases the heart rate, blood pressure, and blood carbon monoxide levels).
- Achieve and maintain normal blood pressure.
- Achieve and maintain normal blood glucose levels.
- State how to reach primary provider with questions or complications.
  - Report increase in symptoms to the primary provider.
  - State time and date of follow-up appointments and testing.
- Identify the need for health promotion (e.g., weight reduction, cessation of tobacco use, stress management), disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, independent activities of daily living.

### Evaluation

Expected patient outcomes may include:

1. Reports that pain is relieved promptly
  - a. Recognizes symptoms
  - b. Takes immediate action
  - c. Seeks medical assistance if pain persists or changes in quality

2. Reports decreased anxiety
  - a. Expresses acceptance of diagnosis
  - b. Expresses control over choices within medical regimen
  - c. Does not exhibit signs and symptoms that indicate a high level of anxiety
3. Understands ways to avoid complications and is free of complications
  - a. Describes the process of angina
  - b. Explains reasons for measures to prevent complications
  - c. Exhibits stable ECG
  - d. Experiences no signs and symptoms of acute MI
4. Adheres to self-care program
  - a. Takes medications as prescribed
  - b. Keeps health care appointments
  - c. Implements plan to reduce risk factors



## Acute Coronary Syndrome and Myocardial Infarction

**Acute coronary syndrome (ACS)** is an emergent situation characterized by an acute onset of myocardial ischemia that results in myocardial death (i.e., MI) if definitive interventions do not occur promptly. (Although the terms *coronary occlusion*, *heart attack*, and *myocardial infarction* are used synonymously, the preferred term is *myocardial infarction*.) The spectrum of ACS includes unstable angina, NSTEMI, and ST-segment elevation myocardial infarction (STEMI).

### Pathophysiology

In unstable angina, there is reduced blood flow in a coronary artery, often due to rupture of an atherosclerotic plaque. A clot begins to form on top of the coronary lesion, but the artery is not completely occluded. This is an acute situation that can result in chest pain and other symptoms that may be referred to as preinfarction angina because the patient will likely have an MI if prompt interventions do not occur.

In an MI, plaque rupture and subsequent thrombus formation result in complete occlusion of the artery, leading to ischemia and necrosis of the myocardium supplied by that artery. Vasospasm (sudden constriction or narrowing) of a coronary artery, decreased oxygen supply (e.g., from acute blood loss, anemia, or low blood pressure), and increased demand for oxygen (e.g., from a rapid heart rate, thyrotoxicosis, or ingestion of cocaine) are other causes of MI. In each case, a profound imbalance exists between myocardial oxygen supply and demand.

The area of infarction develops over minutes to hours. As the cells are deprived of oxygen, ischemia develops, cellular injury occurs, and the lack of oxygen results in infarction, or the death of cells. The expression “time is muscle” reflects the urgency of appropriate treatment to improve patient outcomes. Approximately every 40 seconds, an American will have an MI (Benjamin et al., 2019), and many of these

people will die as a result. Early recognition and treatment of patients presenting with an MI will improve their chances of survival.

Various descriptions are used to further identify an MI: the type (NSTEMI, STEMI), the location of the injury to the ventricular wall (anterior, inferior, posterior, or lateral wall), and the point in time within the process of infarction (acute, evolving, or old). The differentiation between NSTEMI and STEMI is determined by diagnostic tests and is explained later in this chapter.

The 12-lead ECG identifies the type and location of the MI, and other ECG indicators, such as a Q wave, and patient history, identify the timing. Regardless of the location, the goals of medical therapy are to relieve symptoms, prevent or minimize myocardial tissue death, and prevent complications. The pathophysiology of CAD and the risk factors involved were discussed earlier in this chapter.

## Clinical Manifestations

Chest pain that occurs suddenly and continues despite rest and medication is the presenting symptom in most patients with ACS. Some of these patients have prodromal symptoms or a previous diagnosis of CAD, but others report no previous symptoms. Patients may present with a combination of symptoms, including chest pain, shortness of breath, indigestion, nausea, and anxiety. They may have cool, pale, and moist skin. Their heart rate and respiratory rate may be faster than normal. These signs and symptoms, which are caused by stimulation of the sympathetic nervous system, may be present for only a short time or may persist. In many cases, the signs and symptoms of MI cannot be distinguished from those of unstable angina; hence, the evolution of the term *acute coronary syndrome*.

## Assessment and Diagnostic Findings

The diagnosis of ACS is generally based on the presenting symptoms (see Chart 23-6); the 12-lead ECG and laboratory tests (e.g., serial cardiac biomarkers) are performed to clarify whether the patient has unstable angina, NSTEMI, or STEMI (Ibanez et al., 2018). The prognosis depends on the severity of coronary artery obstruction and the presence and extent of myocardial damage. Physical examination is always conducted, but the examination alone does not confirm the diagnosis.

### Patient History

The patient history includes the description of the presenting symptom (e.g., pain), the history of previous cardiac and other illnesses, and the family history of heart disease. The history should also include information about the patient's risk factors for heart disease.

### Electrocardiogram

The 12-lead ECG provides information that assists in ruling out or diagnosing an acute MI. It should be obtained within 10 minutes from the time a patient reports pain or arrives in the ED. By monitoring serial ECG changes over time, the location, evolution, and resolution of an MI can be identified and monitored.

The ECG changes that occur with an MI are seen in the leads that view the involved surface of the heart. The expected ECG changes are T-wave inversion, ST-segment elevation, and development of an abnormal Q wave (see [Fig. 23-5](#)). Because infarction evolves over time, the ECG also changes over time. The first ECG signs of an acute MI are usually seen in the T wave and ST segment (Urdan et al., 2019). As the area of injury becomes ischemic, myocardial repolarization is altered and delayed, causing the T wave to invert. Myocardial injury also causes ST-segment changes. The ST segment is normally flat on the ECG tracing. The injured myocardial cells depolarize normally but repolarize more rapidly than normal cells, causing the ST segment to rise at least 1 mm above the isoelectric line (the area between the T wave and the next P wave is used as the reference for the isoelectric line). This change is measured 0.06 to 0.08 seconds after the end of the QRS—a point called the *J point* (Urdan et al., 2019) (see [Fig. 23-6](#)). An elevation in the ST segment in two contiguous leads is a key diagnostic indicator for MI (i.e., STEMI).

**Chart 23-6****ASSESSMENT**

## Assessing for Acute Coronary Syndrome or Acute Myocardial Infarction

Be alert for the following signs and symptoms:

### **Cardiovascular**

- Chest pain or discomfort not relieved by rest or nitroglycerin; palpitations. Heart sounds may include S<sub>3</sub>, S<sub>4</sub>, and new onset of a murmur.
- Increased jugular venous distention may be seen if the myocardial infarction (MI) has caused heart failure.
- Blood pressure may be elevated because of sympathetic stimulation or decreased because of decreased contractility, impending cardiogenic shock, or medications.
- Irregular pulse may indicate atrial fibrillation.
- In addition to ST-segment and T-wave changes, the electrocardiogram may show tachycardia, bradycardia, or other arrhythmias.

### **Respiratory**

Shortness of breath, dyspnea, tachypnea, and crackles if MI has caused pulmonary congestion. Pulmonary edema may be present.

### **Gastrointestinal**

Nausea, indigestion, and vomiting.

### **Genitourinary**

Decreased urinary output may indicate cardiogenic shock.

### **Skin**

Cool, clammy, diaphoretic, and pale appearance due to sympathetic stimulation may indicate cardiogenic shock.

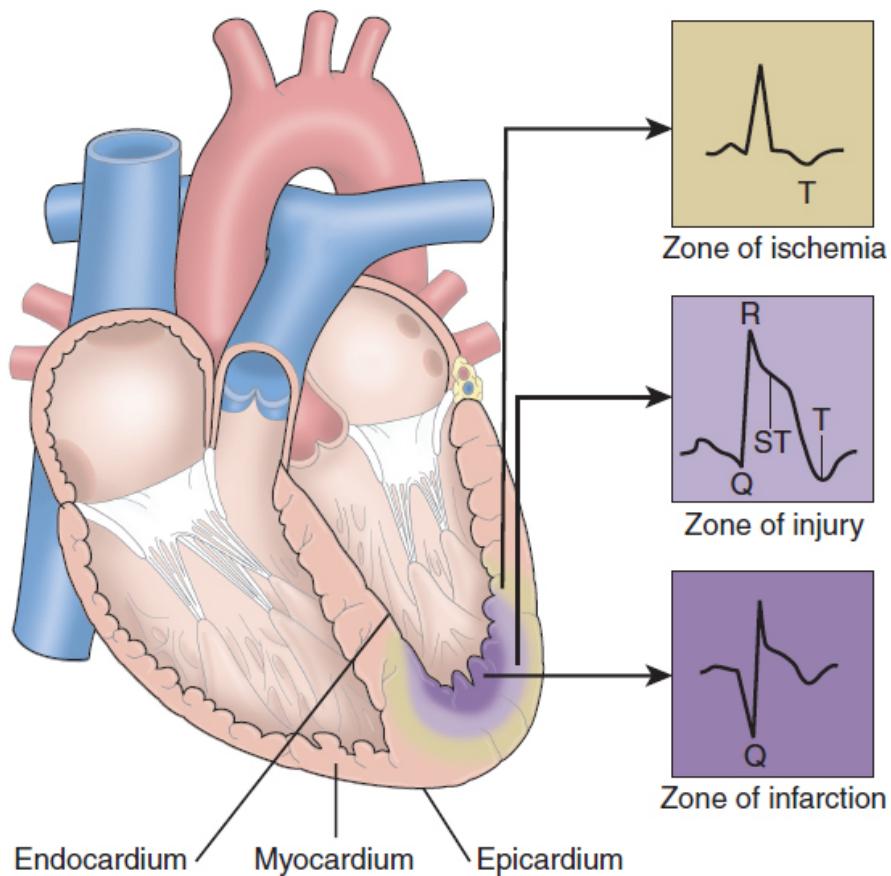
### **Neurologic**

Anxiety, restlessness, and lightheadedness may indicate increased sympathetic stimulation or a decrease in contractility and cerebral oxygenation. The same symptoms may also herald cardiogenic shock.

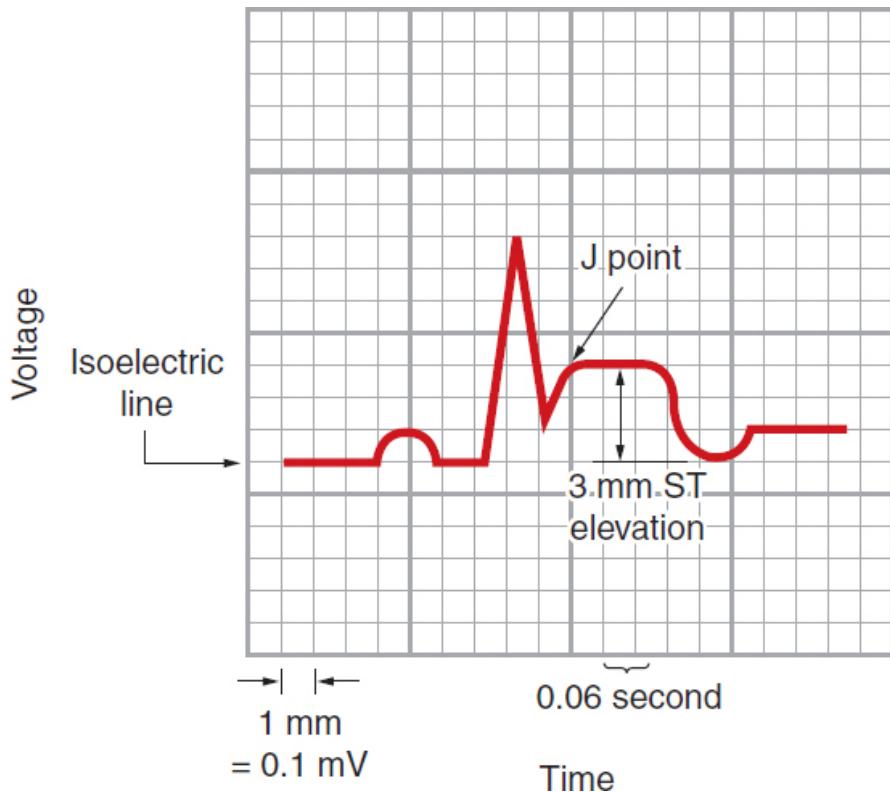
### **Psychological**

Fear with feeling of impending doom, or denial that anything is wrong.

The appearance of abnormal Q waves is another indication of MI. Q waves develop within 1 to 3 days because there is no depolarization current conducted from necrotic tissue (Urden et al., 2019). A new and significant Q wave is 0.04 seconds or longer and 25% of the R wave depth. An acute MI may also cause a significant decrease in the height of the R wave. During an acute MI, injury and ischemic changes are usually present. An abnormal Q wave may be present without ST-segment and T-wave changes, which indicates an old, not acute, MI. For some patients, there is no persistent ST elevation or other ECG changes; therefore, an NSTEMI is diagnosed by blood levels of cardiac biomarkers.



**Figure 23-5 •** Effects of ischemia, injury, and infarction on an electrocardiogram recording. Ischemia causes inversion of the T wave because of altered repolarization. Cardiac muscle injury causes elevation of the ST segment. Later, Q waves develop because of the absence of depolarization current from the necrotic tissue and opposing currents from other parts of the heart.



**Figure 23-6 •** Using the electrocardiogram to diagnose acute myocardial infarction (MI). (ST-segment elevation is measured 0.06 to 0.08 seconds after the J point. An elevation of more than 1 mm in contiguous leads is indicative of acute MI.)

Using the information presented, patients are diagnosed with one of the following forms of ACS:

- *Unstable angina*: The patient has clinical manifestations of coronary ischemia, but ECG and cardiac biomarkers show no evidence of acute MI.
- *STEMI*: The patient has ECG evidence of acute MI with characteristic changes in two contiguous leads on a 12-lead ECG. In this type of MI, there is a significant damage to the myocardium.
- *NSTEMI*: The patient has elevated cardiac biomarkers (e.g., troponin) but no definite ECG evidence of acute MI. In this type of MI, there may be less damage to the myocardium.

During recovery from an MI, the ST segment often is the first ECG indicator to return to normal. Q-wave alterations are usually permanent. An old STEMI is usually indicated by an abnormal Q wave or decreased height of the R wave without ST-segment and T-wave changes.

### Echocardiogram

The echocardiogram is used to evaluate ventricular function. It may be used to assist in diagnosing an MI, especially when the ECG is nondiagnostic. The echocardiogram

can detect hypokinetic and akinetic wall motion and can determine the ejection fraction (see Chapter 21).

## Laboratory Tests

Cardiac enzymes and biomarkers, which include troponin, creatine kinase (CK), and myoglobin, are used to diagnose an acute MI. Cardiac biomarkers can be analyzed rapidly, expediting an accurate diagnosis. These tests are based on the release of cellular contents into the circulation when myocardial cells die.

### Troponin

**Troponin**, a protein found in myocardial cells, regulates the myocardial contractile process. There are three isomers of troponin: C, I, and T. Troponins I and T are specific for cardiac muscle, and these biomarkers are currently recognized as reliable and critical markers of myocardial injury (Norris, 2019). An increase in the level of troponin in the serum can be detected within a few hours during acute MI. It remains elevated for a long period, often as long as 2 weeks, and it therefore can be used to detect recent myocardial damage. It should be noted that cardiac troponin levels may rise during inflammation and other forms of mechanical stress on the myocardium. These include sepsis, heart failure, and respiratory failure (Felker & Fudim, 2018).

### Creatine Kinase and Its Isoenzymes

There are three CK isoenzymes: CK-MM (skeletal muscle), CK-MB (heart muscle), and CK-BB (brain tissue). CK-MB is the cardiac-specific isoenzyme; it is found mainly in cardiac cells and therefore increases when there has been damage to these cells. Elevated CK-MB is an indicator of acute MI; the level begins to increase within a few hours and peaks within 24 hours of an infarct.

### Myoglobin

Myoglobin is a heme protein that helps transport oxygen. Like the CK-MB enzyme, myoglobin is found in cardiac and skeletal muscle. The myoglobin level starts to increase within 1 to 3 hours and peaks within 12 hours after the onset of symptoms. An increase in myoglobin is not very specific in indicating an acute cardiac event; however, negative results can be used to rule out an acute MI.

### Chart 23-7

### Treatment Guidelines for Acute Myocardial Infarction

- Use rapid transit to the hospital.
- Obtain 12-lead electrocardiogram to be read within 10 minutes.
- Obtain laboratory blood specimens of cardiac biomarkers, including troponin.
- Obtain other diagnostics to clarify the diagnosis.
- Begin routine medical interventions:
  - Supplemental oxygen
  - Nitroglycerin
  - Morphine
  - Aspirin
  - Beta-blocker
  - Angiotensin-converting enzyme inhibitor within 24 hours
  - Anticoagulation with heparin and platelet inhibitors
  - Statin
- Evaluate for indications for reperfusion therapy:
  - Percutaneous coronary intervention
  - Thrombolytic therapy
- Continue therapy as indicated:
  - IV heparin, low-molecular-weight heparin, bivalirudin, or fondaparinux
  - Clopidogrel
  - Glycoprotein IIb/IIIa inhibitor
  - Bed rest for a minimum of 12–24 hours
  - Statin prescribed at discharge.

Adapted from Ibanez, B., James, S., Agewall, S., et al. (2018). 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *European Heart Journal*, 39(2), 119–177.

## Medical Management

The goals of medical management are to minimize myocardial damage, preserve myocardial function, and prevent complications. These goals are facilitated by the use of guidelines developed by the ACC and the AHA (see [Chart 23-7](#)).

The goal for treating patients with acute MI is to minimize myocardial damage by reducing myocardial oxygen demand and increasing oxygen supply with medications, oxygen administration, and bed rest. The resolution of pain and ECG changes indicate that demand and supply are in equilibrium; they may also indicate reperfusion. Visualization of blood flow through an open vessel in the catheterization laboratory is evidence of reperfusion.

### Initial Management

The patient with suspected MI should immediately receive supplemental oxygen, aspirin, nitroglycerin, and morphine. Morphine is the drug of choice to reduce pain and anxiety. It also reduces preload and afterload, decreasing the work of the heart. The response to morphine is monitored carefully to assess for hypotension or decreased respiratory rate. Nurses should be aware that evolving research has

suggested an association between morphine and potential adverse outcomes, including larger infarct size, increased length of hospital stay, and mortality, and should stay abreast of changes to clinical guidelines impacting its use (McCarthy, Bhambhani, Pomerantsev, et al., 2018; Neto, 2018). A beta-blocker may also be used if arrhythmias occur. If a beta-blocker is not needed in the initial management period, it should be introduced within 24 hours of admission, once hemodynamics have stabilized and it is confirmed that the patient has no contraindications (Ibanez et al., 2018). Unfractionated heparin or LMWH may also be prescribed along with platelet-inhibiting agents to prevent further clot formation.

### **Emergent Percutaneous Coronary Intervention**

The patient with STEMI is taken directly to the cardiac catheterization laboratory for an immediate PCI (if a cardiac catheterization laboratory is on site). The procedure is used to open the occluded coronary artery and promote reperfusion to the area that has been deprived of oxygen. Superior outcomes have been reported with the use of PCI when compared to thrombolytic agents (Urdan et al., 2019) (also called *fibrinolytic* agents; see the Thrombolytics section). Thus, PCI is preferred as the initial treatment method for acute MI in all age groups (Urdan et al., 2019). The procedure treats the underlying atherosclerotic lesion. Because the duration of oxygen deprivation determines the number of myocardial cells that die, the time from the patient's arrival in the ED to the time PCI is performed should be less than 60 minutes. This is frequently referred to as door-to-balloon time. A cardiac catheterization laboratory and staff must be available if an emergent PCI is to be performed within this short time. The nursing care related to PCI is presented later in this chapter.

### **Thrombolytics**

Thrombolytic therapy is initiated when primary PCI is not available or the transport time to a PCI-capable hospital is too long. These agents are administered IV according to a specific protocol (see [Chart 23-8](#)). The thrombolytic agents used most often are alteplase, reteplase, and tenecteplase. The purpose of **thrombolytics** is to dissolve (i.e., lyse) the thrombus in a coronary artery (thrombolysis), allowing blood to flow through the coronary artery again (reperfusion), minimizing the size of the infarction and preserving ventricular function. However, although thrombolytics may dissolve the thrombus, they do not affect the underlying atherosclerotic lesion. The patient may be referred for a cardiac catheterization and other invasive procedures following the use of thrombolytic therapy. Thrombolytics should not be used if the patient is bleeding or has a bleeding disorder. They should be given within 30 minutes of symptom onset for best results (Norris, 2019). This is frequently referred to as door-to-needle time.

### **Inpatient Management**

Following PCI or thrombolytic therapy, continuous cardiac monitoring is indicated, preferably in a cardiac intensive care unit (ICU). Continuing pharmacologic management includes aspirin, a beta-blocker, and an angiotensin-converting enzyme (ACE) inhibitor. ACE inhibitors prevent the conversion of angiotensin I to

angiotensin II. In the absence of angiotensin II, the blood pressure decreases and the kidneys excrete sodium and fluid (diuresis), decreasing the oxygen demand of the heart. The use of ACE inhibitors in patients after MI decreases mortality rates and prevents remodeling of myocardial cells that is associated with the onset of heart failure. Blood pressure, urine output, and serum sodium, potassium, and creatinine levels need to be monitored closely. If an ACE inhibitor is not suitable, an angiotensin receptor blocker (ARB) should be prescribed (Ibanez et al., 2018). Nicotine replacement therapy and tobacco cessation counseling should also be initiated for all tobacco users.

**Chart 23-8**  **PHARMACOLOGY**

## Administration of Thrombolytic Therapy

### Indications

- Chest pain lasting more than 20 minutes, unrelieved by nitroglycerin
- ST-segment elevation in at least two leads that face the same area of the heart
- Less than 12 hours from onset of pain

### Absolute Contraindications

- Active bleeding
- Known bleeding disorder
- History of hemorrhagic stroke
- History of intracranial vessel malformation
- Recent major surgery or trauma
- Uncontrolled hypertension
- Pregnancy

### Nursing Considerations

- Minimize the number of times the patient's skin is punctured.
- Avoid intramuscular injections.
- Draw blood for laboratory tests when starting the IV line.
- Start IV lines before thrombolytic therapy; designate one line to use for blood draws.
- Avoid continual use of noninvasive blood pressure cuff.
- Monitor for acute arrhythmias and hypotension.
- Monitor for reperfusion: resolution of angina or acute ST-segment changes.
- Check for signs and symptoms of bleeding: decrease in hematocrit and hemoglobin values, decrease in blood pressure, increase in heart rate, oozing or bulging at invasive procedure sites, back pain, muscle weakness, changes in level of consciousness, complaints of headache.
- Treat major bleeding by discontinuing thrombolytic therapy and any anticoagulants; apply direct pressure and notify the primary provider immediately.
- Treat minor bleeding by applying direct pressure if accessible and appropriate; continue to monitor.

Adapted from Urden, L. D., Stacy, K. M., & Lough, M. E. (2019). *Priorities in critical care nursing* (8th ed.). St. Louis, MO: Elsevier.

## Cardiac Rehabilitation

After the patient with an MI is in a stable condition, an active rehabilitation program is initiated. Cardiac rehabilitation is an important continuing care program for patients with CAD that targets risk reduction by providing patient and family education, offering individual and group support, and encouraging physical activity and physical conditioning. The goals of rehabilitation for the patient who has had an MI are to extend life and improve the quality of life. The immediate objectives are to limit the

effects and progression of atherosclerosis, return the patient to work and a pre-illness lifestyle, enhance the patient's psychosocial and vocational status, and prevent another cardiac event. Cardiac rehabilitation programs increase survival, reduce recurrent events and the need for interventional procedures, and improve quality of life (Dickins & Braun, 2017).

Physical conditioning is achieved gradually over time. Many times, patients will "overdo it" in an attempt to achieve their goals too rapidly. Patients are observed for chest pain, dyspnea, weakness, fatigue, and palpitations and are instructed to stop exercise if any of these occur. Patients may also be monitored for an increase in heart rate above the target heart rate, an increase in systolic or diastolic blood pressure of more than 20 mm Hg, a decrease in systolic blood pressure, onset or worsening of arrhythmias, or ST-segment changes on the ECG.

Cardiac rehabilitation programs are categorized into three phases (Dickins & Braun, 2017). Phase I begins with the diagnosis of atherosclerosis, which may occur when the patient is admitted to the hospital for ACS. Because of brief hospital lengths of stay, mobilization occurs early and patient education focuses on the essentials of self-care rather than instituting behavioral changes for risk reduction. Priorities for in-hospital education include the signs and symptoms that indicate the need to call 911 (seek emergency assistance), the medication regimen, rest–activity balance, and follow-up appointments with the primary provider. The patient is reassured that although CAD is a lifelong disease and must be treated as such, they can likely resume a normal life after an MI. The amount and type of activity recommended at discharge depend on the patient's age, his or her condition before the cardiac event, the extent of the disease, the course of the hospital stay, and the development of any complications.

Phase II occurs after the patient has been discharged. The patient attends sessions three times a week for 4 to 6 weeks but may continue for as long as 6 months. The outpatient program consists of supervised, often ECG-monitored, exercise training that is individualized. At each session, the patient is assessed for the effectiveness of and adherence to the treatment. To prevent complications and another hospitalization, the cardiac rehabilitation staff alerts the referring primary provider to any problems. Phase II cardiac rehabilitation also includes educational sessions for patients and families that are given by cardiologists, exercise physiologists, dietitians, nurses, and other health care professionals. These sessions may take place outside a traditional classroom setting. For instance, a dietitian may take a group of patients to a grocery store to examine labels and meat selections or to a restaurant to discuss menu offerings for a heart-healthy diet.

Phase III is a long-term outpatient program that focuses on maintaining cardiovascular stability and long-term conditioning. The patient is usually self-directed during this phase and does not require a supervised program, although it may be offered. The goals of each phase build on the accomplishments of the previous phase.

## NURSING PROCESS

### The Patient with Acute Coronary Syndrome



#### Assessment

One of the most important aspects of care of the patient with ACS is the assessment. It establishes the patient's baseline, identifies the patient's needs, and helps determine the priority of those needs. Systematic assessment includes a careful history, particularly as it relates to symptoms: chest pain or discomfort, dyspnea (difficulty breathing), palpitations, unusual fatigue, syncope (faintness), or other possible indicators of myocardial ischemia. Each symptom must be evaluated with regard to time, duration, and the factors that precipitate the symptom and relieve it, and in comparison with previous symptoms. A focused physical assessment is critical to detect complications and any change in patient status. [Chart 23-6](#) identifies important assessments and possible findings.

Two IV lines are typically placed for any patient with ACS to ensure that access is available for administering emergency medications. Medications are administered IV to achieve rapid onset and to allow for timely adjustment. After the patient's condition stabilizes, IV lines may be changed to a saline lock to maintain IV access.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the clinical manifestations, history, and diagnostic assessment data, major nursing diagnoses may include:

- Acute pain associated with increased myocardial oxygen demand and decreased myocardial oxygen supply
- Risk for impaired cardiac function associated with reduced coronary blood flow
- Risk for hypovolaemia
- Impaired peripheral tissue perfusion associated with impaired cardiac output from left ventricular dysfunction
- Anxiety associated with cardiac event and possible death
- Lack of knowledge about post-ACS self-care

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Acute pulmonary edema (see [Chapter 25](#))
- Heart failure (see [Chapter 25](#))
- Cardiogenic shock (see [Chapter 11](#))
- Arrhythmias and cardiac arrest (see Chapters 22 and 25)
- Pericardial effusion and cardiac tamponade (see [Chapter 25](#))

#### Planning and Goals

The major goals for the patient include relief of pain or ischemic signs (e.g., ST-segment changes) and symptoms, prevention of myocardial damage, maintenance

of effective respiratory function, maintenance or attainment of adequate tissue perfusion, reduced anxiety, adherence to the self-care program, and early recognition of complications. Care of the patient with ACS who has an uncomplicated MI is summarized in the Plan of Nursing Care (see [Chart 23-9](#)).

### Nursing Interventions

#### RELIEVING PAIN AND OTHER SIGNS AND SYMPTOMS OF ISCHEMIA

Balancing myocardial oxygen supply with demand (e.g., as evidenced by the relief of chest pain) is the top priority in the care of the patient with an ACS. Although administering medications as described previously is required to accomplish this goal, nursing interventions are also important. Collaboration among the patient, nurse, and primary provider is critical in evaluating the patient's response to therapy and in altering the interventions accordingly.

Oxygen should be given along with medication therapy to assist with relief of symptoms. Administration of oxygen raises the circulating level of oxygen to reduce pain associated with low levels of myocardial oxygen. The route of administration (usually by nasal cannula) and the oxygen flow rate are documented. A flow rate of 2 to 4 L/min is usually adequate to maintain oxygen saturation levels of at least 95% unless chronic pulmonary disease is present.

Vital signs are assessed frequently as long as the patient is experiencing pain and other signs or symptoms of acute ischemia. Physical rest in bed with the head of the bed elevated or in a supportive chair helps decrease chest discomfort and dyspnea. Elevation of the head and torso is beneficial for the following reasons:

- Tidal volume improves because of reduced pressure from abdominal contents on the diaphragm and better lung expansion.
- Drainage of the upper lung lobes improves.
- Venous return to the heart (preload) decreases, reducing the work of the heart.

The pain associated with an acute MI reflects an imbalance in myocardial oxygen supply and demand or ineffective myocardial tissue perfusion. The pain also results in increases in heart rate, respiratory rate, and blood pressure. Promptly relieving the pain helps to reestablish this balance, thus decreasing the workload of the heart and minimizing damage to the myocardium. Relief of pain also helps to reduce the patient's anxiety level, which in turn reduces the sympathetic stress response, leading to a decrease in workload of the already stressed heart.

#### IMPROVING RESPIRATORY FUNCTION

Regular and careful assessment of respiratory function detects early signs of pulmonary complications. The nurse monitors fluid volume status to prevent fluid overload and encourages the patient to breathe deeply and change position frequently to maintain effective ventilation throughout the lungs. Pulse oximetry guides the use of oxygen therapy.

#### PROMOTING ADEQUATE TISSUE PERfusion

Bed or chair rest during the initial phase of treatment helps reduce myocardial oxygen consumption. This limitation on mobility should remain until the patient is

pain free and hemodynamically stable. Skin temperature and peripheral pulses must be checked frequently to monitor tissue perfusion.

#### **REDUCING ANXIETY**

Alleviating anxiety and decreasing fear are important nursing functions that reduce the sympathetic stress response. Less sympathetic stimulation decreases the workload of the heart, which may relieve pain and other signs and symptoms of ischemia.

#### **Chart 23-9 PLAN OF NURSING CARE**

Care of the Patient with an Uncomplicated Myocardial Infarction

**NURSING DIAGNOSIS:** Risk for impaired cardiac function associated with reduced coronary blood flow

**GOAL:** Relief of chest pain/discomfort

<b>Nursing Interventions</b>	<b>Rationale</b>	<b>Expected Outcomes</b>
<ol style="list-style-type: none"><li>1. Initially assess, document, and report to the primary provider the following:<ol style="list-style-type: none"><li>a. The patient's description of chest discomfort, including location, intensity, radiation, duration, and factors that affect it; other symptoms such as nausea, diaphoresis, or complaints of unusual fatigue</li><li>b. The effect of coronary ischemia on perfusion to the heart (e.g., change in blood pressure, heart rhythm), to the brain (e.g., changes in level of consciousness), to the kidneys (e.g., decrease in urine output), and to the skin (e.g., color, temperature)</li></ol></li><li>2. Obtain a 12-lead ECG recording</li></ol>	<ol style="list-style-type: none"><li>1. These data assist in determining the cause and effect of the chest discomfort and provide a baseline with which post-therapy symptoms can be compared.<ol style="list-style-type: none"><li>a. There are many conditions associated with chest discomfort. There are characteristic clinical findings of ischemic pain and symptoms.</li><li>b. Myocardial infarction (MI) decreases myocardial contractility and ventricular compliance and may produce arrhythmias. Cardiac output is reduced, resulting in reduced blood pressure and decreased organ perfusion.</li></ol></li></ol>	<ul style="list-style-type: none"><li>• Reports beginning relief of chest discomfort and symptoms</li><li>• Appears comfortable and is free of pain and other signs or symptoms</li><li>• Respiratory rate, cardiac rate, and blood pressure return to prediscomfort level</li><li>• Skin warm and dry</li><li>• Adequate cardiac output as evidenced by:<ul style="list-style-type: none"><li>• Stable/improving electrocardiogram (ECG)</li><li>• Heart rate and rhythm</li><li>• Blood pressure</li><li>• Mentation</li><li>• Urine output</li><li>• Serum blood urea nitrogen (BUN) and creatinine</li><li>• Skin color and temperature</li></ul></li><li>• No adverse effects from medications</li></ul>

- during symptomatic events, as prescribed, to assess for ongoing ischemia.
2. An ECG during symptoms may be useful in the diagnosis of ongoing ischemia.
  3. Administer oxygen as prescribed.
  4. Administer medication therapy as prescribed and evaluate the patient's response continuously.
  5. Ensure physical rest: head of bed elevated to promote comfort; diet as tolerated; the use of bedside commode; the use of stool softener to prevent straining at stool. Provide a restful environment and allay fears and anxiety by being calm and supportive. Individualize visitation, based on patient response.
3. Oxygen therapy increases the oxygen supply to the myocardium.
4. Medication therapy (nitroglycerin, morphine, beta-blocker, aspirin) is the first line of defense in preserving myocardial tissue.
5. Physical rest reduces myocardial oxygen consumption. Fear and anxiety precipitate the stress response; this results in increased levels of endogenous catecholamines, which increase myocardial oxygen consumption.

**NURSING DIAGNOSIS:** Risk for impaired cardiac function associated with left ventricular failure

**GOAL:** Absence of respiratory distress

Nursing Interventions	Rationale	Expected Outcomes
1. Initially, every 4 hours, and with chest discomfort or symptoms, assess, document, and report to the primary provider	1. These data are useful in diagnosing left ventricular failure. Diastolic filling sounds ( $S_3$ and $S_4$ ) result from	<ul style="list-style-type: none"> <li>• No shortness of breath, dyspnea on exertion, orthopnea, or paroxysmal nocturnal dyspnea</li> <li>• Respiratory rate &lt;20 breaths/min with</li> </ul>

<p>abnormal heart sounds (<math>S_3</math> and <math>S_4</math> gallop or new murmur), abnormal breath sounds (particularly crackles), decreased oxygenation, and activity intolerance.</p>	<p>decreased left ventricular compliance associated with MI. Papillary muscle dysfunction (from infarction of the papillary muscle) can result in mitral regurgitation and a reduction in stroke volume. The presence of crackles (usually at the lung bases) may indicate pulmonary congestion from increased left heart pressures. The association of symptoms and activity can be used as a guide for activity prescription and a basis for patient education.</p>	<p>physical activity and 16 breaths/min with rest</p> <ul style="list-style-type: none"> <li>• Skin color and temperature normal</li> <li>• <math>\text{SpO}_2</math>, <math>\text{PaO}_2</math>, and <math>\text{PaCO}_2</math> within normal limits</li> <li>• Heart rate &lt;100 bpm and &gt;60 bpm, with blood pressure within patient's normal limits</li> <li>• Chest x-ray unchanged</li> <li>• Appears comfortable and rested</li> </ul>
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**NURSING DIAGNOSIS:** Impaired peripheral tissue perfusion associated with impaired cardiac output

**GOAL:** Maintenance/attainment of adequate tissue perfusion

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Initially, every 4 hours, and with chest discomfort, assess, document, and report to the primary provider the following:           <ol style="list-style-type: none"> <li>a. Hypotension</li> <li>b. Tachycardia and other arrhythmia</li> <li>c. Activity intolerance</li> <li>d. Mentation changes (use</li> </ol> </li> </ol>	<ol style="list-style-type: none"> <li>1. These data are useful in determining a low cardiac output state.</li> </ol>	<ul style="list-style-type: none"> <li>• Blood pressure within the patient's normal range</li> <li>• Ideally, normal sinus rhythm without arrhythmia is maintained, or patient's baseline rhythm is maintained between 60 and 100 bpm without further arrhythmia.</li> <li>• Prescribed activity is well tolerated.</li> </ul>

- family input)
- e. Reduced urine output (<0.5 mL/kg/h)
  - f. Cool, moist, cyanotic extremities, decreased peripheral pulses, prolonged capillary refill
- Remains alert and oriented and without cognitive or behavioral change
  - Appears comfortable
  - Urine output >0.5 mL/kg/h
  - Extremities warm and dry with normal color

**NURSING DIAGNOSIS:** Anxiety associated with cardiac event

**GOAL:** Reduction of anxiety

Nursing Interventions	Rationale	Expected Outcomes
1. Assess, document, and report to the primary provider the patient's and family's level of anxiety and coping mechanisms.	1. These data provide information about psychological well-being. Causes of anxiety are variable and individual, and may include acute illness, hospitalization, pain, disruption of activities of daily living at home and at work, changes in role and self-image due to illness, and financial concerns. Because anxious family members can transmit anxiety to the patient, the nurse must also identify strategies to reduce the family's fear and anxiety.	<ul style="list-style-type: none"> <li>• Reports less anxiety</li> <li>• The patient and family discuss their anxieties and fears about illness and death.</li> <li>• The patient and family appear less anxious.</li> <li>• Appears restful, respiratory rate &lt;16 breaths/min, heart rate &lt;100 bpm without ectopic beats, blood pressure within patient's normal limits, skin warm and dry</li> <li>• Participates actively in a progressive rehabilitation program</li> <li>• Practices stress reduction techniques</li> </ul>
2. Assess the need for spiritual counseling and refer as appropriate.	2. If a patient finds support in a religion, spiritual	
3. Assess the need for social service		

referral.

counseling may assist in reducing anxiety and fear.

3. Social services can assist with posthospital care and financial concerns.

**NURSING DIAGNOSIS:** Lack of knowledge about post-MI self-care

**GOAL:** Adheres to the home health care program; chooses lifestyle consistent with heart-healthy recommendations (see [Chart 23-10](#)).

The development of a trusting and caring relationship with the patient is critical in reducing anxiety. Providing information to the patient and family in an honest and supportive manner encourages the patient to be a partner in care and greatly assists in developing a positive relationship. Other interventions that can be used to reduce anxiety include ensuring a quiet environment, preventing interruptions that disturb sleep, and providing spiritual support consistent with the patient's beliefs. The nurse provides frequent opportunities for the patient to privately share concerns and fears. An atmosphere of acceptance helps the patient know that these concerns and fears are both realistic and normal. Alternative therapies such as pet therapy can help certain patients relax and reduce anxiety (Waite, Hamilton, & O'Brien, 2018). Many hospitals have developed infection control and safety procedures pertaining to the animals, their handlers, and the patients eligible for pet therapy.

**MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Complications that can occur after acute MI are caused by the damage that occurs to the myocardium and to the conduction system from reduced coronary blood flow. Because these complications can be life-threatening, close monitoring for and early identification of their signs and symptoms are critical (see [Chart 23-9](#)).

The nurse monitors the patient closely for changes in cardiac rate and rhythm, heart sounds, blood pressure, chest pain, respiratory status, urinary output, skin color and temperature, mental status, ECG changes, and laboratory values. Any changes in the patient's condition must be reported promptly to the primary provider and emergency measures instituted when necessary.

**PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** The most effective way to increase the probability that the patient will implement a self-care regimen after discharge is to identify the patient's priorities, provide adequate education about heart-healthy living, and facilitate the patient's involvement in a cardiac rehabilitation program (Ibanez et al., 2018). Patient participation in the development of an individualized program enhances the potential for an effective treatment plan (see [Chart 23-10](#)).

**Continuing and Transitional Care.** Depending on the patient's condition and the availability of family assistance, home, community-based, or transitional, care may be indicated. The nurse making a home visit can assist the patient with scheduling and keeping follow-up appointments and with adhering to the prescribed cardiac rehabilitation regimen. The patient may need reminders about follow-up monitoring, including periodic laboratory testing, as well as ongoing assessment of cardiac status. In addition, the home health nurse monitors the patient's adherence to dietary restrictions and to prescribed medications. If the patient is receiving home oxygen, the nurse ensures that the patient is using the oxygen as prescribed and that appropriate home safety measures are maintained. If the patient has evidence of heart failure secondary to an MI, appropriate home care guidelines for the patient with heart failure are followed (see [Chapter 25](#)).

### Evaluation

Expected patient outcomes may include:

1. Experiences relief of angina
2. Has stable cardiac and respiratory status
3. Maintains adequate tissue perfusion
4. Exhibits decreased anxiety
5. Adheres to a self-care program
6. Has no complications

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## INVASIVE CORONARY ARTERY PROCEDURES

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Methods to reperfuse ischemic myocardial tissue when patients are refractory to more conservative management methods include PCIs and CABG surgery, as noted previously. The following sections discuss specific indications for each of these and the nursing management of patients who are having either PCIs or CABGs.

### Percutaneous Coronary Interventions

Invasive interventional procedures to treat CAD include PTCA and intracoronary stent implantation. These procedures are classified as **percutaneous coronary interventions (PCIs)**, as they are performed through a skin puncture rather than a surgical incision.

### Percutaneous Transluminal Coronary Angioplasty

In **percutaneous transluminal coronary angioplasty (PTCA)**, a balloon-tipped catheter is used to open blocked coronary vessels and resolve ischemia. It is used in patients with angina and as an intervention for ACS. Catheter-based interventions can also be used to open blocked CABGs (see later discussion). The purpose of PTCA is to improve blood flow within a coronary artery by compressing the atheroma. The

procedure is attempted when the interventional cardiologist determines that PTCA can improve blood flow to the myocardium.

**Chart 23-10**  **HEALTH PROMOTION**

## Promoting Health After Myocardial Infarction and Other Acute Coronary Syndromes

To extend and improve the quality of life, a patient who has had a myocardial infarction (MI) must make lifestyle adjustments to promote heart-healthy living. With this in mind, the nurse and patient develop a program to help achieve desired outcomes.

### Making Lifestyle Modifications during Convalescence and Healing

Adaptation to an MI is an ongoing process and usually requires some modification of lifestyle. Educate patients to make the following specific modifications:

- Avoid any activity that produces chest pain, extreme dyspnea, or undue fatigue.
- Avoid extremes of heat and cold and walking against the wind.
- Lose weight, if indicated.
- Stop smoking and the use of tobacco; avoid secondhand smoke.
- Develop heart-healthy eating patterns and avoid large meals and hurrying while eating.
- Modify meals to align with the AHA dietary recommendations, the Mediterranean diet, or other recommended diets.
- Adhere to medical regimen, especially in taking medications.
- Follow recommendations that ensure that blood pressure and blood glucose are in control.
- Pursue activities that relieve and reduce stress.

### Adopting an Activity Program

In addition, the patient needs to undertake a structured program of activity and exercise for long-term rehabilitation. Advise patients to:

- Engage in a regimen of physical conditioning with a gradual increase in activity duration and then a gradual increase in activity intensity.
- Enroll in a cardiac rehabilitation program.
- Walk daily, increasing distance and time as prescribed.
- Monitor pulse rate during physical activity.
- Avoid physical exercise immediately after a meal.
- Alternate activity with rest periods (some fatigue is normal and expected during convalescence).
- Participate in a daily program of exercise that develops into a program of regular exercise for a lifetime.

### Managing Symptoms

The patient must learn to recognize and take appropriate action for recurrent symptoms. Make sure that patients know to do the following:

- Call 911 if chest pressure or pain (or prodromal symptoms) is not relieved in 15 minutes by taking 3 nitroglycerin tablets at 5-minute intervals.
- Contact the primary provider if any of the following occur: shortness of breath, fainting, slow or rapid heartbeat, swelling of feet and ankles.

PTCA is carried out in the cardiac catheterization laboratory. Hollow catheters called *sheaths* are inserted, usually in the femoral artery (and sometimes the radial artery), providing a conduit for other catheters. Catheters are then threaded through the femoral or radial artery, up through the aorta, and into the coronary arteries. Angiography is performed using injected radiopaque contrast agents (commonly called *dye*) to identify the location and extent of the blockage. A balloon-tipped dilation catheter is passed through the sheath and positioned over the lesion. The physician determines the catheter position by examining markers on the balloon that can be seen with fluoroscopy. When the catheter is properly positioned, the balloon is inflated with high pressure for several seconds and then deflated. The pressure compresses and often “cracks” the atheroma (see Fig. 23-7). The media and adventitia of the coronary artery are also stretched.

Several inflations with different balloon sizes may be required to achieve the goal, usually defined as an improvement in blood flow and a residual stenosis of less than 10% (Urden et al., 2019). Other measures of the success of PTCA are an increase in the artery’s lumen and no clinically obvious arterial trauma. Because the blood supply to the coronary artery decreases while the balloon is inflated, the patient may complain of chest pain and the ECG may display ST-segment changes. Intracoronary stents are usually positioned in the intima of the vessel to maintain patency of the artery after the balloon is withdrawn.

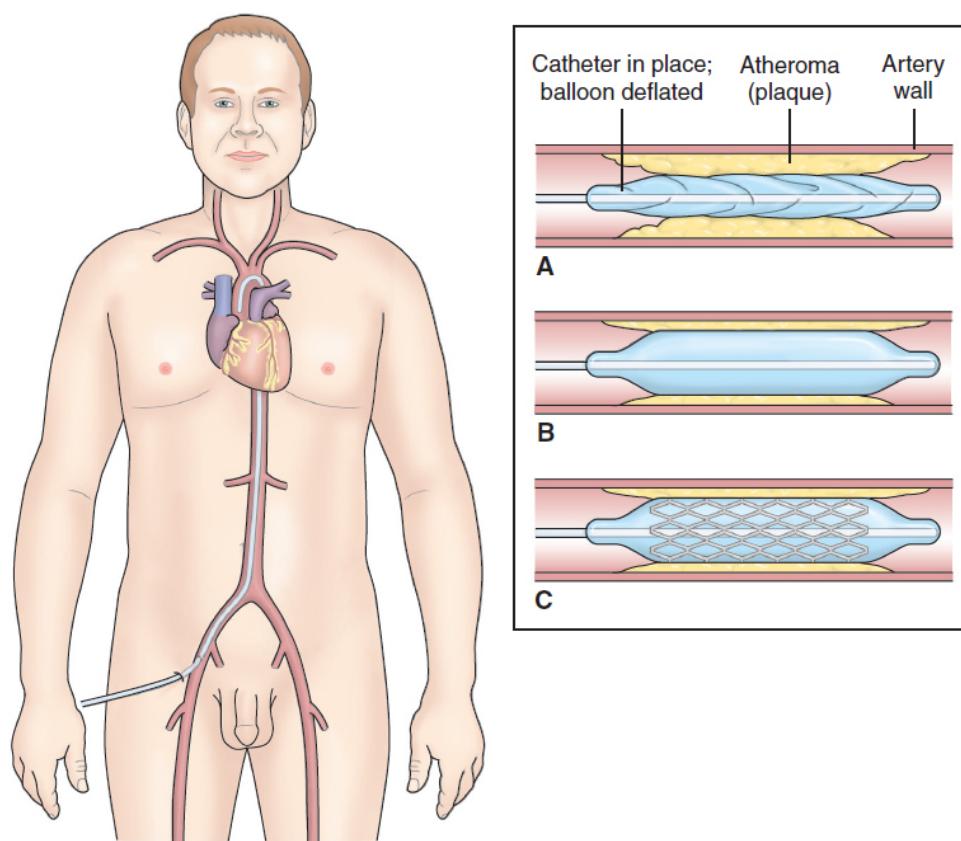
If thick, deep, or concentric calcification is present, the lesion may require the use of devices such as cutting, scoring, or high pressure balloons, rotational atherectomy, orbital atherectomy and excimer lasers to prepare the lesion for stenting (Shlofmitz, Shlofmitz, & Lee, 2019).

In addition to these approaches, intravascular lithotripsy (IVL) is currently being investigated to treat calcified artery blockages with sonic pressure waves in a similar way that is used to treat kidney stones. Pulsatile sonic pressure waves are used during balloon inflation to fracture both intimal and medial calcium in the artery wall but pass through the surrounding soft vascular tissue in a safe manner. This technology is approved for use in peripheral arteries at this time. Further studies are being done to assess their efficacy in coronary arteries (Riley, Corl, & Kereiakes, 2019).

## Coronary Artery Stent

After PTCA, the area that has been treated may close off partially or completely—a process called *restenosis*. The intima of the coronary artery has been injured and responds by initiating an acute inflammatory process. This process may include release of mediators that leads to vasoconstriction, clotting, and scar tissue formation. A coronary artery stent may be placed to overcome these risks. A **stent** is a metal

mesh that provides structural support to a vessel at risk of acute closure. The stent is initially positioned over the angioplasty balloon. When the balloon is inflated, the mesh expands and presses against the vessel wall, holding the artery open. The balloon is withdrawn, but the stent is left permanently in place within the artery (see Fig. 23-7). Eventually, endothelium covers the stent and it is incorporated into the vessel wall. The original stents do not contain medications and are known as bare metal stents. Some stents are coated with medications, such as sirolimus or paclitaxel, which may minimize the formation of thrombi or scar tissue within the coronary artery lesion. These drug-eluting stents (DES) have increased the success of PCI (Mishra, Edla, Tripathi, et al., 2019). Because of the risk of thrombus formation within the stent, the patient receives antiplatelet medications, usually aspirin and clopidogrel. Aspirin should be continued indefinitely and clopidogrel is continued for 1 year following stent placement (Urden et al., 2019).



**Figure 23-7 •** Percutaneous transluminal coronary angioplasty. **A.** A balloon-tipped catheter is passed into the affected coronary artery and placed across the area of the atheroma (plaque). **B.** The balloon is then rapidly inflated and deflated with controlled pressure. **C.** A stent is placed to maintain patency of the artery, and the balloon is removed.

## Complications

Complications that can occur during a PCI procedure include coronary artery dissection, perforation, abrupt closure, or vasospasm. Additional complications

include acute MI, serious arrhythmias (e.g., ventricular tachycardia), and cardiac arrest. Some of these complications may require emergency surgical treatment. Complications after the procedure may include abrupt closure of the coronary artery and a variety of vascular complications, such as bleeding at the insertion site, retroperitoneal bleeding, hematoma, and arterial occlusion (Urden et al., 2019). In addition, there is a risk of acute kidney injury from the contrast agent used during the procedure (see [Table 23-3](#)).

## Postprocedure Care

Patient care is similar to that for a diagnostic cardiac catheterization (see [Chapter 21](#)). Patients who are not already hospitalized are admitted the day of the PCI. Those with no complications go home the same day. When the PCI is performed emergently to treat ACS, patients typically go to a critical care unit and stay in the hospital for a few days. During the PCI, patients receive IV heparin or a thrombin inhibitor (e.g., bivalirudin) and are monitored closely for signs of bleeding. Patients may also receive a GP IIb/IIIa agent (e.g., eptifibatide) for several hours following the PCI to prevent platelet aggregation and thrombus formation in the coronary artery (Urden et al., 2019). Hemostasis is achieved, and femoral sheaths may be removed at the end of the procedure by using a vascular closure device (e.g., Angio-Seal, VasoSeal) or a device that sutures the vessels. Hemostasis after sheath removal may also be achieved by direct manual pressure, a mechanical compression device (e.g., C-shaped clamp), or a pneumatic compression device (e.g., FemoStop).

Patients may return to the nursing unit with the large peripheral vascular access sheaths in place. The sheaths are then removed after blood studies (e.g., activated clotting time) indicate that the heparin is no longer active and the clotting time is within an acceptable range. This usually takes a few hours, depending on the amount of heparin given during the procedure. The patient must remain flat in bed and keep the affected leg straight until the sheaths are removed and then for a few hours afterward to maintain hemostasis. Because immobility and bed rest may cause discomfort, treatment may include analgesics and sedation. Nonpharmacologic interventions include repositioning and heat application for back pain. Sheath removal and the application of pressure on the vessel insertion site may cause the heart rate to slow and the blood pressure to decrease (vasovagal response). A dose of IV atropine is usually given to treat this response.

**TABLE 23-3** Complications After Percutaneous Coronary Interventions

Complication	Clinical Manifestations	Possible Causes	Nursing Actions
Myocardial ischemia	Chest pain Ischemic changes on ECG Arrhythmias	Thrombosis Restenosis of coronary artery	Administer oxygen and nitroglycerin. Obtain 12-lead ECG. Notify cardiologist.
Bleeding and hematoma formation	Continuation of bleeding from vascular access site Swelling at site Formation of hard lump Pain with leg movement Possible hypotension and tachycardia	Anticoagulant therapy Vascular trauma Inadequate hemostasis Leg movement	Keep patient on bed rest. Apply manual pressure over sheath insertion site. Outline hematoma with marking pen. Notify primary provider if bleeding continues.
Retroperitoneal hematoma	Back, flank, or abdominal pain Hypotension Tachycardia Restlessness, agitation	Arterial leak of blood into the retroperitoneal space	Notify primary provider. Stop anticoagulants. Administer IV fluids. Anticipate diagnostic testing (e.g., computed tomography scan). Prepare patient for intervention.
Arterial occlusion	Lost/weakened pulse distal to sheath insertion site Extremity cool, cyanotic, painful	Arterial thrombus or embolus	Notify primary provider. Anticipate intervention.
Pseudoaneurysm formation	Swelling at vascular access site Pulsatile mass, bruit	Vessel trauma during the procedure	Notify primary provider. Anticipate intervention.
Arteriovenous fistula formation	Swelling at vascular access site Pulsatile mass, bruit	Vessel trauma during the procedure	Notify primary provider. Anticipate intervention.
Acute kidney injury	Decreased urine output Elevated BUN, serum creatinine	Nephrotoxic contrast agent	Monitor urine output, BUN, creatinine, electrolytes. Provide adequate hydration. Administer renal protective agents (acetylcysteine) before and after procedure as prescribed.

BUN, blood urea nitrogen; ECG, electrocardiogram; IV, intravenous.

Adapted from Urden, L. D., Stacy, K. M., & Lough, M. E. (2019). *Priorities in critical care nursing* (8th ed.). St. Louis, MO: Elsevier.

Some patients with unstable lesions and at high risk for abrupt vessel closure are restarted on heparin after sheath removal, or they receive an IV infusion of a GP IIb/IIIa inhibitor. These patients are monitored closely and may have a delayed recovery period.

After hemostasis is achieved, a pressure dressing is applied to the site. Patients resume self-care and ambulate unassisted within a few hours of the procedure. The duration of immobilization depends on the size of the sheath inserted, the type of anticoagulant given, the method of hemostasis, the patient's condition, and the physician's preference. On the day after the procedure, the site is inspected and the dressing removed. The patient is instructed to monitor the site for bleeding or development of a hard mass indicative of hematoma.



## Surgical Procedures: Coronary Artery Revascularization

Advances in diagnostics, medical management, and surgical and anesthesia techniques, as well as the care provided in critical care and surgical units, home care, and rehabilitation programs, have continued to make surgery an effective treatment option for patients with CAD. CAD has been treated by myocardial revascularization since the 1960s, and the most common CABG techniques have been performed for more than 40 years. **Coronary artery bypass graft (CABG)** is a surgical procedure in which a blood vessel is grafted to an occluded coronary artery so that blood can flow beyond the occlusion; it is also called a *bypass graft*.

The major indications for CABG are:

- Alleviation of angina that cannot be controlled with medication or PCI
- Treatment for left main coronary artery stenosis or multivessel CAD
- Prevention of and treatment for MI, arrhythmias, or heart failure
- Treatment for complications from an unsuccessful PCI

The recommendation for CABG is determined by a number of factors, including the number of diseased coronary vessels, the degree of left ventricular dysfunction, the presence of other health problems, the patient's symptoms, and any previous treatment. CABG and PCI have shown similar results in outcomes, such as MI rate, mortality, and improvement of angina post-intervention. However, the requirement of a second reperfusion intervention has been shown to be lower with CABG compared to PCI therapy (Gaudino, Spadaccio, & Taggart, 2019).

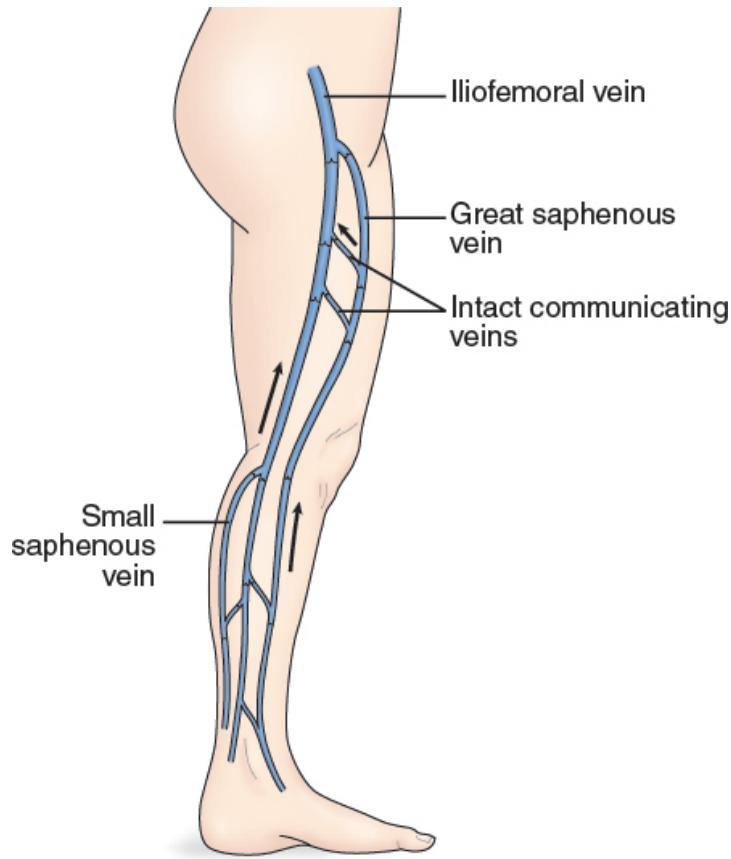
CABG is performed less frequently in women (Angraal, Khera, Wang, et al., 2018). Compared with men, women referred for this surgery tend to be older and have more comorbidities such as diabetes. In addition, they have a higher risk of surgical complications and increased mortality (Angraal et al., 2018). Although some women have good outcomes following CABG, men generally have a better rate of graft patency and symptom relief.

For a patient to be considered for CABG, the coronary arteries to be bypassed must have at least a 70% occlusion, or at least a 50% occlusion in the left main coronary artery (Urden et al., 2019). If significant blockage is not present, flow

through the artery will compete with flow through the bypass, and circulation to the ischemic area of myocardium may not improve. The artery also must be patent beyond the area of blockage or the flow through the bypass will be impeded.

Current guidelines recommend use of the internal thoracic arteries (formerly called the internal mammary arteries) for CABG, because of their histologic characteristics and increased production of vasoactive molecules and anti-inflammatory cytokines which improve arterial patency. Recent studies demonstrate increased survival when using internal thoracic artery grafting. The left internal thoracic artery graft has been shown to have greater than 90% patency after 20 years and is the recommended conduit to use first (Gaudino et al., 2019). Arterial grafts are preferred to venous grafts because they do not develop atherosclerotic changes as quickly and remain patent longer. The surgeon leaves the proximal end of the thoracic artery intact and detaches the distal end of the artery from the chest wall. This end of the artery is then grafted to the coronary artery distal to the occlusion. The internal thoracic arteries may not be long enough to use for multiple bypasses. Because of this, many CABG procedures are performed with a combination of venous and arterial grafts.

A vein commonly used for CABG is the greater saphenous vein, followed by the lesser saphenous vein (see [Fig. 23-8](#)). The vein is removed from the leg and grafted to the ascending aorta and to the coronary artery distal to the lesion. Traditionally, a skin incision was made over the length of vein segment, but new techniques allow small leg incisions. Endovascular methods of vein harvesting have reduced complications such as infection and wound dehiscence, which are associated with longer leg incisions (Gaudino et al., 2019). Lower extremity edema continues to be a common adverse effect of vein removal. The degree of edema varies and usually diminishes over time. The patency of vein grafts can be limited. Within 5 to 10 years, atherosclerotic changes often develop in saphenous vein grafts.



**Figure 23-8 •** The greater and lesser saphenous veins are commonly used in bypass graft procedures.

## Traditional Coronary Artery Bypass Graft

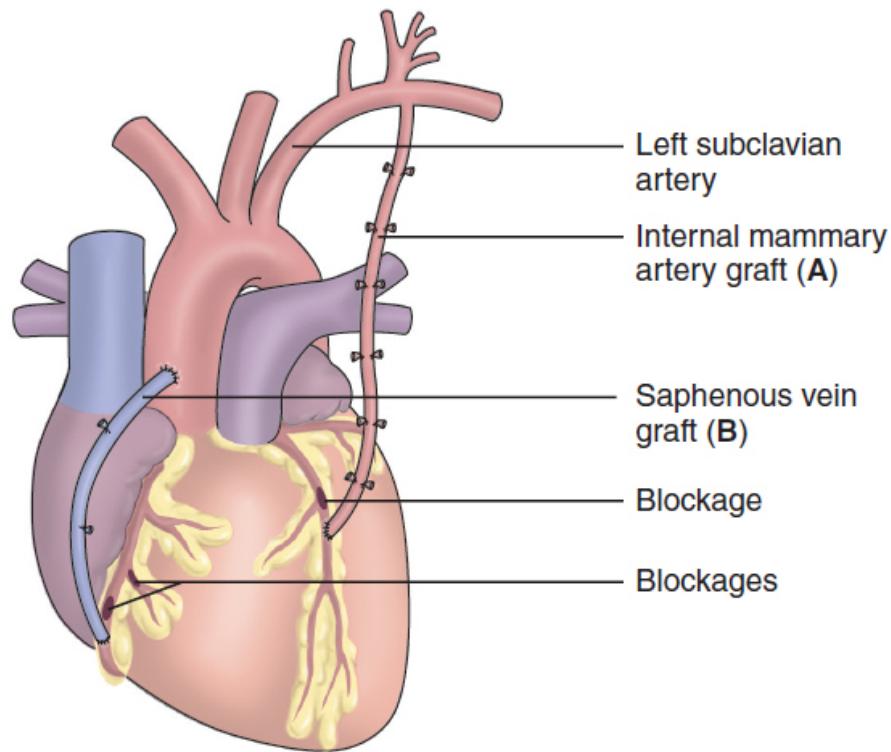
CABG procedures are performed with the patient under general anesthesia. In the traditional CABG procedure, the surgeon performs a median sternotomy and connects the patient to the cardiopulmonary bypass (CPB) machine. Next, a blood vessel from another part of the patient's body (e.g., saphenous vein, left internal thoracic artery) is grafted distal to the coronary artery lesion, bypassing the obstruction (see Fig. 23-9). CPB is then discontinued, chest tubes and epicardial pacing wires are placed, and the incision is closed. The patient is then admitted to a critical care unit.

### Cardiopulmonary Bypass

Many cardiac surgical procedures are possible because of CPB (i.e., extracorporeal circulation). The procedure mechanically circulates and oxygenates blood for the body while bypassing the heart and lungs. CPB maintains perfusion to body organs and tissues and allows the surgeon to complete the anastomoses in a motionless, bloodless surgical field.

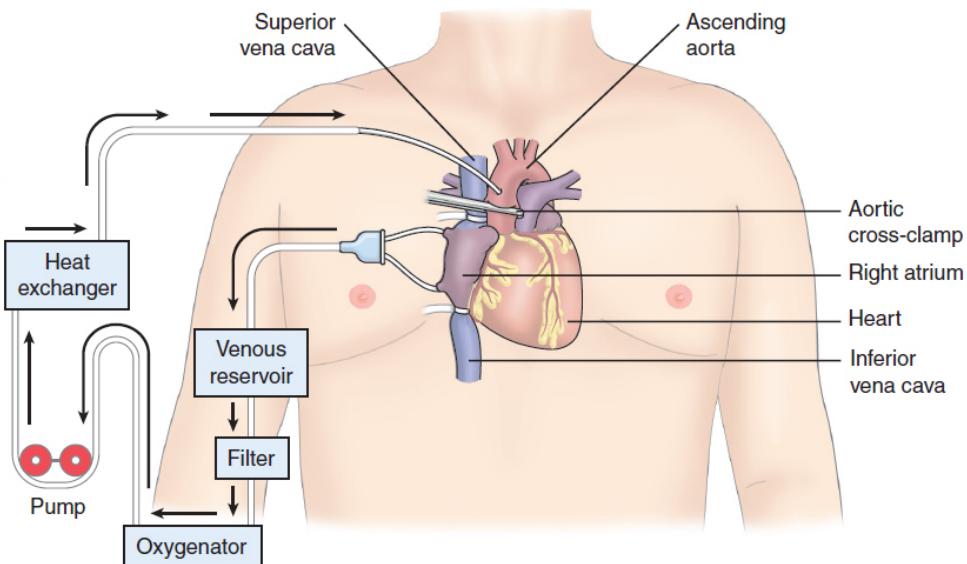
CPB is accomplished by placing a cannula in the right atrium, vena cava, or femoral vein to withdraw blood from the body. The cannula is connected to tubing filled with an isotonic crystalloid solution. Venous blood removed from the body by the cannula is filtered, oxygenated, cooled or warmed by the machine, and then

returned to the body. The cannula used to return the oxygenated blood is usually inserted in the ascending aorta, or it may be inserted in the femoral artery (see Fig. 23-10). The heart is stopped by the injection of a potassium-rich cardioplegia solution into the coronary arteries. The patient receives heparin to prevent clotting and thrombus formation in the bypass circuit when blood comes in contact with the surfaces of the tubing. At the end of the procedure when the patient is disconnected from the bypass machine, protamine sulfate is given to reverse the effects of heparin.



**Figure 23-9 •** Coronary artery bypass grafts. One or more procedures may be performed using various veins and arteries. **A.** Left internal thoracic artery (formerly called the left internal mammary artery), used frequently because of its functional longevity. **B.** Saphenous vein, also used as bypass graft.

During the procedure, hypothermia is maintained at a temperature of about 28°C (82.4°F) (Urden et al., 2019). The blood is cooled during CPB and returned to the body. The cooled blood slows the body's basal metabolic rate, thereby decreasing the demand for oxygen. Cooled blood usually has a higher viscosity, but the crystalloid solution used to prime the bypass tubing dilutes the blood. When the surgical procedure is completed, the blood is rewarmed as it passes through the CPB circuit. Urine output, arterial blood gases, electrolytes, and coagulation studies are monitored to assess the patient's status during CPB.



**Figure 23-10 •** The cardiopulmonary bypass system, in which cannulas are placed through the right atrium into the superior and inferior vena cavae to divert blood from the body and into the bypass system. The pump system creates a vacuum, pulling blood into the venous reservoir. The blood is cleared of air bubbles, clots, and particulates by the filter and then is passed through the oxygenator, releasing carbon dioxide and obtaining oxygen. Next, the blood is pulled to the pump and pushed out to the heat exchanger, where its temperature is regulated. The blood is then returned to the body via the ascending aorta.

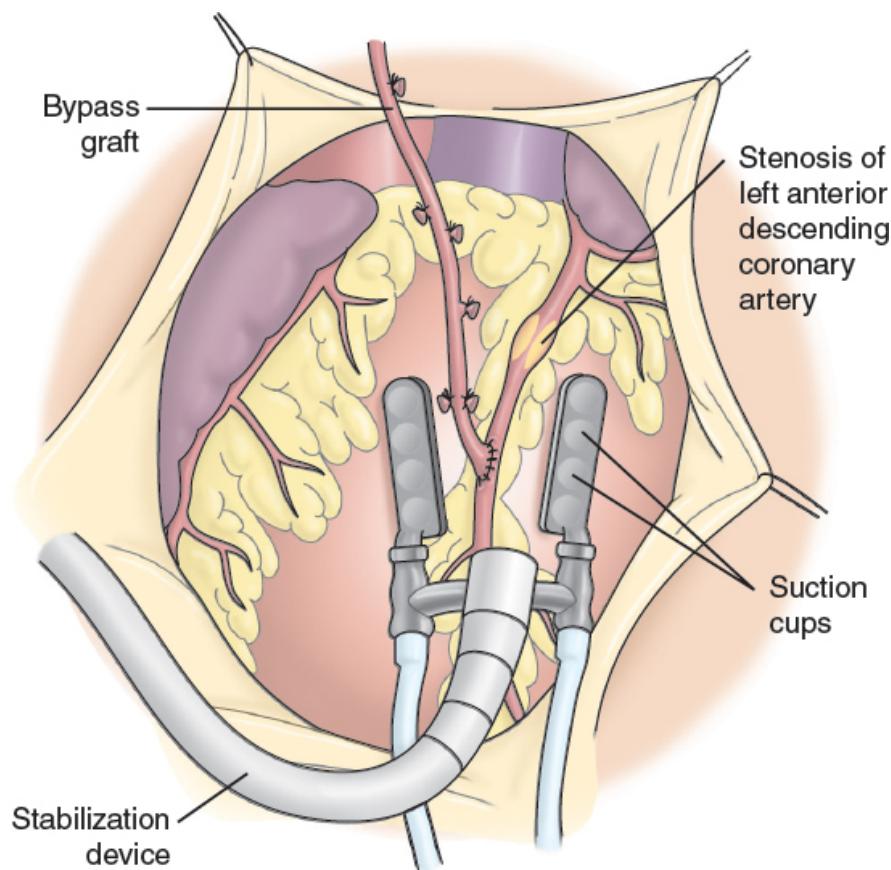
## Alternative Coronary Artery Bypass Graft Techniques

A number of alternative CABG techniques have been developed that may have fewer complications for some groups of patients. Off-pump coronary artery bypass (OPCAB) surgery has been used successfully in many patients. OPCAB involves a standard median sternotomy incision, but the surgery is performed without CPB. A beta-adrenergic blocker may be used to slow the heart rate. The surgeon also uses a myocardial stabilization device to hold the site still for the anastomosis of the bypass graft into the coronary artery while the heart continues to beat (see Fig. 23-11). Research suggests that OPCAB is associated with reduced short-term postoperative morbidity, including stroke and other complications. However, with on-pump CABG, graft patency rate is higher and long-term mortality may be lower (Gaudino et al., 2019).

Minimally invasive surgical techniques that eliminate median sternotomy have also been developed. These endoscopic techniques use smaller incisions via a right or left thoracotomy approach and a robotic system to place bypass grafts. The patient may or may not require CPB (Snyder, 2018). Minimally invasive heart surgery may be considered an acceptable alternative to conventional CABG for select patients, such as those who do not require bypass grafts to several vessels. It is most commonly used to bypass occlusions in the left anterior descending artery (Snyder, 2018). It has allowed patients to recover earlier, require fewer blood transfusions,

experience fewer respiratory complications, and be less likely to experience acute kidney injury (Urden et al., 2019).

The most important criterion when deciding whether a patient needs a CABG or a PCI is the predicted surgical mortality, which takes into consideration the patient's individual characteristics, the anatomic complexity of the coronary lesions, and the ability to achieve revascularization. The cardiac surgeon will assess the following factors to determine risk and the ability to revascularize: clinical history (age, sex, diabetes, hypertension, left ventricular function, arrhythmias), previous cardiovascular events (previous cardiovascular surgery, PCI, MI, or stroke), and disease complexity (number of diseased vessels, concomitant valve disease). In some cases, CABG may still be recommended over PCI for only one lesion to achieve better revascularization (Gaudino et al., 2019).



**Figure 23-11 •** Stabilizer device for off-pump coronary artery bypass surgery.

## Complications of Coronary Artery Bypass Graft

CABG may result in complications such as hemorrhage, arrhythmias, and MI (see Table 23-4). The patient may require interventions for more than one complication at a time. Collaboration among nurses, physicians, pharmacists, respiratory therapists, and dietitians is necessary to achieve the desired patient outcomes. Although most patients improve symptomatically following surgery, CABG is not a cure for CAD,

and angina, exercise intolerance, or other symptoms experienced before CABG may recur. Medications required before surgery may need to be continued. Lifestyle modifications recommended before surgery remain important to treat the underlying CAD and for the continued viability of the newly implanted grafts.



## Nursing Management

Cardiac surgery patients have many of the same needs and require the same perioperative care as other surgical patients (see Unit 3), as well as some special needs.

### Preoperative Management

Comprehensive preoperative medical management prevents complications and improves outcomes. This is particularly important because patients undergoing CABG surgery tend to be older and often have multiple comorbidities. The use of aspirin, beta-blockers, and statins during the preoperative period is associated with better outcomes. Preoperative use of aspirin is associated with a reduction in perioperative morbidity and mortality (Aboul-Hassan, Stankowski, Marczak, et al., 2017). Beta-blockers, when given at least 24 hours before CABG, reduce the incidence of postoperative atrial fibrillation (Urden et al., 2019). Perioperative use of statins has been shown to reduce the rates of postoperative MI, atrial fibrillation, neurologic dysfunction, renal dysfunction, infection, and death (Katsiki, Triposkiadis, Giannoukas, et al., 2018).

### Assessing the Patient

Patients are frequently admitted to the hospital the day of the procedure. Therefore, most of the preoperative evaluation is completed in the physician's office and during preadmission testing.

Nursing and medical personnel perform a history and physical examination. Preoperative testing consists of a chest x-ray; ECG; laboratory tests, including coagulation studies; and blood typing and cross-matching. The preoperative history and health assessment should be thorough and well documented because they provide a basis for postoperative comparison. The nurse assesses the patient for disorders that could complicate or affect the postoperative course, such as diabetes, hypertension, and lung disease.

**TABLE 23-4** Potential Complications of Cardiac Surgery

Complication	Cause	Assessment and Management
<b>Cardiac Complications</b>		
Hypovolemia (most common cause of decreased cardiac output after cardiac surgery)	Net loss of blood and intravascular volume Vasodilation due to postoperative rewarming Intravascular fluid loss to the interstitial spaces because surgery and anesthesia increase capillary permeability	Arterial hypotension, tachycardia, low CVP, and low PAWP are often seen. Fluid replacement may be prescribed. Replacement fluids include colloid (albumin), packed red blood cells, or crystalloid solution (normal saline, lactated Ringer's solution).
Persistent bleeding	Cardiopulmonary bypass causes platelet dysfunction, and hypothermia alters clotting mechanisms. Surgical trauma causes tissues and blood vessels to ooze bloody drainage. Intraoperative anticoagulant (heparin) therapy. Postoperative coagulopathy may also result from liver dysfunction and depletion of clotting components.	Accurate measurement of wound bleeding and chest tube blood is essential. Drainage should not exceed 200 mL/h for the first 4–6 h. Drainage should decrease and stop within a few days, while progressing from serosanguinous to serous. Serial hemoglobin, hematocrit, and coagulation studies guide therapy. Administration of blood products: packed red blood cells, fresh frozen plasma, platelet concentrate, recombinant factor VII Protamine sulfate may be given to neutralize unfractionated heparin. Administration of desmopressin acetate (DDAVP) to enhance platelet function If bleeding persists, the patient may return to the operating room.
Cardiac tamponade	Fluid and clots accumulate in the pericardial sac, which compress the heart, preventing blood from filling the ventricles.	Signs and symptoms include arterial hypotension, tachycardia, decreased urine output, and ↑ CVP. Arterial pressure waveform may show pulsus paradoxus (decrease of >10 mm Hg systolic blood pressure during inspiration). The chest drainage system is checked to eliminate possible kinks or obstructions in the tubing. Chest x-ray may show a widening mediastinum. Bedside echocardiogram may be done to confirm tamponade. Emergency medical management is required; may include return to surgery.
Fluid overload	IV fluids and blood products increase circulating volume.	High CVP and pulmonary artery pressures, as well as crackles, indicate fluid overload. Diuretics are prescribed, and the rate of IV fluid administration is reduced.

Hypothermia	Low body temperature leads to vasoconstriction, shivering, and arterial hypertension.	Alternative treatments include continuous renal replacement therapy and dialysis. Patient is rewarmed gradually after surgery, decreasing vasoconstriction.
Hypertension	Results from postoperative vasoconstriction. It may stretch suture lines and cause postoperative bleeding. The condition is usually transient.	Vasodilators (nitroglycerin, nitroprusside) may be used to treat hypertension. Administer cautiously to avoid hypotension.
Tachyarrhythmias	Increased heart rate is common with perioperative volume changes. Rapid atrial fibrillation commonly occurs during the first few days postoperatively.	If a tachyarrhythmia is the primary problem, the heart rhythm is assessed and medications (e.g., amiodarone, diltiazem) may be prescribed. Antiarrhythmic agents (e.g., beta-blockers) are often given before coronary artery bypass graft to minimize the risk. Cardioversion and defibrillation are alternatives for symptomatic tachyarrhythmias.
Bradycardias	Decreased heart rate due to surgical trauma and edema affecting the cardiac conduction system	Many postoperative patients have temporary pacer wires that can be attached to an external pacemaker to stimulate the heart to beat faster. Less commonly, atropine or other medications may be used to increase heart rate.
Cardiac failure	Myocardial contractility may be decreased perioperatively.	The nurse observes for and reports signs of heart failure, including hypotension, ↑ CVP, ↑ PAWP, venous distention; labored respirations; and edema. Medical management includes diuretics and IV inotropic agents.
MI (may occur intraoperatively or postoperatively)	Portion of the cardiac muscle dies; therefore, contractility decreases. Impaired ventricular wall motion further decreases cardiac output. Symptoms may be masked by the postoperative surgical discomfort or the anesthesia-analgesia regimen.	Careful assessment to determine the type of pain the patient is experiencing; MI is suspected if the mean blood pressure is low with normal preload. Serial electrocardiograms and cardiac biomarkers assist in making the diagnosis (alterations may be due to the surgical intervention).
<b>Pulmonary Complications</b>		
Impaired gas exchange	During and after anesthesia, patients require mechanical assistance to breathe. Anesthetic agents stimulate production of mucus, and chest	Pulmonary complications are detected during assessment of breath sounds, oxygen saturation levels, arterial blood gases, and ventilator readings. Extended periods of mechanical ventilation may be required while

	<p>incision pain may decrease the effectiveness of ventilation.</p> <p>Potential for postoperative atelectasis</p>	complications are treated.
<b>Neurologic Complications</b>		
Neurologic changes; stroke	<p>Thrombi and emboli may cause cerebral infarction, and neurologic signs may be evident when patients recover from anesthesia.</p>	<p>Inability to follow simple commands within 6 h of recovery from anesthetic; weakness on one side of body or other neurologic changes may indicate stroke.</p> <p>Patients who are older or who have renal or hepatic failure may take longer to recover from anesthesia.</p>
<b>Kidney Injury and Electrolyte Imbalance</b>		
Acute kidney injury	<p>May result from hypoperfusion of the kidneys or from injury to the renal tubules by nephrotoxic drugs</p>	<p>May respond to diuretics or may require continuous renal replacement therapy or dialysis.</p> <p>Fluids, electrolytes, and urine output are monitored frequently.</p> <p>May result in chronic kidney disease and require ongoing dialysis.</p>
Electrolyte imbalance	<p>Postoperative imbalances in potassium, magnesium, sodium, calcium, and blood glucose are related to surgical losses, metabolic changes, and the administration of medications and IV fluids.</p>	<p>Monitor electrolytes and basic metabolic studies frequently.</p> <p>Implement treatment to correct electrolyte imbalance promptly (see Chart 23-11).</p>
<b>Other Complications</b>		
Hepatic failure	<p>Surgery and anesthesia stress the liver. Most common in patients with cirrhosis, hepatitis, or prolonged right-sided heart failure.</p>	<p>The use of medications metabolized by the liver must be minimized.</p> <p>Bilirubin and albumin levels are monitored, and nutritional support is provided.</p>
Infection	<p>Surgery and anesthesia alter the patient's immune system. Multiple invasive devices used to monitor and support the patient's recovery may serve as a source of infection.</p>	<p>Monitor for signs of possible infection: body temperature, white blood cell and differential counts, incision and puncture sites, urine (clarity, color, and odor), bilateral breath sounds, sputum (color, odor, amount).</p> <p>Antibiotic therapy may be instituted or modified as necessary.</p> <p>Continuous insulin infusion to maintain blood glucose concentrations to <math>\leq 180</math> mg/dL while avoiding hypoglycemia may reduce the incidence of deep sternal wound infections.</p> <p>Invasive devices are discontinued as soon as they are no longer required.</p> <p>Institutional protocols for maintaining and replacing invasive lines and devices are followed to minimize the risk of infection.</p>

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↑, increased; CVP, central venous pressure; IV, intravenous; MI, myocardial infarction; PAWP, pulmonary artery wedge pressure.

Adapted from Urden, L. D., Stacy, K. M., & Lough, M. E. (2019). *Priorities in critical care nursing* (8th ed.). St. Louis, MO: Elsevier.

The health assessment focuses on obtaining baseline physiologic, psychological, and social information. Cognitive status is carefully assessed, as patients with impaired cognitive status will need more assistance after surgery and may require subacute care prior to returning home. Older adults are at a high risk for suffering adverse cognitive outcomes following cardiac surgery (Jones, Matalanis, Mårtensson, et al., 2019). The patient's and family's education needs are identified and addressed. Of particular importance are the patient's usual functional level, coping mechanisms, and available support systems. These factors affect the patient's postoperative course, discharge plans, and rehabilitation.

The status of the cardiovascular system is determined by reviewing the patient's symptoms, including past and present experiences with chest pain, palpitations, dyspnea, intermittent claudication (leg pain that occurs with walking), and peripheral edema. The patient's history of major illnesses; previous surgeries; medication; and the use of illicit and over-the-counter drugs, herbal supplements, alcohol, and tobacco is also obtained. Particular attention is paid to blood glucose control in patients with diabetes because there is a higher incidence of postoperative complications when glycemic control is poor (Gordon, Lauver, & Buck, 2018).

The psychosocial assessment and the assessment of the patient's and family's learning needs are also important. Anticipation of cardiac surgery is a source of great stress to the patient and family, and patients with high anxiety levels have poorer outcomes (Ramesh, Nayak, Pai, et al., 2017). However, some anxiety is expected, and the work of worrying can help patients identify priorities and find coping strategies that help them face the threat of surgery. Questions may be asked to obtain the following information:

- Knowledge and understanding of the surgical procedure, postoperative course, and recovery
- Fears and concerns regarding the surgery and future health status
- Coping mechanisms helpful to the patient
- Support systems available during and following hospitalization

### Reducing Fear and Anxiety

The nurse gives the patient and family time and opportunity to express their fears. Topics of concern may be pain, changes in body image, fear of the unknown, and fear of disability or death. It may be helpful to describe the sensations that the patient can expect, including the preoperative sedation, surgical anesthesia, and postoperative pain management. The nurse reassures the patient that the fear of pain is normal, that some pain will be experienced, that medication to relieve pain will be provided, and that the patient will be closely monitored. In addition, the nurse instructs the patient to request analgesic medication before the pain becomes severe. If the patient has concerns about scarring from surgery, the nurse encourages them to discuss this issue and corrects any misconceptions. The patient and family may want to discuss their

fear of the patient dying. After the fear is expressed, the nurse can assure the patient and family that this fear is normal and further explore their feelings. For patients with extreme anxiety or fear and for whom emotional support and education are not successful, antianxiety medication such as lorazepam may be helpful.

### Monitoring and Managing Potential Complications

Angina may occur because of increased stress and anxiety related to the forthcoming surgery. The patient who develops angina usually responds to typical therapy for angina, most commonly nitroglycerin. Some patients require oxygen and IV nitroglycerin infusions. Physiologically unstable patients may require preoperative management in a critical care unit.



### Providing Patient Education

Prior to surgery, patients and their families are given specific instructions. This includes information on how the patient should take or stop specific medications, including anticoagulant agents, antihypertensive medications, and medications that control diabetes. The patient is instructed to shower with an antiseptic solution such as chlorhexidine gluconate and to apply mupirocin calcium 2% ointment to each nostril to help reduce the risk of surgical site infections (Reiser, Scherag, Forstner, et al., 2017). Cardiac surgical infections are often caused by *Staphylococcus aureus* which is found in the nasal passages. Studies have shown that decolonizing the nasal passage preoperatively is effective in reducing sternal wound infections associated with cardiac surgery (Lemaignen, Armand-Lefevre, Birgand, et al., 2018) (see [Chapter 14](#) for further discussion of preoperative preparation).

Education also includes information about the hospitalization and surgery. The nurse informs the patient and family about the equipment, tubes, and lines that will be present after surgery and their purposes. They should expect monitors, several IV lines, chest tubes, and a urinary catheter. Explaining the purpose and the approximate time that these devices will be in place helps reassure the patient. Most patients remain intubated and on mechanical ventilation for several hours after surgery. It is important for patients to know that this will prevent them from talking, and the nurse should reassure them that the staff will be able to assist them with other means of communication.

The nurse takes care to answer the patient's questions about postoperative care and procedures. After the nurse explains deep breathing and coughing, the use of the incentive spirometer, and foot exercises, the nurse practices these procedures with the patient. The benefit of early and frequent ambulation is discussed. The family's questions at this time usually focus on the length of the surgery, who will discuss the results of the procedure with them after surgery, where to wait during the surgery, the visiting procedures for the critical care unit, and how they can support the patient before surgery and in the critical care unit.

### Intraoperative Management

The perioperative nurse performs assessments and prepares the patient as described in Chapters 14 and 15. In addition to assisting with the surgical procedure, perioperative

nurses are responsible for the comfort and safety of the patient.

Possible intraoperative complications include low cardiac output, arrhythmias, hemorrhage, MI, organ failure from shock, and thromboembolic events including stroke (Urdan et al., 2019). Astute intraoperative nursing assessment is critical to prevent, detect, and initiate prompt intervention for these complications. Before the chest incision is closed, chest tubes are inserted to evacuate air and drainage from the mediastinum and the thorax.

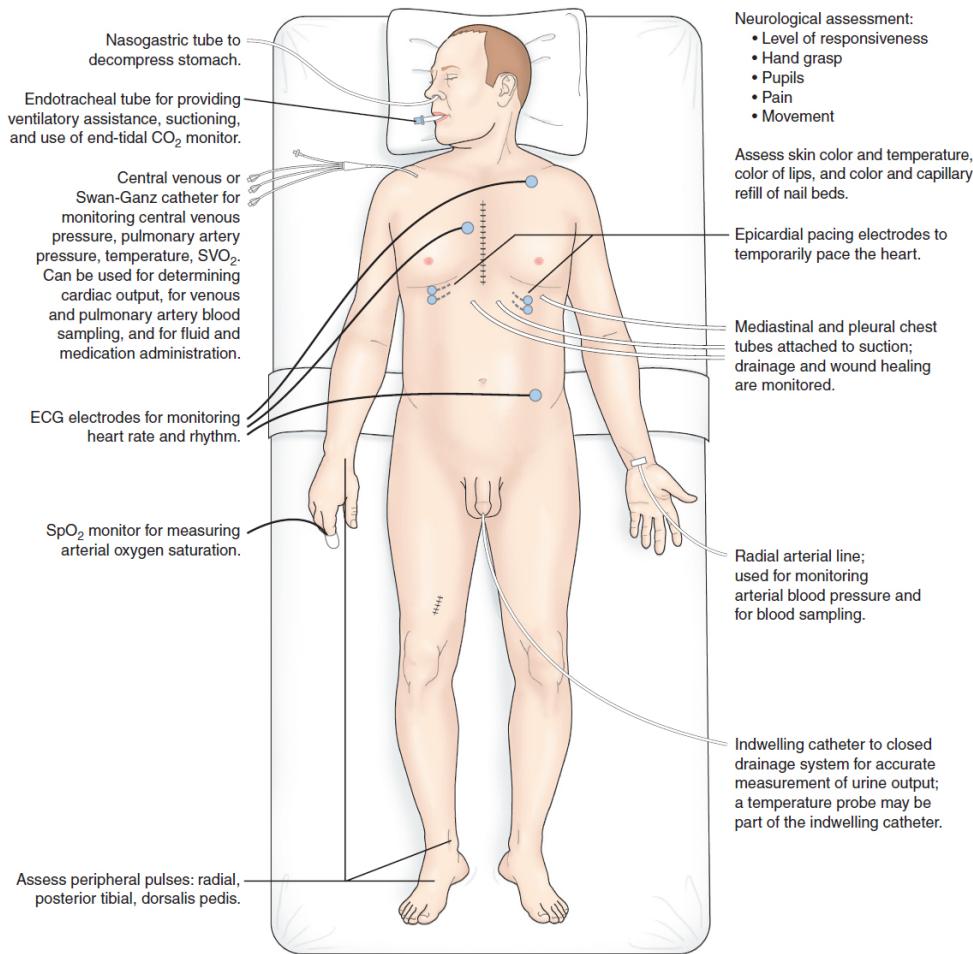


For the procedural guideline for setup and management of chest drainage systems, go to [thepoint.lww.com/Brunner15e](http://thepoint.lww.com/Brunner15e).

Temporary epicardial pacemaker electrodes may be implanted on the surface of the right atrium and the right ventricle. These epicardial electrodes can be connected to an external pacemaker if the patient has persistent bradycardia perioperatively (see [Chapter 22](#) for a discussion of pacemakers).

## Postoperative Nursing Management

Initial postoperative care focuses on achieving or maintaining hemodynamic stability and recovery from general anesthesia. Care may be provided in the postanesthesia care unit (PACU) or ICU. The immediate postoperative period for the patient who has undergone cardiac surgery presents many challenges to the health care team. All efforts are made to facilitate the transition from the operating room to the ICU or PACU with minimal risk. Specific information about the surgical procedure and important factors about postoperative management are communicated by the surgical team and anesthesia personnel to the critical care or PACU nurse, who then assumes responsibility for the patient's care. [Figure 23-12](#) presents an overview of the many aspects of postoperative care of the cardiac surgical patient.



**Figure 23-12 •** Postoperative care of the patient who has undergone cardiac surgery requires the nurse to be proficient in interpreting hemodynamics, correlating physical assessment data with laboratory results, sequencing interventions, and evaluating progress toward desired outcomes.

After the patient's cardiac status and respiratory status are stable, the patient is transferred to a surgical progressive care unit with telemetry. Care in both the ICU and progressive care unit focuses on monitoring of cardiopulmonary status, pain management, wound care, progressive activity, and nutrition. Education about medications and risk factor modification is emphasized.

A typical plan of postoperative nursing care is presented in [Chart 23-11](#).

### Assessing the Patient

When the patient is admitted to the critical care unit or PACU, nursing and medical personnel perform a complete assessment of all systems at least every 4 hours. It is necessary to assess the following parameters:

*Neurologic status:* level of responsiveness, pupil size and reaction to light, facial symmetry, movement of the extremities, and hand grip strength

*Cardiac status:* heart rate and rhythm, heart sounds, pacemaker status, arterial blood pressure, central venous pressure (CVP); in select patients, hemodynamic

parameters: pulmonary artery pressure, pulmonary artery wedge pressure (PAWP), cardiac output and index, systemic and pulmonary vascular resistance, mixed venous oxygen saturation ( $Sv-O_2$ ). A pulmonary artery catheter is often used to monitor these parameters. Alternatively, minimally invasive monitoring of stroke volume, systemic vascular resistance, and cardiac output are calculated through pressures obtained in the arterial line (e.g., Vigileo monitor with FloTrac sensor). (See [Chapter 21](#) for a detailed description of hemodynamic monitoring.)

Chart 23-11



## PLAN OF NURSING CARE

Care of the Patient After Cardiac Surgery

**NURSING DIAGNOSIS:** Impaired cardiac output associated with blood loss and compromised myocardial function

**GOAL:** Restoration of cardiac output to maintain organ and tissue perfusion

<b>Nursing Interventions</b>	<b>Rationale</b>	<b>Expected Outcomes</b>
<p>1. Monitor cardiovascular status. Serial readings of blood pressure, other hemodynamic parameters, and cardiac rhythm and rate are obtained, recorded, and correlated with the patient's overall condition.</p> <p>a. Assess arterial blood pressure every 15 minutes until stable; then arterial or cuff blood pressure every 1–4 hours × 24 hours; then every 8–12 hours until hospital discharge.</p> <p>b. Auscultate for heart sounds and rhythm.</p> <p>c. Assess peripheral pulses (pedal, tibial, radial).</p> <p>d. Monitor hemodynamic parameters to assess cardiac output, volume status, and vascular tone.</p>	<p>1. Effectiveness of cardiac output is evaluated by continuous monitoring.</p> <p>a. Blood pressure is one of the most important physiologic parameters to monitor; vasoconstriction after cardiopulmonary bypass may require treatment with an IV vasodilator.</p> <p>b. Auscultation provides evidence of pericarditis (precordial rub), arrhythmias.</p> <p>c. Presence or absence and quality of pulses provide data about cardiac output as well as obstructive lesions.</p> <p>d. Rising CVP and PAWP may indicate congestive heart failure or pulmonary edema. Low pressures may indicate need</p>	<ul style="list-style-type: none"> <li>The following parameters are within the patient's normal ranges:             <ul style="list-style-type: none"> <li>Arterial pressure</li> <li>Central venous pressure (CVP)</li> <li>Pulmonary artery pressures</li> <li>Pulmonary artery wedge pressure (PAWP)</li> <li>Heart sounds</li> <li>Pulmonary and systemic vascular resistance</li> <li>Cardiac output and cardiac index</li> <li>Peripheral pulses</li> <li>Cardiac rate and rhythm</li> <li>Cardiac biomarkers</li> <li>Urine output</li> <li>Skin and mucosal color</li> <li>Skin temperature</li> </ul> </li> <li>&lt;200 mL/h of drainage through chest tubes during first 4–6 hours</li> <li>Vital signs stable</li> <li>CVP and other hemodynamic parameters within normal limits</li> </ul>

- |   |   |   |
|---|---|---|
| e. Watch for trends in hemodynamics, and note that mechanical ventilation may alter hemodynamics.                         | for volume replacement.   | • Urinary output within normal limits         |
| f. Monitor electrocardiogram (ECG) pattern for cardiac arrhythmias and ischemic changes.                                  | e. Trends are more important than isolated readings. Mechanical ventilation increases intrathoracic pressure.   | • Skin color normal                           |
| g. Assess cardiac biomarker results.  | f. Arrhythmias may occur with coronary ischemia, hypoxia, bleeding, and acid-base or electrolyte disturbances. ST-segment changes may indicate myocardial ischemia. | • Respirations unlabored, clear breath sounds |
| h. Measure urine output every $\frac{1}{2}$ to 1 hour at first, then with vital signs.                                    | Pacemaker capture and antiarrhythmic medications are used to maintain heart rate and rhythm and to support blood pressure.  | • Pain limited to incision                    |
| i. Observe buccal mucosa, nail beds, lips, earlobes, and extremities.   | g. Elevations may indicate myocardial infarction.   |   |
| j. Assess skin; note temperature and color.   | h. Urine output $<0.5$ mL/kg/h indicates decreased renal perfusion and may reflect decreased cardiac output.  |   |
| 2. Observe for persistent bleeding: excessive chest tube drainage of blood; hypotension; low CVP; tachycardia. Prepare to | i. Duskeness and cyanosis may indicate decreased cardiac output.  |   |
|   | j. Cool moist skin indicates vasoconstriction and decreased cardiac output.   |   |

- administer blood products, IV fluids.
3. Observe for cardiac tamponade: hypotension; rising CVP and PAWP, pulsus paradoxus; jugular vein distention; decreasing urinary output. Check for diminished amount of blood in chest drainage collection system. Prepare for reoperation.
  4. Observe for signs of cardiac failure. Prepare to administer diuretics, IV inotropic agents.
2. Bleeding can result from surgical trauma to tissues, anticoagulant medications, and clotting defects.
3. Cardiac tamponade results from bleeding into the pericardial sac or accumulation of fluid in the sac, which compresses the heart and prevents adequate filling of the ventricles. Decrease in chest drainage may indicate that fluid and clots are accumulating in the pericardial sac.
4. Cardiac failure results from decreased pumping action of the heart; can cause deficient perfusion to vital organs.

#### NURSING DIAGNOSIS: Impaired gas exchange associated with chest surgery

**GOAL:** Adequate gas exchange

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Maintain mechanical ventilation until the patient is able to breathe independently.</li> <li>2. Monitor ABGs, tidal volume, peak inspiratory pressure, and extubation parameters.</li> </ol>	<ol style="list-style-type: none"> <li>1. Ventilatory support is used to decrease work of the heart, to maintain effective ventilation, and to provide an airway in the event of complications.</li> <li>2. ABGs and ventilator parameters indicate effectiveness of ventilator and changes that need to be made to improve gas exchange.</li> </ol>	<ul style="list-style-type: none"> <li>• Airway patent</li> <li>• Arterial blood gases (ABGs) within normal range</li> <li>• Endotracheal tube correctly placed, as evidenced by x-ray</li> <li>• Breath sounds clear bilaterally</li> <li>• Ventilator synchronous with respirations</li> <li>• Breath sounds clear after</li> </ul>

- |   |   |   |
|---|---|---|
| 3. Auscultate chest for breath sounds.  | 3. Crackles indicate pulmonary congestion; decreased or absent breath sounds may indicate pneumothorax, hemothorax, dislodgement of tube. | suctioning/coughing   |
| 4. Sedate patient adequately, as prescribed, and monitor respiratory rate and depth.  | 4. Sedation helps the patient to tolerate the endotracheal tube and to cope with mechanical ventilation.                                  | • Nail beds and mucous membranes pink                                       |
| 5. Suction tracheobronchial secretions as needed, using strict aseptic technique.   | 5. Retention of secretions leads to hypoxia and possible infection.   | • Mental acuity consistent with amount of sedatives and analgesics received |
| 6. Assist in weaning and endotracheal tube removal.   | 6. Extubation decreases risk of pulmonary infections and enhances ability of patient to communicate.                                      | • Oriented to person; able to respond "yes" and "no" appropriately          |
| 7. After extubation, promote deep breathing, coughing, and turning. Encourage the use of the incentive spirometer and compliance with breathing treatments. Instruct about incisional splinting with a "cough pillow" to decrease discomfort. | 7. Aids in keeping airway patent, preventing atelectasis, and facilitating lung expansion   | • Able to be weaned successfully from ventilator                            |

**NURSING DIAGNOSIS:** Risk for hypovolaemia or hypervolaemia and electrolyte imbalance associated with alterations in blood volume

**GOAL:** Fluid and electrolyte balance

Nursing Interventions	Rationale	Expected Outcomes
1. Monitor fluid and electrolyte balance. a. Accurately document intake	1. Adequate circulating blood volume is necessary for optimal cellular activity; fluid and electrolyte	• Fluid intake and output balanced • Hemodynamic assessment

	<p>and output; record urine volume every half hour to 4 hours while in critical care unit; then every 8–12 hours while hospitalized.</p> <p>b. Assess blood pressure, hemodynamic parameters, weight, electrolytes, hematocrit, jugular venous pressure, breath sounds, urinary output, and nasogastric tube drainage.</p> <p>c. Measure postoperative chest drainage; cessation of drainage may indicate kinked or blocked chest tube. Ensure patency and integrity of the drainage system.</p> <p>d. Weigh daily and correlate with intake and output.</p>	<p>imbalance can occur after surgery.</p> <p>a. Provides a method to determine positive or negative fluid balance and fluid requirements</p> <p>b. Provides information about state of hydration</p> <p>c. Excessive blood loss from chest cavity can cause hypovolemia.</p> <p>d. Indicator of fluid balance</p>	<p>parameters negative for fluid overload or hypovolemia</p> <ul style="list-style-type: none"> <li>• Normal blood pressure with position changes</li> <li>• Absence of arrhythmia</li> <li>• Stable weight</li> <li>• Arterial blood pH 7.35–7.45</li> <li>• Serum potassium 3.5–5.0 mEq/L (3.5–5.0 mmol/L)</li> <li>• Serum magnesium 1.8–2.6 mg/dL (0.74–1.07 mmol/L)</li> <li>• Serum sodium 135–145 mEq/L (135–145 mmol/L)</li> <li>• Serum calcium 8.8–10.4 mg/dL (2.2–2.6 mmol/L)</li> <li>• Serum glucose ≤180 mg/dL</li> </ul>
2.	<p>Be alert to changes in serum electrolyte levels.</p> <p>a. Hypokalemia (low potassium) <i>Effects:</i> Arrhythmias: premature ventricular contractions, ventricular tachycardia. Observe for</p>	<p>A specific concentration of electrolytes is necessary in both extracellular and intracellular body fluids to sustain life.</p> <p>a. Causes: Inadequate intake, diuretics, vomiting, excessive nasogastric drainage,</p>	

	specific ECG changes. Administer IV potassium replacement as prescribed.	perioperative stress response
b.	Hyperkalemia (high potassium) <i>Effects:</i> ECG changes, tall peaked T waves, wide QRS, bradycardia. Be prepared to administer diuretic or an ion-exchange resin (sodium polystyrene sulfonate); or IV insulin and glucose.	b. <i>Causes:</i> Increased intake, hemolysis from cardiopulmonary bypass/mechanical assist devices, acidosis, renal insufficiency. The resin binds potassium and promotes intestinal excretion of it. Insulin assists the cells with glucose and potassium absorption.
c.	Monitor serum magnesium, sodium, and calcium.	c. Low levels of magnesium are associated with arrhythmias. Low levels of sodium are associated with weakness and neurologic symptoms. Low levels of calcium can lead to arrhythmias and muscle spasm.
d.	Hyperglycemia (high blood glucose) <i>Effects:</i> Increased urine output, thirst, impaired healing. Administer insulin as prescribed.	d. <i>Cause:</i> Stress response to surgery. Affects both patients with diabetes and those without diabetes.

**NURSING DIAGNOSIS:** Risk for acute confusion associated with alteration in sleep-wake cycle, impaired metabolic functioning, use of multiple medications  
**GOAL:** Prevention of acute confusion/postcardiotomy delirium

<b>Nursing Interventions</b>	<b>Rationale</b>	<b>Expected Outcomes</b>
<ol style="list-style-type: none"> <li>1. Use measures to prevent postcardiotomy delirium:             <ol style="list-style-type: none"> <li>a. Explain all procedures and the need for patient cooperation.</li> <li>b. Plan nursing care to provide for periods of uninterrupted sleep with patient's normal day-night pattern.</li> <li>c. Promote continuity of care.</li> <li>d. Orient to time and place frequently. Encourage family to visit.</li> <li>e. Assess for medications that may contribute to delirium.</li> </ol> </li>   <li>2. Observe for perceptual distortions, hallucinations, disorientation, and paranoid delusions.</li> </ol>	<ol style="list-style-type: none"> <li>1. Postcardiotomy delirium may result from alterations in sleep-wake cycle, impaired metabolic functioning, and use of multiple medications. Normally, sleep cycles are at least 50 minutes long. The first cycle may be as long as 90–120 minutes and then shorten during successive cycles. Sleep deprivation results when the sleep cycles are interrupted or are inadequate in number.</li> <li>2. Delirium can indicate a serious medical condition such as hypoxia, acid-base imbalance, metabolic abnormalities, and cerebral infarction.</li> </ol>	<ul style="list-style-type: none"> <li>• Cooperates with procedures</li> <li>• Sleeps for long, uninterrupted intervals</li> <li>• Oriented to person, place, time</li> <li>• Experiences no perceptual distortions, hallucinations, disorientation, delusions</li> </ul>

**NURSING DIAGNOSIS:** Acute pain associated with surgical trauma and pleural irritation caused by chest tubes  
**GOAL:** Relief of pain

<b>Nursing Interventions</b>	<b>Rationale</b>	<b>Expected Outcomes</b>
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|--|--|---|
| <ol style="list-style-type: none"> <li>Record nature, type, location, intensity, and duration of pain.</li> <li>Encourage routine pain medication dosing for the first 24–72 hours, and observe for side effects of lethargy, hypotension, tachycardia, respiratory depression.</li> </ol> | <ol style="list-style-type: none"> <li>Pain and anxiety increase pulse rate, oxygen consumption, and cardiac workload.</li> <li>Analgesia promotes rest, decreases oxygen consumption caused by pain, and aids patient in performing deep-breathing and coughing exercises; pain medication is more effective when taken before pain is severe.</li> </ol> | <ul style="list-style-type: none"> <li>States pain is decreasing in severity</li> <li>Restlessness decreased</li> <li>Vital signs stable</li> <li>Participates in deep-breathing and coughing exercises</li> <li>Verbalizes fewer complaints of pain each day</li> <li>Positions self; participates in care activities</li> <li>Gradually increases activity</li> </ul> |
|--|--|---|

**NURSING DIAGNOSIS:** Risk for impaired cardiac function associated with alterations in afterload that may compromise renal perfusion

**GOAL:** Maintenance of adequate cardiac output and renal perfusion

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>Assess renal function:           <ol style="list-style-type: none"> <li>Measure urine output every half hour to 4 hours in critical care, then every 8–12 hours until hospital discharge.</li> <li>Monitor and report lab results: BUN, serum creatinine, serum electrolytes.</li> </ol> </li> </ol>	<ol style="list-style-type: none"> <li>Renal injury can be caused by deficient perfusion, hemolysis, low cardiac output, and the use of vasopressor agents to increase blood pressure.           <ol style="list-style-type: none"> <li>&lt;0.5 mL/kg/h indicates decreased renal function.</li> <li>These tests indicate the kidneys' ability to excrete waste products.</li> </ol> </li> <li>These agents promote renal function and increase cardiac output and renal blood flow.</li> <li>Provides patient with the opportunity to ask questions and prepare for the procedure</li> </ol>	<ul style="list-style-type: none"> <li>Urine output consistent with fluid intake; &gt;0.5 mL/kg/h</li> <li>Urine specific gravity 1.005–1.030</li> <li>Blood urea nitrogen (BUN), creatinine, electrolytes within normal limits</li> </ul>

2. Prepare to administer rapid-acting diuretics or inotropic drugs (e.g., dobutamine).
3. Prepare patient for dialysis or continuous renal replacement therapy if indicated.

**NURSING DIAGNOSIS:** Impaired thermoregulation associated with infection or postpericardiotomy syndrome

**GOAL:** Maintenance of normal body temperature

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Assess temperature every hour.</li> <li>2. Use aseptic technique when changing dressings, suctioning endotracheal tube; maintain closed systems for all IV and arterial lines and for indwelling urinary catheter.</li> <li>3. Observe for symptoms of postpericardiotomy syndrome.</li> <li>4. Obtain cultures and other lab work (CBC, ESR); administer antibiotic agents as prescribed.</li> <li>5. Administer anti-inflammatory agents as directed.</li> </ol>	<ol style="list-style-type: none"> <li>1. Fever can indicate infectious or inflammatory process.</li> <li>2. Decreases risk of infection</li> <li>3. Occurs in 10% of patients after cardiac surgery.</li> <li>4. Antibiotic agents treat documented infection.</li> <li>5. Anti-inflammatory agents relieve symptoms of inflammation.</li> </ol>	<ul style="list-style-type: none"> <li>• Normal body temperature</li> <li>• Incisions are free of infection and are healing.</li> <li>• Absence of symptoms of postpericardiotomy syndrome: fever, malaise, pericardial effusion, pericardial friction rub, arthralgia</li> </ul>

**NURSING DIAGNOSIS:** Lack of knowledge about self-care activities

**GOAL:** Ability to perform self-care activities

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Develop education plan for patient and family. Provide specific instructions for the following:</p> <ul style="list-style-type: none"> <li>• Diet and daily weights</li> <li>• Activity progression</li> <li>• Exercise</li> <li>• Deep breathing, coughing, lung expansion exercises</li> <li>• Temperature and pulse monitoring</li> <li>• Medication regimen</li> <li>• Incision care</li> <li>• Access to the emergency medical system</li> </ul> <p>2. Provide verbal and written instructions; provide several education sessions for reinforcement and answering questions.</p> <p>3. Involve family in education sessions.</p> <p>4. Provide contact information for surgeon and</p>	<p>1. Each patient has unique learning needs.</p> <p>2. Repetition promotes learning by allowing for questions and clarification of misinformation.</p> <p>3. Family members responsible for home care are usually anxious and require adequate time for learning.</p> <p>4. Arrangements for contacts with health care personnel help to allay anxieties.</p> <p>5. Learning, recovery, and lifestyle changes continue after discharge from the hospital.</p>	<ul style="list-style-type: none"> <li>• Patient and family members explain and comply with therapeutic regimen.</li> <li>• Patient and family members identify necessary lifestyle changes.</li> <li>• Has copy of discharge instructions (in the patient's primary language and at appropriate reading level; has an alternate format if indicated)</li> <li>• Keeps follow-up appointments</li> </ul>

cardiologist and instructions about follow-up visit with surgeon.

5. Make appropriate referrals: home care agency, cardiac rehabilitation program, community support groups.

*Respiratory status:* chest movement, breath sounds, ventilator settings (e.g., rate, tidal volume, oxygen concentration, mode such as assist-control, positive end-expiratory pressure, pressure support), respiratory rate, peak inspiratory pressure, percutaneous oxygen saturation ( $\text{SpO}_2$ ), end-tidal carbon dioxide ( $\text{CO}_2$ ), pleural chest tube drainage, arterial blood gases. (See Chapters 17 and 19 for detailed descriptions of respiratory assessment and ventilatory management, respectively.)

*Peripheral vascular status:* peripheral pulses; color of skin, nail beds, mucosa, lips, and earlobes; skin temperature; edema; condition of dressings and invasive lines

*Renal function:* urinary output; serum creatinine and electrolytes

*Fluid and electrolyte status:* strict intake and output, including all IV fluids and blood products, output from all drainage tubes; clinical and laboratory indicators of imbalance

*Pain:* nature, type, location, and duration; apprehension; response to analgesics

Assessment also includes checking all equipment and tubes to ensure that they are functioning properly: endotracheal tube, ventilator, end-tidal  $\text{CO}_2$  monitor,  $\text{SpO}_2$  monitor, pulmonary artery catheter,  $\text{Sv-O}_2$  monitor, arterial and IV lines, IV infusion devices and tubing, cardiac monitor, pacemaker, chest tubes, and urinary drainage system.

As the patient regains consciousness and progresses through the postoperative period, the nurse also assesses indicators of psychological and emotional status. The patient may exhibit behavior that reflects denial or depression or may experience postoperative delirium. Characteristic signs of delirium include transient perceptual illusions, visual and auditory hallucinations, disorientation, and paranoid delusions. Patients who have delirium after cardiac surgery have poorer outcomes than do similar patients without this complication (Jones et al., 2019).

The family's needs also must be assessed. The nurse ascertains how family members are coping with the situation; determines their psychological, emotional, and spiritual needs; and finds out whether they are receiving adequate information about the patient's condition.

### Monitoring for Complications

The patient is continuously assessed for impending complications (see [Table 23-4](#)). The nurse and the surgical team function collaboratively to prevent complications, to

identify early signs and symptoms of complications, and to institute measures to reverse their progression.

### **Decreased Cardiac Output**

A decrease in cardiac output is always a threat to the patient who has had cardiac surgery, and it can have a variety of causes. Preload alterations occur when too little blood volume returns to the heart as a result of persistent bleeding and hypovolemia. Excessive postoperative bleeding can lead to decreased intravascular volume, hypotension, and low cardiac output. Bleeding problems are common after cardiac surgery because of the effects of CPB, trauma from the surgery, and anticoagulation. Preload can also decrease if there is a collection of fluid and blood in the pericardium (cardiac tamponade), which impedes cardiac filling. Cardiac output is also altered if too much volume returns to the heart, causing fluid overload.

Afterload alterations occur when the arteries are constricted as a result of postoperative hypertension or hypothermia, increasing the workload of the heart. Heart rate alterations from bradycardia, tachycardia, and arrhythmias can lead to decreased cardiac output, and contractility can be altered in cardiac failure, MI, electrolyte imbalances, and hypoxia.

### **Fluid Volume and Electrolyte Imbalance**

Fluid and electrolyte imbalance may occur after cardiac surgery. Nursing assessment for these complications includes monitoring of intake and output, weight, hemodynamic parameters, hematocrit levels, neck vein distention, edema, breath sounds (e.g., fine crackles, wheezing), and electrolyte levels. The nurse reports changes in serum electrolytes promptly so that treatment can be instituted. Especially important are dangerously high or dangerously low levels of potassium, magnesium, sodium, and calcium. Elevated blood glucose levels are common in the postoperative period. Administration of IV insulin is recommended in patients both with and without diabetes to achieve the glycemic control necessary to promote wound healing, decrease infection, and improve survival after surgery (Gordon et al., 2018). Implementing an insulin infusion protocol that targets moderate glycemic control has been demonstrated as effective in treating acute hyperglycemia following cardiac surgery while also decreasing the incidence of hypoglycemia (Gordon et al., 2018).

### **Impaired Gas Exchange**

Impaired gas exchange is another possible complication after cardiac surgery. All body tissues require an adequate supply of oxygen for survival. To achieve this after surgery, an endotracheal tube with ventilator assistance may be used for hours to days. The assisted ventilation is continued until the patient's blood gas values are acceptable and the patient demonstrates the ability to breathe independently. Patients who are stable after surgery may be extubated as early as 2 to 4 hours after surgery, which reduces their discomfort and anxiety and facilitates patient–nurse communication.

While receiving mechanical ventilation, the patient is continuously assessed for signs of impaired gas exchange: restlessness, anxiety, cyanosis of mucous membranes and peripheral tissues, tachycardia, and fighting the ventilator. Breath sounds are assessed often to detect pulmonary congestion and monitor lung expansion. Arterial blood gases, SpO<sub>2</sub>, and end-tidal CO<sub>2</sub> are assessed for decreased oxygen and

increased CO<sub>2</sub>. Following extubation, aggressive pulmonary interventions such as turning, coughing, deep breathing, and early ambulation are necessary to prevent atelectasis and pneumonia.

### **Impaired Cerebral Circulation**

Hypoperfusion or microemboli during or following cardiac surgery may produce injury to the brain. Brain function depends on a continuous supply of oxygenated blood. The brain does not have the capacity to store oxygen and must rely on adequate continuous perfusion by the heart. The nurse observes the patient for signs and symptoms of cerebral hypoxia: restlessness, confusion, dyspnea, hypotension, and cyanosis. An assessment of the patient's neurologic status includes the level of consciousness, response to verbal commands and painful stimuli, pupil size and reaction to light, facial symmetry, movement of the extremities, and hand grip strength. The nurse documents any indication of a change in status and reports abnormal findings to the surgeon because they may signal the onset of a complication such as a stroke.

### **Maintaining Cardiac Output**

Ongoing evaluation of the patient's cardiac status continues as the nurse monitors the effectiveness of cardiac output through clinical observations and routine measurements: serial readings of blood pressure, heart rate, CVP, arterial pressure, and pulmonary artery pressures.

Renal function is related to cardiac function, as blood pressure and cardiac output drive glomerular filtration; therefore, urinary output is measured and recorded. Urine output less than 0.5 mL/kg/h may indicate a decrease in cardiac output or inadequate fluid volume.

Body tissues depend on adequate cardiac output to provide a continuous supply of oxygenated blood to meet the changing demands of the organs and body systems. Because the buccal mucosa, nail beds, lips, and earlobes are sites with rich capillary beds, they are observed for cyanosis or duskeness as possible signs of reduced cardiac output. Distention of the neck veins when the head of the bed is elevated to 30 degrees or more may signal right-sided heart failure.

Arrhythmias may develop due to decreased perfusion to or irritation of the myocardium from surgery. The most common arrhythmias encountered during the postoperative period are atrial fibrillation, bradycardias, tachycardias, and ectopic beats (Urdan et al., 2019). Continuous observation of the cardiac monitor for arrhythmias is essential.

The nurse reports any indications of decreased cardiac output promptly. The assessment data are used to determine the cause of the problem. After a diagnosis has been made, the primary provider and the nurse work collaboratively to restore cardiac output and prevent further complications. When indicated, blood components; fluids; and antiarrhythmics, diuretics, vasodilators, or vasoconstrictors are prescribed. If additional interventions are necessary, such as the placement of an intra-aortic balloon pump, the patient and family are prepared for the procedure.

### **Promoting Adequate Gas Exchange**

To ensure adequate gas exchange, the patency of the endotracheal tube is assessed and maintained. The tube must be secured to prevent it from slipping out or down into the right mainstem bronchus. Suctioning is necessary when crackles or coughing is present.



*For the procedural guideline for performing tracheal suction, go to [thePoint.lww.com/Brunner15e](http://thePoint.lww.com/Brunner15e).*

Arterial blood gas determinations are compared with baseline data, and changes are reported to the primary provider promptly.

When the patient's hemodynamic parameters stabilize, body position is changed every 1 to 2 hours. Frequent changes of patient position provide for optimal pulmonary ventilation and perfusion, allowing the lungs to expand more fully.

Physical assessment and arterial blood gas results guide the process of weaning the patient from the ventilator. The nurse assists with the weaning process and eventually with the removal of the endotracheal tube. After extubation, the nurse encourages deep breathing and coughing at least every 1 to 2 hours to clear secretions, open the alveolar sacs, and promote effective ventilation. See [Chapter 19](#) for discussion of weaning the patient from the ventilator.

### Maintaining Fluid and Electrolyte Balance

To promote fluid and electrolyte balance, the nurse carefully assesses the intake and output to determine positive or negative fluid balance. It is necessary to record all fluid intake, including IV, nasogastric tube, and oral fluids, as well as all output, including urine, nasogastric drainage, and chest drainage.

Hemodynamic parameters (e.g., blood pressure, CVP, cardiac output) are correlated with intake, output, and weight to determine the adequacy of hydration and cardiac output. Serum electrolytes are monitored, and the patient is observed for signs of potassium, magnesium, sodium, or calcium imbalance (see [Chapter 10](#)).

Indications of dehydration, fluid overload, or electrolyte imbalance are reported promptly, and the primary provider and nurse work collaboratively to restore fluid and electrolyte balance and monitor the patient's response to therapies.

### Minimizing Confusion

Some patients exhibit abnormal behaviors and acute confusion that occur with varying intensity and duration. The risk of delirium is high in patients who have undergone cardiac surgery and increases with patients' age (Jones et al., 2019; Smulter, Lingehall, Gustafson, et al., 2019; see the Nursing Research Profile in [Chart 23-12](#)). Clinical manifestations of postoperative delirium include restlessness, agitation, visual and auditory hallucinations, and paranoia. The delirium typically appears after a 2- to 5-day stay in an ICU. Patients are assessed for this problem with tools such as the Confusion Assessment Method for the ICU (CAM-ICU) (Price,

Garvan, Hizel, et al., 2017) (see [Chapter 8, Chart 8-7](#), for discussion of CAM). The CAMU-ICU scale assesses for key indicators of delirium such as disorganized thinking and inattention. When this testing is positive, further assessment of the patient's physiologic and psychological status is required. Presumed causes of postoperative delirium include anxiety, sleep deprivation, increased sensory input, medications, and physiologic problems such as hypoxemia and metabolic imbalance (Blair, Mehmood, Rudnick, et al., 2019). Treatment includes correction of identified physiologic problems such as metabolic and electrolyte imbalances. In addition, behavioral interventions are used (e.g., frequent reorientation). Sedative medications such as haloperidol were once thought to reduce agitation and improve survival, but recent studies note that use of haloperidol causes oversedation and does not reliably treat or prevent delirium (Blair et al., 2019). The delirium often resolves after the patient is transferred from the unit, but nonetheless can be associated with negative outcomes including cognitive and functional decline, longer lengths of hospital stay, and higher mortality (Delaney, Hammond, & Litton, 2018).

### Chart 23-12 NURSING RESEARCH PROFILE

## Use of a Postoperative Delirium Screening Scale in Older Adults After Cardiac Surgery

Smulter, N., Lingeblad, H. C., Gustafson, Y., et al. (2019). The use of a screening scale improves the recognition of delirium in older patients after cardiac surgery: A retrospective observational study. *Journal of Clinical Nursing*, 28(11-12), 2309–2318.

### Purpose

Postoperative delirium (POD) is a frequent occurrence in older patients undergoing cardiac surgery. However, it is often not recognized by health care providers and therefore may go undiagnosed. The purpose of this study was to assess whether the use of a delirium screening tool by nurses postoperatively will improve the recognition and diagnosis of POD.

### Design

This study was a retrospective observational analysis. Seventy eight patients aged 70 and older who had cardiac surgery were diagnosed with POD. Nurses used the Nursing Delirium Screening Scale (Nu-DESC) to screen for delirium symptoms. This scale uses five items to assess for delirium: disorientation, inappropriate behavior, inappropriate communication, illusions and hallucinations, and psychomotor retardation. Each item is graded from 0 to 2 with a maximum score of 10. A Nu-DESC score of 2 or greater is thought to indicate the presence of delirium. The screening was conducted three times daily, beginning post-op day 1 through discharge.

Data describing the incidence and nature of POD from the clinical database and discharge summaries were retrospectively collected. This information was compared to the results of symptom screening using the Nu-DESC.

### Findings

POD was correctly identified in 41 of 78 (52.6%) patients. “Inappropriate behavior” was the most common descriptor used by nurses and physicians within discharge summaries. Terminology like “confused,” “aggressive/restless,” and “disoriented” were commonly used to describe delirium symptoms. The cause and specific treatment of delirium was not addressed within the discharge summaries.

Screening using the Nu-DESC identified 56 of 78 (72%) patients with POD. Use of the Nu-DESC showed greater sensitivity in identifying symptoms of delirium than the information documented within the discharge summaries and database.

### Nursing Implications

Delirium is a serious complication that is underdiagnosed in patients after cardiac surgery and, when present, not well documented. Use of a validated screening scale, such as the Nu-DESC, can improve the ability of nurses to recognize delirium in postoperative patients.

For all postoperative patients, basic comfort measures are used in conjunction with prescribed analgesics and sedatives to promote rest. Invasive lines and tubes are discontinued as soon as possible. Patient care is coordinated to provide undisturbed periods of rest. As the patient’s condition stabilizes and the patient is disturbed less frequently for monitoring and therapeutic procedures, rest periods can be extended.

Uninterrupted sleep is provided as much as possible, especially during the patient's normal hours of sleep.

Careful explanations of all procedures and of the patient's role in facilitating them help keep the patient positively involved throughout the postoperative course. Continuity of care is desirable; a familiar face and a nursing staff with a consistent approach help the patient feel safe. The patient's family should be welcomed at the bedside. A well-designed and individualized plan of nursing care can assist the nursing team in coordinating its efforts for the emotional well-being of the patient.

### Relieving Pain

Patients who have had cardiac surgery may have pain in the peri-incisional area or throughout the chest, shoulders, and back. Pain results from trauma to the chest wall and irritation of the pleura by the chest tubes as well as incisional pain from peripheral vein or artery graft harvest sites.

The nurse assesses patients for verbal and nonverbal indicators of pain and records the nature, type, location, and duration of the pain. To reduce the amount of pain, the nurse encourages the patient to accept medication on a regular basis. The addition of adjunctive pain relievers (anti-inflammatory agents, muscle relaxants) to opioids decreases the amount of opioids required for pain relief and increases patient comfort. Patients report the most pain during coughing, turning, and moving. Physical support of the incision with a folded bath blanket or small pillow during deep breathing and coughing helps minimize pain. The patient should then be able to participate in respiratory exercises and to progressively increase self-care. Patient comfort improves after removal of the chest tubes.

Pain produces distress, which may stimulate the central nervous system to release catecholamines, resulting in constriction of the arterioles and increased heart rate. This can cause increased afterload and decreased cardiac output. Opioids alleviate pain and induce sleep and feelings of well-being, which reduce the metabolic rate and oxygen demands. After the administration of opioids, it is necessary to document observations indicating relief of apprehension and pain in the patient's record. The nurse observes the patient for any adverse effects of opioids, including respiratory depression, hypotension, constipation, ileus, or urinary retention. If respiratory depression occurs, an opioid antagonist (e.g., naloxone) may be required (see [Chapter 9](#) for further discussion of nonpharmacologic pain interventions).

### Maintaining Adequate Tissue Perfusion

The nurse routinely palpates peripheral pulses (e.g., pedal, tibial, femoral, radial, brachial) to assess for arterial obstruction. If a pulse is absent in any extremity, the cause may be prior catheterization of that extremity, chronic peripheral vascular disease, or a thromboembolic obstruction. The nurse immediately reports newly identified absence of any pulse.

Thromboembolic events can result from vascular injury, dislodgment of a clot from a damaged valve, loosening of mural thrombi, or coagulation problems. Air embolism can result from CPB or central venous cannulation. Symptoms of embolization vary according to site. The usual embolic sites are the lungs, coronary arteries, mesentery, spleen, extremities, kidneys, and brain. The patient is observed for the onset of the following:

- Acute onset of chest pain and respiratory distress, as occur in pulmonary embolus or MI
- Abdominal or back pain, as occur in mesenteric emboli
- Pain, cessation of pulses, blanching, numbness, or coldness in an extremity
- One-sided weakness and pupillary changes, as occur in stroke

The nurse promptly reports any of these symptoms.

Venous stasis, which can cause venous thromboembolism (e.g., deep vein thrombosis, pulmonary embolism), may occur after surgery. It can be prevented by using the following measures:

- Apply sequential pneumatic compression devices as prescribed.
- Discourage crossing of legs.
- Avoid elevating the knees on the bed.
- Omit pillows in the popliteal space.
- Begin passive exercises followed by active exercises to promote circulation and prevent venous stasis.

Inadequate renal perfusion can occur as a complication of cardiac surgery. One possible cause is low cardiac output. Trauma to blood cells during CPB can cause hemolysis of red blood cells, which then occlude the renal glomeruli. The use of vasopressor agents to increase blood pressure may constrict the renal arterioles and reduce blood flow to the kidneys.

Nursing management includes accurate measurement of urine output. An output less than 0.5 mL/kg/h may indicate hypovolemia or renal insufficiency. The primary provider may prescribe fluids to increase cardiac output and renal blood flow, or IV diuretics may be given to increase urine output. The nurse should be aware of the patient's blood urea nitrogen, serum creatinine, glomerular filtration rate, and serum electrolyte levels. The nurse should report abnormal levels promptly, because it may be necessary to adjust fluids and the dose or type of medication given. If efforts to maintain renal perfusion are ineffective, the patient may require continuous renal replacement therapy or dialysis (see [Chapter 48](#)).

### Maintaining Normal Body Temperature

Patients are usually hypothermic when admitted to the critical care unit following the cardiac surgical procedure. Because induced hypothermia from CPB and anesthesia lower the patient's core temperature, the patient must be gradually warmed to a normal temperature. This is accomplished partially by the patient's own basal metabolic processes and often with the assistance of heated air blanket systems. While the patient is hypothermic, shivering and hypertension are common. Lowering the blood pressure with a vasodilator such as nitroprusside may be necessary. These problems typically resolve as warming occurs.

After cardiac surgery, the patient is at risk for developing elevated body temperature as a result of tissue inflammation or infection. The inflammatory/immune response to surgery includes the release of cytokines that cause fever (Norris, 2019). The resultant increase in metabolic rate increases tissue oxygen demands and increases cardiac workload. Antipyretics and other measures are used to lower body temperature.

Common sites of postoperative infection include the lungs, urinary tract, incisions, and intravascular catheters. Meticulous care is used to prevent contamination at the sites of catheter and tube insertions. Aseptic technique is used when changing dressings and when providing endotracheal tube and catheter care. Clearance of pulmonary secretions is accomplished by frequent repositioning of the patient, suctioning, and chest physical therapy, as well as educating and encouraging the patient to breathe deeply and cough. All invasive lines and tubes are discontinued as soon as possible after surgery to avoid infection.

Postpericardiotomy syndrome may occur in patients who undergo cardiac surgery. The syndrome is characterized by fever, pericardial pain, pleural pain, dyspnea, pericardial effusion, pericardial friction rub, and arthralgia. These signs and symptoms may occur days to weeks after surgery, often after the patient has been discharged from the hospital.

Postpericardiotomy syndrome must be differentiated from other postoperative complications (e.g., infection, incisional pain, MI, pulmonary embolus, bacterial endocarditis, pneumonia, atelectasis). Treatment depends on the severity of the signs and symptoms. Use of colchicine and anti-inflammatory agents may produce an improvement in symptoms (Lehto, Kiviniemi, Gunn, et al., 2018).

### Promoting Home, Community-Based, and Transitional Care



#### Educating Patients About Self-Care

Depending on the type of surgery and postoperative progress, the patient may be discharged from the hospital 3 to 5 days after surgery. Following recovery from the surgery, patients can expect fewer symptoms from CAD and an improved quality of life. CABG has been shown to increase the lifespan of high-risk patients, including those with left main artery blockages and left ventricular dysfunction with multivessel blockages (Urden et al., 2019).

Although the patient may be eager to return home, the patient and family usually are apprehensive about this transition. Family members often express the fear that they are not capable of caring for the patient at home or that they are unprepared to handle complications that may occur.

The nurse helps the patient and family set realistic, achievable goals. An education plan that meets the patient's individual needs is developed with the patient and family. Specific instructions are provided about incision care; signs and symptoms of infection; diet; activity progression and exercise; deep breathing, incentive spirometry, and tobacco use cessation; weight and temperature monitoring; the medication regimen; and follow-up visits with home health nurses, the rehabilitation personnel, the surgeon, and the cardiologist or internist.

Some patients have difficulty learning and retaining information after cardiac surgery. The patient may experience recent memory loss, short attention span, difficulty with simple math, poor handwriting, and visual disturbances. Patients with these difficulties often become frustrated when they try to resume normal activities. The patient and family are reassured that the difficulty is almost always temporary and will subside, usually in 6 to 8 weeks. In the meantime, instructions are given to

the patient at a slower pace than normal, and a family member assumes responsibility for making sure that the prescribed regimen is followed.

Chart 23-13



## HOME CARE CHECKLIST

### Discharge After Cardiac Surgery

#### At the completion of education, the patient and/or caregiver will be able to:

- Name the procedure that was performed and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- Identify interventions and strategies (e.g., durable medical equipment, adaptive equipment) used in recovery period.
- Describe ongoing postoperative therapeutic regimen, including diet and activities to perform (e.g., walking and breathing exercises) and to limit or avoid (e.g., lifting weights, driving a car, contact sports).
- State the name, dose, side effects, frequency, and schedule for all medications.
- State how to obtain medical supplies and carry out dressing changes, wound care, and other prescribed regimens.
- Identify durable medical equipment needs, proper usage, and maintenance necessary for safe utilization.
- Describe signs and symptoms of complications.
- State time and date of follow-up appointments.
- Relate how to reach the primary provider with questions or complications.
- Identify community resources for peer and caregiver/family support:
  - Identify sources of support (e.g., friends, relatives, faith community)
  - Identify contact information of support groups for people and their caregivers/families
- Identify the need for health promotion (e.g., weight reduction, cessation of tobacco use, stress management), disease prevention, and screening activities

ADLs, activities of daily living; IADLs, independent activities of daily living.

### Continuing and Transitional Care

Arrangements are made for home, community-based, or transitional care when appropriate. Because the hospital stay is relatively short, it is particularly important for the nurse to assess the patient's and family's ability to manage care in the home. The nurse making a home visit continues the education process (see [Chart 23-13](#)), monitors vital signs and incisions, assesses for signs and symptoms of complications, and provides support for the patient and family. Additional interventions may include dressing changes, diet counseling, and tobacco use cessation strategies. Patients and families need to know that cardiac surgery did not cure the patient's underlying heart disease process. Lifestyle changes for risk factor reduction are essential, and

medications taken before surgery to control problems such as blood pressure and hyperlipidemia will still be necessary.

The nurse encourages the patient to contact the surgeon, cardiologist, or office nurse with problems or questions. This provides the patient and family with reassurance that professional support is available. The patient is expected to have at least one follow-up visit with the surgeon.

Education does not end at the time of discharge from the hospital, transitional, or home health care. Many patients and families benefit from supportive programs, including cardiac rehabilitation. These programs provide monitored exercise; instructions about diet and stress reduction; information about resuming work, driving, and sex; assistance with tobacco use cessation; and support groups for patients and families. Hospital or community-based support groups provide information as well as an opportunity for families to share experiences.

### CRITICAL THINKING EXERCISES

**1 ipc** You have been assigned to care for a 64-year-old woman who was admitted last night complaining of fatigue and mild shortness of breath. She tells you that she does not understand why the doctors are so concerned about her heart. She states, “there is nothing wrong with my heart. I am just tired. What’s all the fuss about?” What education would you provide to the patient about women and heart disease? What clinical manifestations can indicate heart disease in women? What laboratory and diagnostic studies would you expect to be done for this patient? Which members of the interprofessional team would you want to involve in her care?

**2 pq** You are assigned to care for a 56-year-old man who is scheduled for a drug eluting stent today. You receive report from the cardiac catheter lab nurse that the patient tolerated the procedure well, is currently stable, and will be returning shortly to your unit. How will you prioritize care for this patient once he returns to his room? What assessments will you do first? What complications is he most at risk of developing and how will you know if they are occurring? What patient education should be initiated and when?

**3 ebp** You have been asked to provide an educational in-service to a group of nursing students on how to care for a patient presenting with a myocardial infarction (MI). One of the students asks you to explain how you determine if the patient is having an NSTEMI or a STEMI. How do you respond? According to evidence-based guidelines, what medications are used in the initial management of patients with an acute MI? According to these guidelines, under what conditions would a patient receive thrombolytic therapy rather than undergo a percutaneous coronary intervention?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

- American Diabetes Association, [www.diabetes.org/](http://www.diabetes.org/)  
 American Heart Association, [www.heart.org](http://www.heart.org)  
 Framingham Heart Study, [www.framinghamheartstudy.org](http://www.framinghamheartstudy.org)  
 National Heart, Lung, and Blood Institute, <https://www.nhlbi.nih.gov/>

# **24 Management of Patients with Structural, Infectious, and Inflammatory Cardiac Disorders**

## **LEARNING OUTCOMES**

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

- 1.** Define valvular disorders of the heart and describe the pathophysiology, clinical manifestations, as well as the medical and nursing management of patients with mitral and aortic disorders.
- 2.** Differentiate between the different types of cardiac valve repair and replacement procedures used to treat valvular problems and the care needed by patients who undergo these procedures.
- 3.** Identify the pathophysiology, clinical manifestations, as well as the medical and nursing management of patients with cardiomyopathies.
- 4.** Describe the pathophysiology, clinical manifestations, as well as the medical and nursing management of patients with infections of the heart.
- 5.** Use the nursing process as a framework for care of the patient with a cardiomyopathy or the patient with pericarditis.

## **NURSING CONCEPTS**

Infection  
Inflammation  
Perfusion

## GLOSSARY

**annuloplasty:** repair of a cardiac valve's outer ring

**aortic valve:** semilunar valve located between the left ventricle and aorta

**autograft:** heart valve replacement made from the patient's own heart valve (e.g., pulmonic valve excised and used as an aortic valve)

**bioprosthetic:** heart valve replacement made of tissue from an animal heart valve (*synonym:* heterograft)

**cardiomyopathy:** disease of the heart muscle

**chordae tendineae:** nondistensible fibrous strands connecting papillary muscles to atrioventricular (mitral, tricuspid) valve leaflets

**commissurotomy:** splitting or separating fused cardiac valve leaflets

**ejection fraction:** percentage of the end-diastolic blood volume ejected from the ventricle with each heartbeat

**homograft:** heart valve replacement made from a human heart valve (*synonym:* allograft)

**leaflet repair:** repair of a cardiac valve's movable "flaps" (leaflets)

**mitral valve:** atrioventricular valve located between the left atrium and left ventricle

**orthotopic transplantation:** the recipient's heart is removed and a donor heart is grafted into the same site

**prolapse: (of a valve):** stretching of an atrioventricular heart valve leaflet into the atrium during systole

**pulmonic valve:** semilunar valve located between the right ventricle and pulmonary artery

**regurgitation:** backward flow of blood through a heart valve (*synonym:* insufficiency)

**stenosis:** narrowing or obstruction of a cardiac valve's orifice

**total artificial heart:** mechanical device used to aid a failing heart, replacing the right and left ventricles

**tricuspid valve:** atrioventricular valve located between the right atrium and right ventricle

**valve replacement:** insertion of either a mechanical prosthetic valve or a bioprosthetic, homograft, or autograft tissue valve at the site of a malfunctioning heart valve to restore normal blood flow through the heart

**valvuloplasty:** repair of a stenosed or regurgitant cardiac valve by commissurotomy, annuloplasty, or leaflet repair (or a combination of procedures)

**ventricular assist device:** mechanical device used to aid a failing right or left ventricle

Structural, infectious, and inflammatory disorders of the heart present many challenges for the patient, family, and health care team. Various mechanisms, heart valve disorders, cardiomyopathies, and infectious diseases of the heart alter cardiac output. Treatments for these disorders may be noninvasive or invasive. Noninvasive treatments often consist of medication therapy, diet changes, and activity modification. Invasive treatments include valve repair or replacement, ventricular assist devices (VADs), total artificial hearts (TAHs), cardiac transplantation, or other surgical procedures. Nurses have an integral role in the care of patients with structural, infectious, and inflammatory cardiac conditions.

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## VALVULAR DISORDERS

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Valves of the heart control blood flow through the heart into the pulmonary artery and aorta by opening and closing in response to blood pressure changes during the cardiac cycle (systole, or heart contraction, and diastole, or relaxation of the heart).

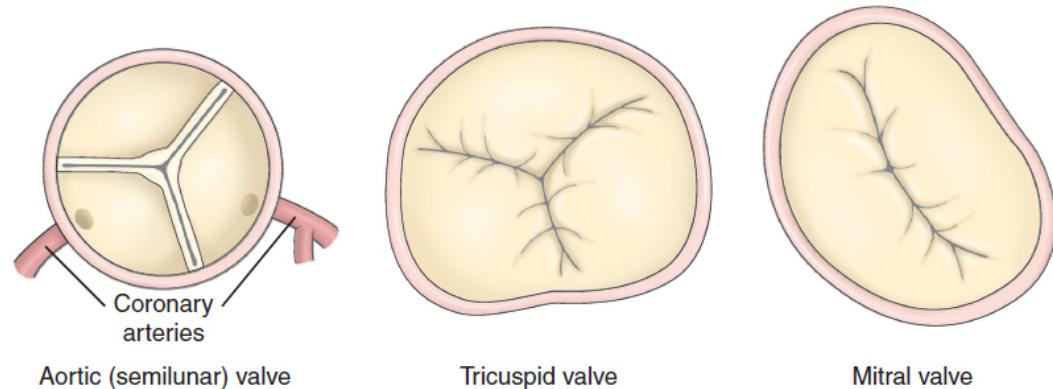
Atrioventricular valves separate the atria from the ventricles and include the **tricuspid valve**, which separates the right atrium from the right ventricle, and the **mitral valve**, which separates the left atrium from the left ventricle. The tricuspid valve has three leaflets; the mitral valve has two. Both valves have **chordae tendineae**, which are nondistensible fibrous strands that anchor valve leaflets to papillary muscles of the ventricles.

Semilunar valves are located between the ventricles and their corresponding arteries. The **pulmonic valve** lies between the right ventricle and the pulmonary artery; the **aortic valve** lies between the left ventricle and the aorta. [Figure 24-1](#) shows valves in the closed position (also refer to Chapter 21, [Fig. 21-1](#) to review the structure of a normal heart).

When any heart valve does not close or open properly, blood flow is affected. When valves do not close completely, blood flows backward through the valve, a condition called **regurgitation** (also referred to as insufficiency). When valves do not open completely, a condition called **stenosis**, blood flow through the valve is reduced.

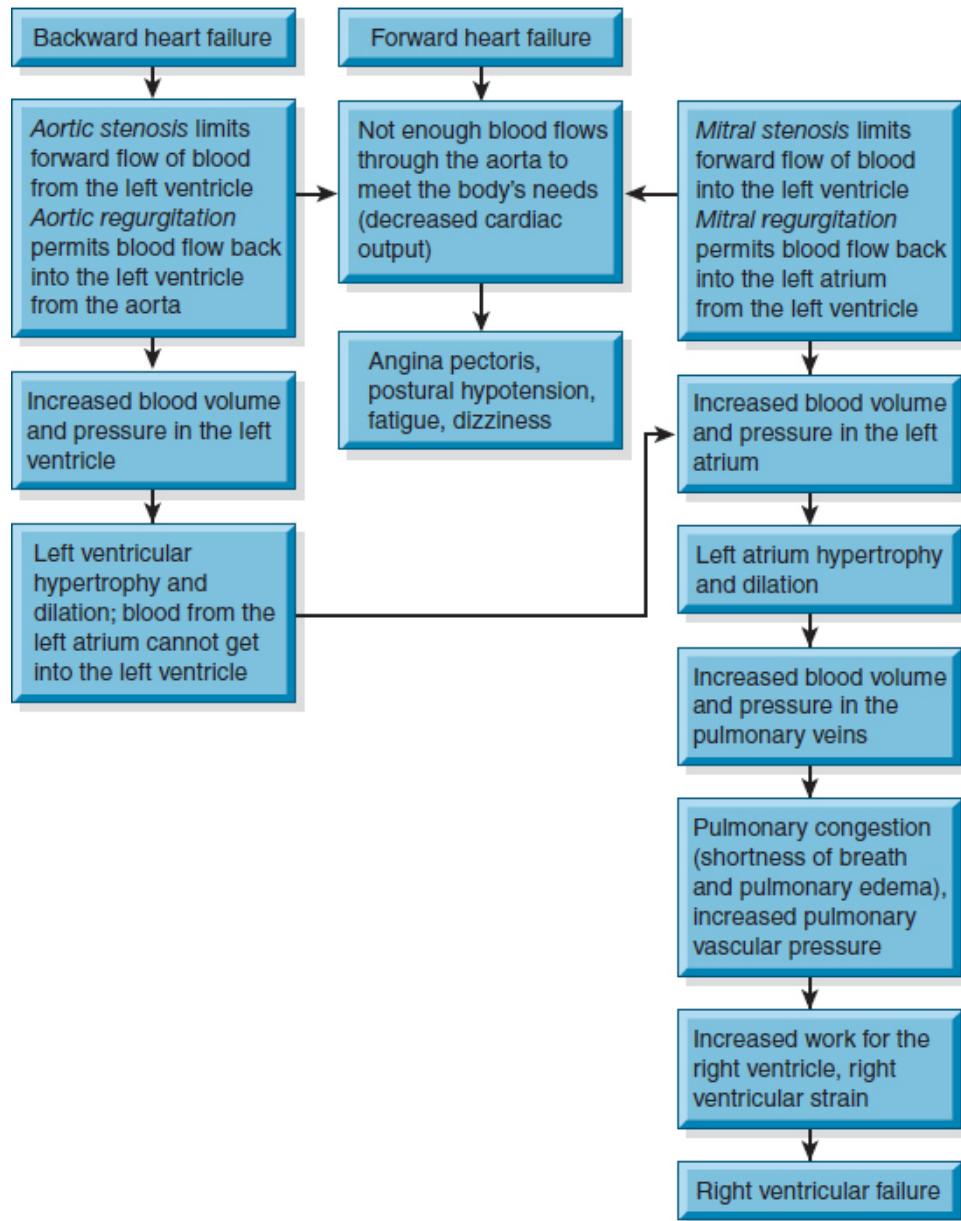
Regurgitation and stenosis may affect any heart valve. The mitral valve may also **prolapse** (i.e., stretching of the valve leaflet into the atrium during systole). For patients without symptoms, heart valve conditions may be monitored without treatment. If a patient has symptoms related to a valve disorder, treatment is based on the severity of symptoms, and patients may need to make lifestyle changes, take medications, or undergo repair or replacement of the valve. Disorders of the mitral and aortic valve typically cause more symptoms, require treatment, and cause more complications than

disorders of the tricuspid and pulmonic valves. Regurgitation and stenosis may occur at the same time in the same or different valves (Fig. 24-2).



**Figure 24-1 •** Valves of the heart (aortic or semilunar, tricuspid, and mitral) in closed positions.

## Physiology/Pathophysiology



**Figure 24-2 • Pathophysiology.** Left-sided heart failure as a result of aortic and mitral valvular heart disease and development of right ventricular failure.

## Mitral Valve Prolapse

Mitral valve prolapse is a deformity that usually produces no symptoms. Rarely, it progresses and can result in sudden death (Han, Ha, Teh, et al., 2018;

Nalliah, Mahajan, Elliott, et al., 2019). This condition occurs in up to 2.5% of the general population and twice as frequently in women as in men (Han et al., 2018). In most cases, there is no clear cause, but it has been associated with inherited connective tissue disorders, causing enlargement of one or both of the mitral valve leaflets (Asher, Chen, & Kallish, 2018; Wozniak-Mielczarek, Sabiniewicz, Drezek-Nojowicz, et al., 2019). The annulus often dilates; chordae tendineae and papillary muscles may elongate or rupture.

## Pathophysiology

In mitral valve prolapse, a portion of one or both mitral valve leaflets balloons back into the atrium during systole. Rarely, ballooning stretches the leaflet to the point that the valve does not remain closed during systole. Blood then regurgitates from the left ventricle back into the left atrium. Although uncommon, mitral valve prolapse can result in mitral regurgitation, which can cause heart enlargement, atrial fibrillation, pulmonary hypertension, or heart failure (Ma, Igata, Strachan, et al., 2019).

## Clinical Manifestations

Most people with mitral valve prolapse never have symptoms. A small number of patients will have fatigue, shortness of breath, lightheadedness, dizziness, syncope, palpitations, chest pain, or anxiety. To date, there has been no consensus about the cause of symptoms. Patients may report fatigue, regardless of activity level and amount of rest or sleep, as well as shortness of breath, palpitations, and chest pain (Althunayyan, Petersen, Lloyd, et al., 2019).

## Assessment and Diagnostic Findings

Often, the first and only sign of mitral valve prolapse is an extra heart sound, referred to as a mitral click. A systolic click is an early sign that a valve leaflet is ballooning into the left atrium. In addition to the mitral click, a murmur of mitral regurgitation may be heard if the valve opens during systole and blood flows back into the left atrium. If mitral regurgitation exists, a patient may experience signs and symptoms of heart failure (see [Chapter 25](#)). Echocardiography is used to diagnose and monitor progression of mitral valve prolapse (Han et al., 2018).

## Medical Management

Medical management is directed at controlling symptoms. If a patient who reports palpitations is found to have an arrhythmia, the patient may be advised

to eliminate caffeine and alcohol from the diet and to stop the use of tobacco products as well as electronic nicotine delivery systems (ENDS), including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars. Most patients do not require medication, but some are prescribed antiarrhythmic medications (see [Chapter 22](#), [Table 22-1](#)). Prophylactic antibiotics are not recommended prior to dental or invasive procedures (Nishimura, Otto, Bonow, et al., 2017).

Patients who have chest pain related to mitral valve prolapse rarely require medical therapies, such as nitrates, calcium channel blockers, or beta-blockers. Heart failure, if present, is treated as it would be for any other case of heart failure (see [Chapter 25](#)). Patients with severe mitral regurgitation and symptomatic heart failure may require mitral valve repair or replacement (discussed later in this chapter).

## Nursing Management

The nurse educates the patient about the diagnosis and the possibility that the condition is hereditary. First-degree relatives (e.g., parents, siblings) may be advised to have screening cardiac evaluations or echocardiograms. Because most patients with mitral valve prolapse are asymptomatic, the nurse explains the need to inform the patient's primary provider about any symptoms that may develop. The nurse encourages the patient to read product labels, particularly on over-the-counter products such as cough medicine, because these products may contain alcohol, caffeine, ephedrine, and epinephrine, which may produce arrhythmias and other symptoms. The nurse also explores diet, activity, sleep, and other lifestyle factors that may correlate with symptoms. (Treatment of arrhythmias, chest pain, heart failure, or other complications of mitral valve prolapse is described in Chapters 22 and 25.) Women diagnosed with mitral valve prolapse without mitral regurgitation or other complications may complete pregnancies without close cardiac monitoring and can safely proceed with vaginal deliveries (Yuan & Yan, 2016).

## Mitral Regurgitation

Mitral regurgitation is a condition in which blood flows from the left ventricle back into the left atrium during systole. Often, the edges of mitral valve leaflets do not close completely during systole because leaflets and chordae tendineae have thickened and become fibrotic, resulting in abnormal contraction. Mitral regurgitation may be chronic or, less commonly, acute. The most common causes of mitral valve regurgitation in developed countries are degenerative changes of the mitral valve (including mitral valve prolapse) and ischemia of the left ventricle (Harb & Griffin, 2017). The most common cause

in developing countries is rheumatic heart disease and its sequelae (Negi, Mahajan, Rana, et al., 2018).

Other conditions that lead to chronic mitral regurgitation include pathologic myxomatous changes, which enlarge and stretch the left atrium and ventricle, causing leaflets and chordae tendineae to stretch or rupture (Harb & Griffin, 2017). Infective endocarditis may cause acute mitral regurgitation through leaflet perforation, or scarring following an infection that may cause retraction of leaflets or chordae tendineae (Watanabe, 2019). Collagen vascular diseases (e.g., systemic lupus erythematosus), cardiomyopathy, and ischemic heart disease may result in changes in the left ventricle, causing papillary muscles, chordae tendineae, or leaflets to stretch, shorten, or rupture. These conditions are often referred to as functional, or secondary, mitral regurgitation (Dziadzko, Dziadzko, Medina-Inojosa, et al., 2019).

## Pathophysiology

Mitral regurgitation may result from problems with one or more leaflets, chordae tendineae, the annulus, or the papillary muscles. A mitral valve leaflet may shorten or tear, and chordae tendineae may elongate, shorten, or tear. The annulus may be stretched by heart enlargement, as in functional mitral regurgitation, or it may be deformed by calcification. A papillary muscle may rupture, stretch, or be pulled out of position by changes in the ventricular wall (e.g., scar from a myocardial infarction, ventricular dilation). Papillary muscles may be unable to contract because of ischemia, a condition referred to as ischemic mitral regurgitation. Regardless of the cause, the effect is backward blood flow into the atrium during systole.

In this disorder, each beat of the left ventricle pushes blood backward into the left atrium, adding to blood flowing in from the lungs. This excess blood causes the left atrium to stretch and eventually thicken, or hypertrophy, then dilate. Over time, blood coming in from the ventricle prevents blood flow from the lungs into the atrium. As a result, the lungs become congested, eventually adding extra strain to the right ventricle. During diastole, the increased blood volume from the atrium fills the ventricle. The volume overload causes ventricular hypertrophy. Eventually, the ventricle dilates, and systolic heart failure develops.

## Clinical Manifestations

Chronic mitral regurgitation is often asymptomatic, but acute mitral regurgitation (e.g., resulting from a myocardial infarction) usually manifests as severe and sudden congestive heart failure (Harb & Griffin, 2017). Dyspnea, fatigue, and weakness are the most common symptoms. Palpitations, shortness of breath on exertion, and cough from pulmonary congestion also occur.

## Assessment and Diagnostic Findings

The systolic murmur of mitral regurgitation is a blowing sound best heard at the apex. The murmur may radiate to the left axilla (Harb & Griffin, 2017). The pulse may be regular, or it may be irregular because of extrasystolic beats or atrial fibrillation. Echocardiography is used to diagnose and monitor progression of this disorder (Nishimura et al., 2017).

## Medical Management

Patients with mitral regurgitation who develop pulmonary congestion are managed with medications used for heart failure. Patients with mitral regurgitation and heart failure benefit from afterload reduction (arterial dilation) by treatment with angiotensin-converting enzyme (ACE) inhibitors (e.g., captopril, lisinopril) or angiotensin receptor blockers (ARBs) (e.g., losartan, valsartan), direct arterial dilators (e.g., hydralazine), and beta-blockers (e.g., carvedilol, metoprolol) (see [Chapter 25, Table 25-3](#)). Symptoms of heart failure also are an indication to consider surgical intervention by mitral **valvuloplasty** (i.e., surgical repair of the valve) or **valve replacement** (replacement of the dysfunctional valve with either a mechanical valve or a type of tissue valve; discussed later in this chapter) (Nishimura et al., 2017).

## Mitral Stenosis

Mitral stenosis results in reduced blood flow from the left atrium into the left ventricle. It is usually caused by rheumatic endocarditis, which progressively thickens mitral valve leaflets and chordae tendineae, causing the leaflets to fuse together (Negi et al., 2018). Eventually, the mitral valve orifice narrows and progressively obstructs blood flow into the ventricle.

## Pathophysiology

Normally, the mitral valve orifice is as wide as the diameter of three fingers. In severe mitral stenosis, the orifice narrows to the width of a pencil. Because of increased resistance through the narrowed valve orifice, the left atrium is less able to push blood into the left ventricle. This results in increased residual blood volume in the left atrium, which over time causes left atrial hypertrophy and dilation. Decreased blood flow into the left ventricle leads to reduced ventricular filling and decreased cardiac output. A stenotic valve fails to protect pulmonary veins from backward flow of blood from the atrium, resulting in congestion of the pulmonary circulation. The right ventricle must then contract against abnormally high pulmonary arterial pressure and is subjected to excessive strain. Over time, the right ventricle hypertrophies,

enlarges, and eventually fails. If the heart rate increases, diastole is shortened; thus, the amount of time for forward flow of blood decreases, and more blood backs into the pulmonary veins. Therefore, as the heart rate increases, cardiac output further decreases, and pulmonary pressures increase.

## Clinical Manifestations

Often, the first symptom of mitral stenosis is dyspnea on exertion (DOE) caused by pulmonary venous hypertension. Symptoms do not usually develop until after the valve opening is reduced by one third to one half its usual size (Harb & Griffin, 2017). Patients may experience progressive fatigue and decreased exercise tolerance because of low cardiac output. An enlarged left atrium may create pressure on the left bronchial tree, resulting in a dry cough or wheezing. In cases of severe mitral stenosis with significant pulmonary congestion, patients may expectorate blood (i.e., hemoptysis) or experience palpitations, orthopnea, paroxysmal nocturnal dyspnea (PND), or repeated respiratory infections. Increased blood volume and pressure cause the left atrium to dilate, hypertrophy, and become electrically unstable, which may result in patients developing atrial arrhythmias (Negi et al., 2018).

## Assessment and Diagnostic Findings

Patients with mitral stenosis will have a low-pitched, rumbling diastolic murmur, best heard at the apex. Patients may have a weak and irregular pulse if they develop atrial fibrillation and may have signs or symptoms of heart failure (Nishimura et al., 2017). Echocardiography is used to diagnose and quantify the severity of mitral stenosis. Electrocardiography (ECG), exercise testing, and cardiac catheterization with angiography may be used to help determine the severity of mitral stenosis.

## Prevention

Since rheumatic heart disease may result in mitral stenosis, prevention is aimed at decreasing the risk of contracting and providing early treatment for bacterial infections (see prevention of endocarditis later in this chapter). Prevention of acute rheumatic fever depends on effective antibiotic treatment of group A streptococcal infection (Nishimura et al., 2017). Antibiotic prophylaxis for recurrent rheumatic fever with rheumatic carditis may require 10 or more years of antibiotic coverage (e.g., penicillin G intramuscularly every 4 weeks, penicillin V orally twice daily, sulfadiazine orally daily, or erythromycin orally twice daily) (Szczygielska, Hernik, Kolodziejczyk, et al., 2018).

## Medical Management

Congestive heart failure is treated as described in Chapter 25. Patients with severe left atrial dilation in mitral stenosis may benefit from anticoagulant medications to decrease the risk of developing atrial thrombi. If atrial fibrillation develops, cardioversion may be attempted to restore normal sinus rhythm. If unsuccessful, the ventricular rate is controlled with beta-blockers, digoxin, or calcium channel blockers; furthermore, patients will require anticoagulation for thromboembolism prevention (January, Wann, Calkins, et al., 2019). Patients with severe mitral stenosis are advised to avoid strenuous activities, competitive sports, and pregnancy, all of which increase heart rate (Nishimura et al., 2017). Surgical intervention consists of valvuloplasty, usually a **commissurotomy** (i.e., splitting or separating leaflets) to open the fused commissure of the valve. The *commissure* is the site where valve leaflets meet. Percutaneous transluminal valvuloplasty or valve replacement may be performed.

## Aortic Regurgitation

Aortic regurgitation is backward flow of blood into the left ventricle from the aorta during diastole. It may be caused by a congenital valve abnormality (e.g., a bicuspid aortic valve), inflammatory lesions that deform aortic valve leaflets, or dilation of the aorta, preventing complete closure of the aortic valve. Chronic or acute aortic regurgitation may also be caused by infections such as rheumatic endocarditis or syphilis, or by a dissecting aortic aneurysm resulting in dilation or tearing of the ascending aorta, blunt chest trauma, or deterioration of a surgically replaced aortic valve (Akinseye, Pathak, & Ibebuogu, 2018; Nishimura et al., 2017).

## Pathophysiology

During diastole, blood is normally delivered into the left atrium from the aorta. In aortic regurgitation, blood flows back into the left ventricle, which will dilate to accommodate increased blood volume. Over time, the left ventricle hypertrophies to expel more blood with above-normal force, thus increasing systolic blood pressure. Arteries attempt to compensate for higher pressures by reflex vasodilation; peripheral arterioles relax, reducing peripheral resistance and diastolic blood pressure.

## Clinical Manifestations

Aortic regurgitation, also called aortic insufficiency, develops without symptoms in most patients. Some patients are aware of a pounding or forceful

heartbeat, especially in the head or neck. Patients who develop left ventricular hypertrophy may have visible or palpable arterial pulsations at the carotid or temporal arteries due to increased force and blood volume. As aortic regurgitation worsens, DOE and fatigue follow; there may eventually be signs and symptoms of progressive left ventricular failure including increased shortness of breath, orthopnea, or PND (Akinseye et al., 2018).

## Assessment and Diagnostic Findings

A high-pitched, blowing diastolic murmur is heard at the third or fourth intercostal space at the left sternal border. The difference between systolic and diastolic pressures (i.e., the pulse pressure) may be widened in patients with aortic regurgitation. One characteristic sign is the water hammer (Corrigan's) pulse, in which the pulse strikes a palpating finger with a quick, sharp stroke and then collapses (Pabba & Boudi, 2019). The diagnosis may be confirmed by echocardiography (preferably transesophageal), cardiac magnetic resonance imaging (MRI), or cardiac catheterization. Patients with symptoms usually have echocardiograms every 6 months, and those without symptoms have echocardiograms every 2 to 5 years (Nishimura et al., 2017).

## Prevention

Prevention of aortic regurgitation is primarily based on prevention of and treatment for bacterial infections (see prevention of endocarditis later in this chapter). The same strategies aimed at preventing acute and recurrent rheumatic fever previously described for the patient with mitral stenosis apply to patients with aortic regurgitation.

## Medical Management

A patient who is symptomatic or has developed a significant decrease in left ventricular function is advised to avoid physical exertion, competitive sports, and isometric exercise until the valve has been replaced (Gati, Malhotra, & Sharma, 2019). If arrhythmias and heart failure occur, they are treated as described in Chapters 22 and 25. Controlling high blood pressure in patients with aortic regurgitation can improve forward blood flow through the heart. ACE inhibitors and dihydropyridine calcium channel blockers may be recommended for management of hypertension; these are effective at reducing afterload. Beta-blockers are less commonly used, due to concern that a lower heart rate may actually increase blood pressure through negative chronotropic effects (Akinseye et al., 2018). Patients who are symptomatic should be instructed to restrict sodium intake to prevent volume overload and will require valve replacement (Nishimura, Otto, Bonow, et al., 2014).

The treatment of choice is aortic valve replacement or valvuloplasty (described later), preferably performed before left ventricular failure occurs. Surgery is recommended for any patient with significant left ventricular dilation, regardless of the presence or absence of symptoms (Nishimura et al., 2014). Surgery is also recommended for any patient who is symptomatic (Flint, Wunderlich, Shmueli, et al., 2019).

## Aortic Stenosis

Aortic valve stenosis is narrowing of the orifice between the left ventricle and aorta. In adults, stenosis is usually caused by degenerative calcification. Calcification may be caused by proliferative and inflammatory changes that occur in response to years of normal mechanical stress, similar to changes that occur in atherosclerotic cardiovascular disease (Joseph, Naqvi, Giri, et al., 2017). Congenital leaflet malformations or an abnormal number of leaflets (i.e., one or two rather than three) are less common causes. Rheumatic endocarditis may cause adhesions or fusion of the commissures and valve ring, stiffening of the cusps, and calcific nodules on the cusps.

## Pathophysiology

Typically, aortic stenosis progresses gradually over several years to several decades. As the valve orifice narrows, the left ventricle overcomes obstruction by contracting more slowly and more forcibly. Obstruction to left ventricular outflow increases pressure on the left ventricle, so the ventricular wall hypertrophies. When these compensatory mechanisms are insufficient to allow for normal heart function, clinical signs and symptoms of heart failure will develop (Joseph et al., 2017).

## Clinical Manifestations

Many patients with aortic stenosis are asymptomatic. Often, the first symptom to appear is DOE, caused by increased pulmonary venous pressure due to a dilating left ventricle. Over time, left ventricular failure may occur, causing orthopnea, PND, and pulmonary edema. Reduced blood flow to the brain may cause dizziness, and in more severe aortic stenosis, syncope. Patients may also report angina pectoris, which is caused by limited blood flow into the coronary arteries, decreased time in diastole to allow for myocardial perfusion, and simultaneously increased oxygen demand of the hypertrophied left ventricle. Blood pressure is usually normal but may be low. In the setting of decreased blood flow, there may be a low pulse pressure (30 mm Hg or less).

## Assessment and Diagnostic Findings

On physical examination, a loud, harsh systolic murmur is heard over the aortic area (i.e., right second intercostal space) and may radiate to the carotid arteries and apex of the left ventricle. The murmur may be described as low pitched, crescendo-decrescendo, rough, rasping, and vibrating (Libby, Zipes, Bonow, et al., 2018). An S<sub>4</sub> sound may be heard (see [Chapter 21](#) for discussion of heart sounds). By having the patient lean forward during auscultation and palpation, especially during exhalation, the murmur may be accentuated. There may also be a palpable vibration extending from the base of the heart (second intercostal space next to the sternum and above the suprasternal notch) and up along the carotid arteries. The vibration is caused by turbulent blood flow across the narrowed valve orifice.

Cardiac imaging is used to diagnose and monitor the progression of aortic stenosis. This may consist of echocardiography, cardiac MRI, or computed tomography (CT) scanning (Lindman, Dweck, Lancellotti, et al., 2019). Patients with symptoms usually have echocardiograms every 6 to 12 months, and those without symptoms have echocardiograms every 2 to 5 years, depending on how severely the orifice is narrowed (Lindman et al., 2019; Nishimura et al., 2014). Left ventricular hypertrophy may be seen on a 12-lead ECG or an echocardiogram. Once aortic stenosis has progressed sufficiently to consider surgical intervention, left-sided heart catheterization is needed to measure the severity of the valvular abnormality and to evaluate the coronary arteries. Pressure measurements are taken from the left ventricle and base of the aorta. The systolic pressure in the left ventricle is considerably higher than that in the aorta during systole. Graded exercise studies (stress tests) to assess exercise capacity are performed with caution for patients with severe aortic stenosis due to the high risk of inducing ventricular tachycardia or fibrillation, and should not be performed on symptomatic patients (Joseph et al., 2017).

## Prevention

Prevention of aortic stenosis is primarily focused on controlling risk factors for proliferative and inflammatory responses—namely, through treating diabetes, hypertension, hypercholesterolemia, and elevated triglycerides, and avoiding tobacco products and ENDS (see prevention of endocarditis later in this chapter).

## Medical Management

Medications are prescribed to treat arrhythmias or left ventricular failure (see Chapters 22 and 25). Definitive treatment for aortic stenosis is replacement of the aortic valve, which may be done surgically or nonsurgically. Nonsurgical

valve replacement, known as transcatheter aortic valve replacement (TAVR), is described in more detail later in this chapter. Patients who are symptomatic and are not candidates for valve replacement may benefit from one- or two-balloon percutaneous valvuloplasty procedures, which can provide symptom relief (Sandhu, Krishnamoorthy, Afif, et al., 2017).

## Nursing Management: Valvular Heart Disorders

The nurse educates the patient with valvular heart disease about the diagnosis, progressive nature of the disease, and treatment plan. The patient is instructed to report new symptoms or changes in symptoms to the primary provider. The nurse also educates the patient that an infectious pathogen, usually a bacterium, can adhere to a diseased heart valve more readily than to a normal valve. Once attached to the valve, the infectious agent multiplies, resulting in endocarditis and further damage to the valve. In addition, the nurse educates the patient about how to minimize the risk of developing infective endocarditis (discussed later in this chapter).

The nurse measures the patient's heart rate, blood pressure, and respiratory rate, compares these results with previous data, and notes any changes. Heart and lung sounds are auscultated, and peripheral pulses palpated. The nurse assesses the patient with valvular heart disease for the following:

- Signs and symptoms of heart failure, such as fatigue, DOE, decreased activity tolerance, an increase in coughing, hemoptysis, multiple respiratory infections, orthopnea, and PND (see [Chapter 25](#))
- Arrhythmias, by palpating the patient's pulse for strength and rhythm (i.e., regular or irregular) and asking whether the patient has experienced palpitations or felt forceful heartbeats (see [Chapter 22](#))
- Symptoms such as dizziness, syncope, increased weakness, or angina pectoris (see [Chapter 23](#))

The nurse collaborates with the patient to develop a medication schedule and provides education about the name, dosage, actions, adverse effects, and any drug–drug or drug–food interactions of prescribed medications for heart failure, arrhythmias, angina pectoris, or other symptoms. Specific precautions are emphasized, such as the risk to patients with aortic stenosis who experience angina pectoris and take nitroglycerin. The venous dilation that results from nitroglycerin use decreases blood return to the heart, thus decreasing cardiac output and increasing the risk of syncope and decreased coronary artery blood flow. The nurse educates the patient about the importance of attempting to relieve the symptoms of angina with rest and relaxation before taking nitroglycerin and to anticipate potential adverse effects.

For patients with heart failure, the nurse provides education on taking a daily weight and reporting sudden weight gain, as defined by the primary provider. The nurse may assist the patient with planning activity and rest periods to allow symptom relief while preventing deconditioning or loss of function. Care of patients treated with valvuloplasty or surgical valve replacement is described later in this chapter.

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## SURGICAL MANAGEMENT: VALVE REPAIR AND REPLACEMENT PROCEDURES

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### Valvuloplasty

Repair, rather than replacement, of a cardiac valve is referred to as valvuloplasty. The recommended procedure for valvuloplasty will depend on the cause and type of valve dysfunction. Repair may be made to commissures between the leaflets in a procedure known as commissurotomy, to the annulus of the valve by **annuloplasty** (i.e., specifically, repair of the cardiac valve's outer ring), or to leaflets. Transesophageal echocardiogram (TEE) is usually performed at the conclusion of a valvuloplasty to evaluate the effectiveness of the procedure.

Some valvuloplasty procedures are open-heart surgeries, which are performed under general anesthesia and usually use cardiopulmonary bypass. However, there are now several valvuloplasty procedures which utilize nonsurgical, or percutaneous, techniques; these do not require general anesthesia or cardiopulmonary bypass and can be performed in a cardiac catheterization laboratory or hybrid room. A hybrid room is an operating room with imaging capability (e.g., fluoroscopy, CT, MRI) and interventional devices for open, minimally invasive, image-guided and catheter-based procedures. Percutaneous partial cardiopulmonary bypass is used in some cardiac catheterization laboratories and hybrid rooms. (Cardiopulmonary bypass is described in Chapter 23.)

### Commissurotomy

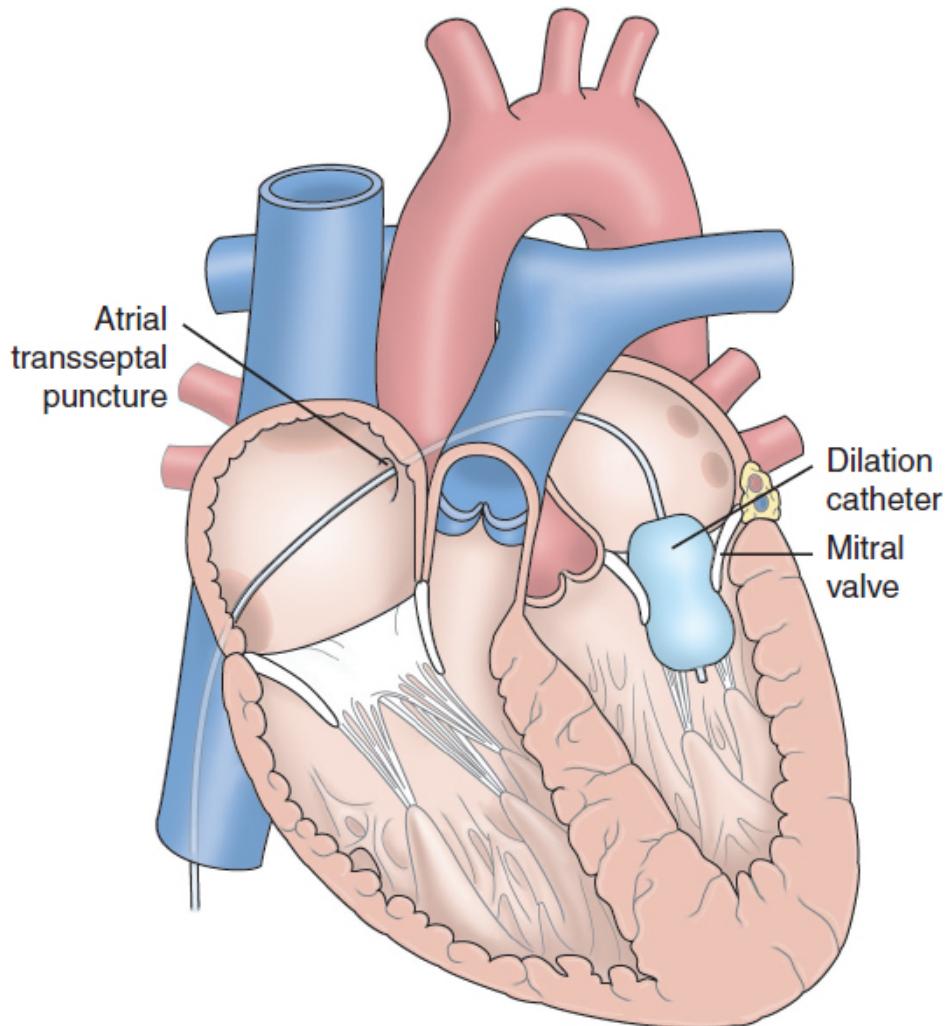
Valve leaflets may adhere to one another and close the commissure (i.e., stenosis). Less commonly, leaflets adhere to one another in a manner which creates stenosis as well as regurgitation, or backward blood flow. A commissurotomy is performed to separate the fused leaflets.

### Closed Commissurotomy/Balloon Valvuloplasty

Commissurotomy is usually used for mitral valve stenosis. The preferred method is percutaneous transvenous mitral commissurotomy, which may be used for patients with congenital mitral stenosis, severe calcified mitral stenosis, left atrial thrombus, moderate to severe coexisting mitral regurgitation, or in patients with coexisting moderate to severe tricuspid regurgitation who would also benefit from tricuspid valve repair (Nishimura et al., 2014).

Most often used for mitral and aortic valve stenosis, balloon valvuloplasty is less commonly used to treat tricuspid and pulmonic valve stenosis. With more widely available percutaneous valve repair and replacement procedures, balloon valvuloplasty is becoming a less common procedure in the United States (Kumar, Paniagua, Hira, et al., 2016). Balloon valvuloplasty may still be used for mitral valve stenosis in younger patients, and for patients with complex medical conditions that place them at high risk for complications of more extensive surgical procedures. The procedure is contraindicated for patients with left atrial or ventricular thrombus, severe aortic root dilation, significant mitral valve regurgitation, and severe valvular calcification (Nishimura et al., 2014).

Balloon valvuloplasty (Fig. 24-3) is performed in a cardiac catheterization laboratory. The patient may receive light or moderate sedation and a local anesthetic. Mitral balloon valvuloplasty involves advancing one or two catheters into the right atrium, through the atrial septum into the left atrium, across the mitral valve, and into the left ventricle. A guidewire is placed through each catheter, and the original catheter is removed. Most often a specially designed balloon catheter is placed over the guidewire and positioned with the balloon across the mitral valve. The balloon has three sections with progressively greater resistance to inflation. The balloon first expands in the ventricle to help position the catheter at the valve. The second section of the balloon expands above the valve, holding the catheter across the valve. Finally, the middle section of the balloon expands in the valve orifice opening the commissures. Alternately, two balloons are used. Guidewires may be advanced into the aorta to stabilize the balloons' positions. These single-section balloons are inflated simultaneously and expand their entire length. The advantage of two balloons is that each is smaller than the one large balloon, making smaller atrial septal defects. As the two balloons are inflated, they usually do not completely occlude the valve, thereby permitting some forward flow of blood during the inflation period. Balloons are inflated with a dilute angiographic solution for 10 to 30 seconds. Multiple inflations usually are required to achieve the desired results (Hermann & Mack, 2019).



**Figure 24-3 •** Balloon valvuloplasty. Cross-section of heart illustrating the dilation catheter placed through an atrial transseptal puncture and across the mitral valve. The Inoue-Balloon inflates in three stages: first below the valve, then above, and finally in the valve orifice (this diagram shows the first two sections inflated).

All patients will have some degree of mitral regurgitation following mitral balloon valvuloplasty. Other possible complications include bleeding from the catheter insertion sites, emboli resulting in complications such as strokes, and, rarely, left-to-right atrial shunts through the atrial septal defect created during the procedure.

Aortic balloon valvuloplasty is usually performed by introducing a catheter through the aorta, across the aortic valve, and into the left ventricle; less commonly, it may be performed by passing the balloon or balloons through the atrial septum. The one- or two-balloon technique can be used for treating aortic stenosis. Balloons are inflated for 15 to 60 seconds, and inflation is usually repeated multiple times. Possible complications include aortic

regurgitation, emboli, ventricular perforation, rupture of the aortic valve annulus, ventricular arrhythmia, mitral valve damage, and bleeding from the catheter insertion sites. The aortic valve procedure is not as effective as the mitral valve procedure, and the rate of restenosis is approximately 50% in the first 6 months after the procedure. It is usually used for palliation of symptoms in patients who are not suitable candidates for TAVR, but may sometimes be used as a bridge to TAVR (Hermann & Mack, 2019; Kumar et al., 2016).

### **Open Commissurotomy**

Since percutaneous commissurotomy has fewer risks with similar outcomes, open commissurotomy has become less commonly used in recent decades. The patient is under general anesthesia, and a midsternal or left thoracic incision is made. Cardiopulmonary bypass is initiated, and an incision is made into the heart. The valve is exposed, and a scalpel, finger, balloon, or dilator is used to open the commissures. One advantage of directly visualizing the valve and surrounding structures is that a thrombus or calcifications may be removed. Chordae or papillary muscles may also be inspected and surgically repaired as necessary.

### **Annuloplasty**

The junction at which valve leaflets connect to the heart wall is an annulus. Annuloplasty refers to repair of the valve annulus, resulting in narrowing of the valve orifice. It is used for valvular regurgitation. For the mitral valve, there may be damage or rupture of the chordae tendineae resulting in severe mitral regurgitation, which is an indication for urgent valve repair by annuloplasty with resuspension of the chordae tendineae. General anesthesia and cardiopulmonary bypass are required for most annuloplasties.

There are two annuloplasty techniques. One technique uses an annuloplasty ring ([Fig. 24-4](#)), which may be rigid/semirigid or flexible. Leaflets of the valve are sutured to a ring, creating an annulus of the desired size. When the ring is in place, tension created by moving blood and the contracting heart is borne by the ring rather than by the valve or a suture line, thereby preventing progressive regurgitation.

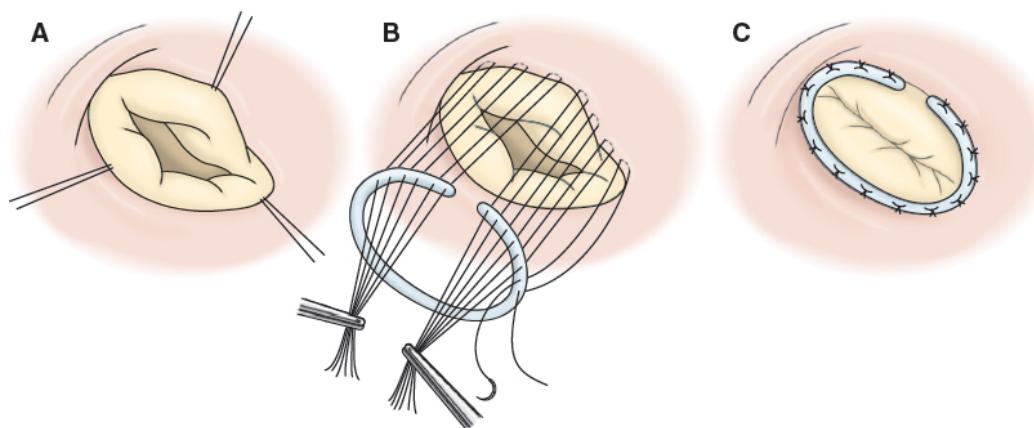
A second technique to tighten the annulus involves folding elongated tissue over onto itself in leaflets or tacking leaflets to the atrium or each other with sutures. Because the valve's leaflets and suture lines are subjected to direct forces of the blood and heart muscle movement, the repair may degenerate more quickly than one using an annuloplasty ring (Maisano, Skantharaja, Denti, et al., 2019).

### **Leaflet Repair**

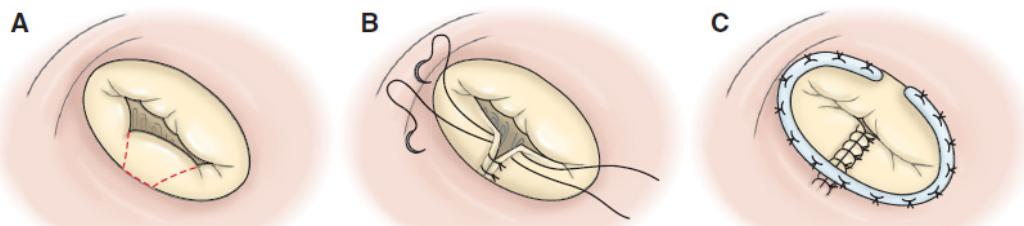
Damage to cardiac valve leaflets may result from stretching, shortening, or tearing. **Leaflet repair** for elongated, ballooning, or other excess tissue leaflets is achieved by removing the extra tissue. Elongated tissue may be tucked and sutured, a technique called plication. A wedge of tissue may be cut from the middle of the leaflet and the gap sutured closed (i.e., leaflet resection) (Fig. 24-5). After short chordae are released, leaflets often unfurl and resume their normal function, allowing the valve to close during systole. A leaflet may be extended by suturing a piece of pericardium to it. A pericardial or synthetic patch may be used to repair holes in the leaflets.

## Valve Replacement

Valve replacement is preferred for patients with valves with anatomy which decreases the chance of success with repair; this includes valves that have extensive calcification, or with severely fibrotic or fused leaflets, chordae tendineae, or papillary muscles. Ideally, a multidisciplinary team (e.g., cardiologists, cardiac thoracic surgeons, structural valve interventionists, anesthesiologists, nurses) will work together with the patient to determine candidacy for surgical versus more minimally invasive replacement (Nishimura et al., 2014). General anesthesia and cardiopulmonary bypass are used for surgical valve replacements. The standard surgical procedure is performed through a median sternotomy (i.e., incision through the sternum), although the mitral valve may be approached through a right thoracotomy incision.



**Figure 24-4 •** Annuloplasty ring insertion. **A.** Mitral valve regurgitation; leaflets do not close. **B.** Insertion of an annuloplasty ring. **C.** Completed valvuloplasty; leaflets close.



**Figure 24-5 •** Valve leaflet resection and repair with a ring annuloplasty. **A.** Mitral valve regurgitation; the section indicated by *dashed lines* is excised. **B.** Approximation of edges and suturing. **C.** Completed valvuloplasty, leaflet repair, and annuloplasty ring.

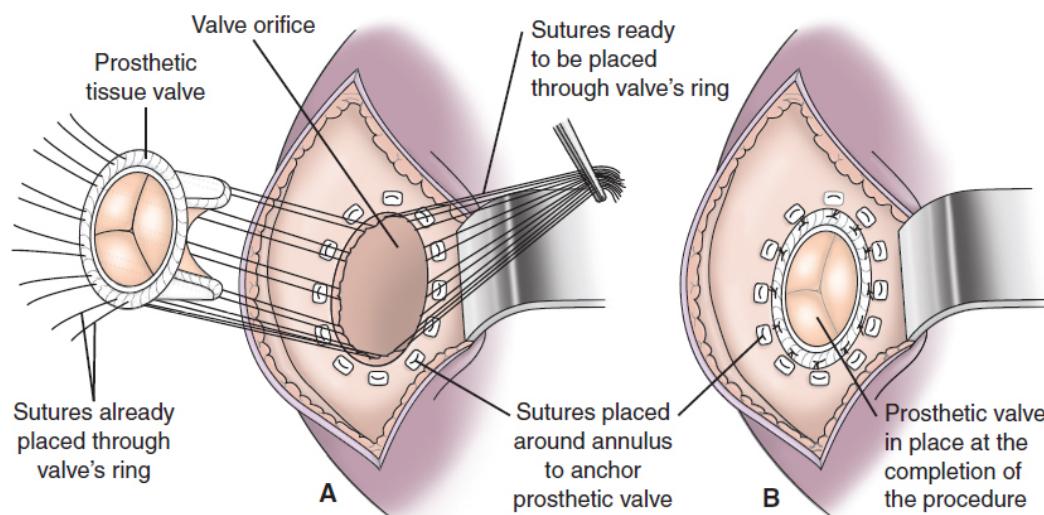
Mitral and aortic valve replacements may be performed with minimally invasive techniques that do not involve cutting through the length of the sternum. Instead, a 2- to 4-inch incision is made in only the upper or lower half of the sternum or between ribs, or performed percutaneously. Some minimally invasive procedures are robot assisted; surgical instruments are connected to a robot, and the surgeon, watching a video display, uses a joystick to control the robot and surgical instruments. With these procedures, patients have lower rates of bleeding, pain, infection, and scarring, and shorter length of stay in the hospital (Nishimura et al., 2014).

After the valve is visualized, leaflets of the aortic or pulmonic valve are removed, but some or all of the mitral valve structures (leaflets, chordae, and papillary muscles) are left in place to help maintain the shape and function of the left ventricle after mitral valve replacement. Sutures are placed around the annulus and then through the valve prosthesis. The replacement valve is slid down the suture into position and tied into place (Fig. 24-6). The patient is weaned from cardiopulmonary bypass, the quality of the surgical repair is often assessed with color flow Doppler TEE, and then surgery is completed.

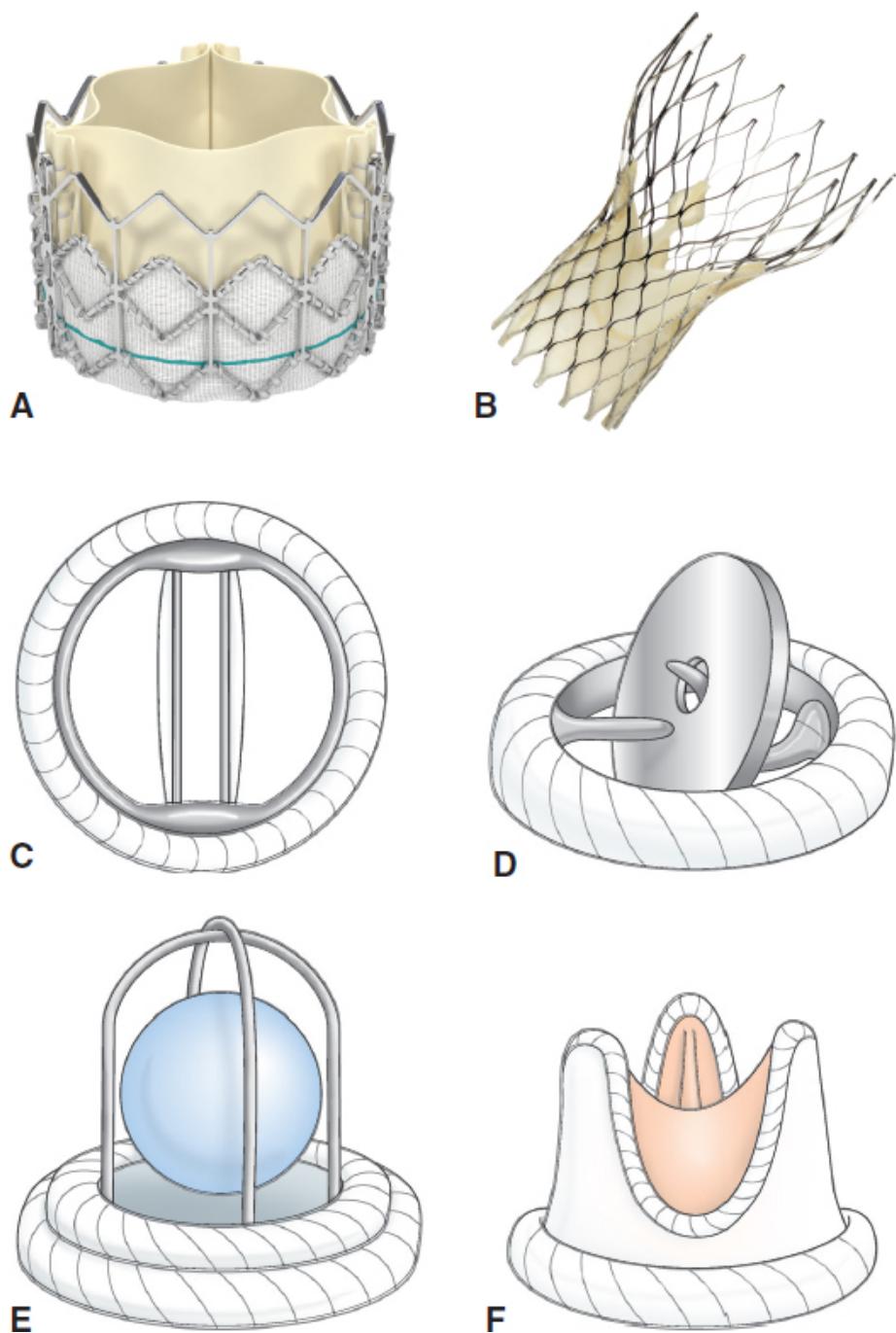
TAVR, a minimally invasive aortic valve replacement procedure, may be performed in a catheterization laboratory or hybrid room. It does not involve cardiopulmonary bypass or sternotomy. TAVR was traditionally used only for patients who could not safely undergo surgical valve replacement, but more recently has been recommended for patients who have intermediate or even low risk (Nishimura et al., 2017). With the patient under general anesthesia, a balloon valvuloplasty is performed. Then, a bioprosthetic (tissue) replacement valve (Fig. 24-7A,B) attached to a catheter is inserted percutaneously, positioned at the aortic valve, and implanted. The MitraClip has been approved as a transcatheter procedure to treat degenerative forms of mitral regurgitation. This procedure is currently used primarily in patients with severe symptomatic mitral regurgitation and high surgical risk, but increasingly is considered even for lower-risk patients. The procedure creates a mechanical bridge between two leaflets and has demonstrated decreased regurgitant flow, resulting in

fewer symptoms, and decreased rates of hospitalization for heart failure (Dahl & Ailawadi, 2018).

In patients requiring valve replacement or repair, the heart had gradually adjusted to the pathology; surgery abruptly “corrects” the way blood flows through the heart and may result in complications related to the sudden changes in intracardiac pressures. All prosthetic valve replacements create a degree of stenosis when they are implanted in the heart. Usually, the stenosis is mild and does not affect heart function. If valve replacement was for a stenotic valve, blood flow through the heart is often improved, and signs and symptoms of heart failure resolve in a few hours or days. If valve replacement was for a regurgitant valve, it may take months for the chamber into which blood had been regurgitating to achieve its optimal postoperative function. Signs and symptoms of heart failure resolve gradually as heart function improves. Patients are at risk for many postoperative complications, such as bleeding, thromboembolism, infection, heart failure, hypertension, arrhythmias, hemolysis, and mechanical obstruction of the valve.



**Figure 24-6 •** Valve replacement. **A.** The native valve is trimmed, and the prosthetic valve is sutured in place. **B.** Once all sutures are placed through the ring, the surgeon slides the prosthetic valve down the sutures and into the natural orifice. Sutures are then tied off and trimmed.



**Figure 24-7 •** Common mechanical and tissue valve replacements. **A.** Transcatheter aortic valve (Edwards SAPIEN transcatheter heart valve, tissue). Used with the permission of Edwards Lifesciences LLC, Irvine, CA; Edwards SAPIEN and SAPIEN are trademarks of Edwards Lifesciences Corporation. **B.** Transcatheter aortic valve (Medtronic The CoreValve® system, tissue). Used with the permission of Medtronic. The CoreValve System® CoreValve is a registered trademark of Medtronic CV Luxembourg S.A.R.L.). **C.** Bileaflet (St. Jude, mechanical). **D.** Tilting disc valve (Medtronic-Hall,

mechanical). **E.** Caged ball valve (Starr-Edwards, mechanical). **F.** Porcine bioprosthesis valve (Carpenter-Edwards, tissue).

Several types of mechanical and tissue valve prostheses may be used (Chikwe & Castillo, 2017) ([Fig. 24-7](#)).

## Mechanical Valves

Mechanical valves are of the bileaflet ([Fig. 24-7C](#)), tilting disc ([Fig. 24-7D](#)), or caged ball ([Fig. 24-7E](#)) design and are thought to be more durable than tissue prosthetic valves ([Fig. 24-7F](#)); therefore, they often are used for younger patients. These valves also are used for patients requiring valve replacement who also have kidney injury, hypercalcemia, endocarditis, or sepsis, since mechanical valves do not deteriorate or become infected as easily as tissue valves. Significant complications associated with mechanical valves are thromboemboli and complications that can be associated with long-term use of required anticoagulants (see [Chapter 26](#) for further discussion).

## Tissue Valves

There are three types of tissue valves: bioprostheses, homografts, and autografts. Tissue valves are less likely than mechanical valves to generate thromboemboli, and long-term anticoagulation is not required. Tissue valves are not as durable as mechanical valves and require replacement more rapidly (Chikwe & Castillo, 2017; Nishimura et al., 2014).

### Bioprostheses

**Bioprostheses** are valves made from animal tissues (i.e., heterografts) used for aortic, mitral, and tricuspid valve replacement. They are not thrombogenic; therefore, patients do not need long-term anticoagulation therapy. They are used for women of childbearing age to avoid potential complications of long-term anticoagulation associated with menses, placental transfer to a fetus, and delivery of a child. They also are considered for patients older than 70 years and others who cannot tolerate long-term anticoagulation. Most bioprostheses are from pigs (porcine), but some are from cows (bovine) or horses (equine). They may be stented or nonstented. Viability is 7 to 15 years.

### Homografts

**Homografts**, also called allografts (i.e., human valves), are obtained from cadaver tissue donations and are used for aortic and pulmonic valve replacement. The aortic valve and a portion of aorta or the pulmonic valve and a portion of pulmonary artery are harvested and stored cryogenically.

Homografts are not always available and are very expensive. They last for about 10 to 15 years and are typically used in younger patients who require replacement of the valve root (Tudorache, Horke, Cebotari, et al., 2016).

### Autografts

**Autografts** (i.e., autologous valves) are obtained by excising the patient's own pulmonic valve and a portion of the pulmonary artery for use as the aortic valve. Anticoagulation is unnecessary because the valve is the patient's own tissue and is not thrombogenic. The autograft is an alternative for children (it may grow as the child grows), women of childbearing age, young adults, patients with a history of peptic ulcer disease, and people who cannot tolerate anticoagulation. Aortic valve autografts have remained viable for more than 20 years (Mazine, El-Hamamsy, Verma, et al., 2018). If pulmonary vascular pressures are normal, some surgeons elect not to then replace the pulmonic valve. Patients can recover without a valve between the right ventricle and pulmonary artery.

However, most aortic valve autograft procedures are double valve replacement procedures with a homograft pulmonic valve replacement also performed. If there is recurrent valve dysfunction after this procedure, patients may undergo further corrective surgery, often in the form of a reversal of the original homograft. In patients who are older and for whom this may no longer be an option, replacement with a prosthetic valve may be considered (Hussain, Majdalany, Dunn, et al., 2018).



## Nursing Management: Valvuloplasty and Valve Replacement

The nurse assists the patient and family to prepare for the procedure, reinforces and supplements explanations provided by the primary provider, and provides psychosocial support. (See Chapters 14 through 16 for care of the surgical patient.)

Patients who have undergone percutaneous balloon valvuloplasty with or without percutaneous valve replacement may be admitted to a telemetry unit or intensive care unit (ICU). The nurse assesses for signs and symptoms of heart failure and emboli (see [Chapter 25](#)), auscultates the chest for changes in heart sounds at least every 4 hours, and provides the patient with the same care as for postprocedure cardiac catheterization or percutaneous transluminal coronary angioplasty (see [Chapter 23](#)). After undergoing percutaneous balloon valvuloplasty, the patient usually remains in the hospital for 24 to 48 hours.

Patients who have undergone surgical valvuloplasty or valve replacements are admitted to the ICU. Care focuses on recovery from anesthesia and

hemodynamic stability. Vital signs are assessed every 5 to 15 minutes and as needed until the patient recovers from anesthesia or sedation, and then are assessed according to unit protocol. Intravenous (IV) medications may be used to increase or decrease blood pressure, to treat arrhythmias, to increase or decrease heart rate; and, their effects are monitored. Medications are gradually decreased until they are no longer required, or the patient takes the needed medication by another route (e.g., oral, topical). Patient assessments are conducted every 1 to 4 hours and as needed, with attention to neurologic, respiratory, and cardiovascular systems (see [Chapter 23](#), Chart 23-11).

After the patient has recovered from anesthesia and sedation, is hemodynamically stable without the use IV medications, and has stable physical assessment parameters, they are usually transferred to a telemetry unit, typically within 24 to 72 hours of surgery. Nursing care continues as for most postoperative patients, including wound care and patient education regarding diet, activity, medications, and self-care. The patient usually is discharged from the hospital in 3 to 7 days.

The nurse educates the patient about anticoagulant therapy, explaining the need for frequent follow-up appointments and blood laboratory studies. Patients who take warfarin have individualized target international normalized ratios, usually between 2 and 3.5 for mitral valve replacement and 1.8 and 2.2 for aortic valve replacement. Patients who have been treated with an annuloplasty ring or a tissue valve replacement usually require anticoagulation for only 3 months unless there are other risk factors such as atrial fibrillation or a history of thromboembolism. Aspirin is prescribed with warfarin for patients with bioprostheses or at high risk for embolic events (e.g., history of embolic event or having two or more preexisting conditions: diabetes, hypertension, coronary artery disease, congestive heart failure, older than 75 years) (Nishimura et al., 2014). The nurse provides education about all prescribed medications, including the name of medication, dosage, actions, prescribed schedule, potential adverse effects, and any drug-drug or drug-food interactions.

Patients with a mechanical valve prosthesis (including annuloplasty rings and other prosthetic materials used in valvuloplasty) require education to prevent infective endocarditis. Patients may be at risk for infective endocarditis, caused by bacteria entering the bloodstream and adhering to abnormal valve structures or prosthetic devices. The nurse educates the patient about how to minimize the risk of developing infective endocarditis (see prevention of endocarditis later in this chapter).

Transitional, home health, office, or clinic nurses help reinforce all new information and self-care instructions with patients and families for 4 to 8 weeks after the procedure ([Chart 24-1](#)). An echocardiogram may be performed 3 to 4 weeks after hospital discharge to further evaluate the effects and results of surgery. The echocardiogram also provides a baseline for future comparison

if cardiac symptoms or complications develop. Echocardiograms usually are repeated every 1 to 2 years.

## Cardiomyopathy

**Cardiomyopathy** is disease of the heart muscle that is associated with cardiac dysfunction. It is classified according to the structural and functional abnormalities of the heart muscle: dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy (RCM), arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), and unclassified cardiomyopathy (Elliott, Andersson, Arbustini, et al., 2008). A patient may have pathology representing more than one of these classifications, such as a patient with HCM with restrictive physiology. *Ischemic cardiomyopathy* is a term frequently used to describe an enlarged heart caused by coronary artery disease, which is usually accompanied by heart failure (see [Chapter 25](#)). In 2006, the American Heart Association proposed a set of Contemporary Classifications for cardiomyopathies, which continues to be in widespread use. Under this classification system, cardiomyopathies are divided into two major groups based on predominant organ involvement. These include *primary cardiomyopathies* (genetic, nongenetic, and acquired), which are focused primarily on the heart muscle, and *secondary cardiomyopathies*, which show myocardial involvement secondary to the influence of a vast list of disease processes that include, but are not limited to, amyloidosis, Fabry disease, sarcoidosis, and scleroderma (Maron, Towbin, Thiene, et al., 2006). This chapter focuses on the primary cardiomyopathies.

## Pathophysiology

The pathophysiology of all cardiomyopathies is a series of events that culminate in impaired cardiac output. Decreased stroke volume stimulates the sympathetic nervous system and the renin–angiotensin–aldosterone response, resulting in increased systemic vascular resistance and increased sodium and fluid retention, which place an increased workload on the heart. Often, the decrease in cardiac output can be seen on echocardiogram as a decrease in **ejection fraction**, expressed as a percentage of the end-diastolic blood volume ejected from the ventricle with each heartbeat. These alterations can lead to heart failure (see [Chapter 25](#)).

Chart 24-1



HOME CARE CHECKLIST

## Discharge After Valve Replacement

**At the completion of education, the patient and/or caregiver will be able to:**

- Name the procedure that was performed, any complications that occurred, and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- Identify interventions and strategies (e.g., durable medical equipment, adaptive equipment) used in recovery period.
- Describe ongoing postoperative therapeutic regimen, including diet and activities to perform (e.g., walking and breathing exercises) and to limit or avoid (e.g., lifting weights, driving a car, contact sports).
- State the name, dose, side effects, frequency, and schedule for all medications, including anticoagulant.
  - Identify the need to take anticoagulant for prescribed length of time
  - Take anticoagulant at same time each day
  - Keep appointments for laboratory tests, if indicated
  - Avoid injury that can cause bleeding
  - Report signs that could suggest occult bleeding to primary provider (e.g., bleeding gums, petechiae formation, tarry stools)
- State how to obtain medical supplies and carry out dressing changes, wound care, and other prescribed regimens.
- Identify durable medical equipment needs, proper usage, and maintenance necessary for safe utilization.
- Describe signs and symptoms of complications, including infective endocarditis, if at risk.
  - Report fevers, chills, clusters of petechiae, malaise, and weight loss to primary provider
- Engage in activities aimed at preventing infective endocarditis, as indicated:
  - Notify all health care providers of surgery and possible need for antibiotic prophylaxis preprocedure, if endorsed by primary provider
  - Practice good oral hygiene, including brushing, flossing, and regular appointments with dental hygienist
- State time and date of follow-up appointments.
- Relate how to reach the primary provider with questions or complications.
- Identify community resources for peer and caregiver/family support:
  - Identify sources of support (e.g., friends, relatives, faith community)
  - Identify the contact details for support services for patients and their caregivers/families

- Identify the need for health promotion (e.g., weight reduction, smoking cessation, stress management), disease prevention, and screening activities

ADL, activities of daily living; IADL, independent activities of daily living.



### Concept Mastery Alert

Sodium is the major electrolyte involved with cardiomyopathy. Cardiomyopathy often leads to heart failure, which develops, in part, from fluid overload. Fluid overload is often associated with elevated sodium intake.

## Dilated Cardiomyopathy

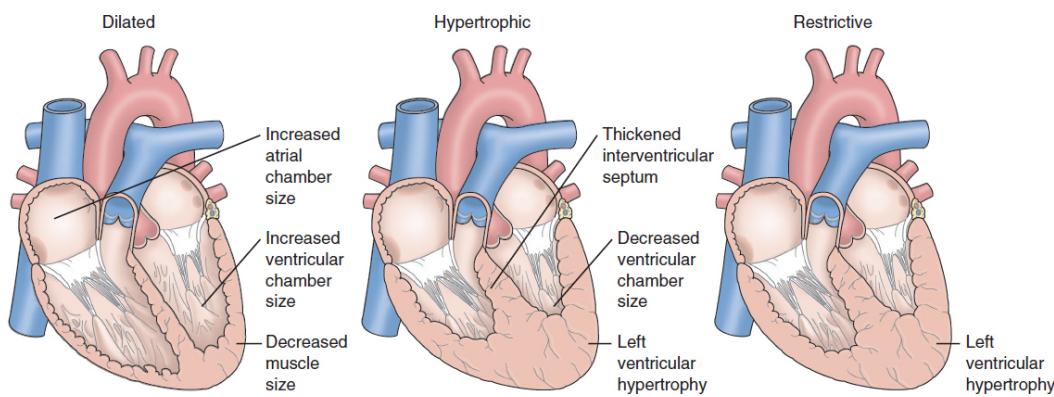
DCM is the most common form of cardiomyopathy, with a general prevalence of between 1 in 250 and 1 in 2500 (Merlo, Cannata, Gobbo, et al., 2018). DCM is distinguished by significant dilation of the ventricles without simultaneous hypertrophy and systolic dysfunction (Fig. 24-8). The ventricles have elevated systolic and diastolic volumes but a decreased ejection fraction.

Microscopic examination of the muscle tissue shows diminished contractile elements (actin and myosin filaments) of the muscle fibers and diffuse necrosis of myocardial cells. The result is poor systolic function. The structural changes decrease the amount of blood ejected from the ventricle with systole, increasing the amount of blood remaining in the ventricle after contraction. Less blood is then able to enter the ventricle during diastole, increasing end-diastolic pressure and eventually increasing pulmonary and systemic venous pressures. Altered valve function, usually regurgitation, can result from an enlarged stretched ventricle. Poor blood flow through the ventricle may also cause ventricular or atrial thrombi, which may embolize to other locations in the body.

More than 75 conditions and diseases may cause DCM, including pregnancy, hypertension, heavy alcohol intake, viral infection (e.g., influenza), chemotherapeutic medications (e.g., daunorubicin, doxorubicin), thyrotoxicosis, myxedema, persistent tachycardia, and Chagas disease. When the causative factor cannot be identified, the diagnosis is idiopathic DCM which accounts for 20% to 30% of nonischemic DCM cases (McNally & Mestroni, 2017; Merlo et al., 2018). Familial DCM accounts for approximately 30% to 50% of all DCM cases and approximately 40% of familial DCM cases have a definitive genetic etiology (McNally & Mestroni, 2017). An elucidation of family history by the nurse is therefore a very important component of the assessment process. Early diagnosis and treatment can prevent or delay significant symptoms and sudden death from DCM.

## Hypertrophic Cardiomyopathy

The estimated prevalence of HCM is 0.16% to 0.29% of the adult population (Marian & Braunwald, 2017). HCM is an autosomal dominant genetic disorder that leads to increased heart muscle size and mass, especially along the septum (see Fig. 24-8) but can involve other areas of the heart. The phenotypic expression of the disease is age dependent. HCM is the leading cause of sudden death in adolescents and young adults, particularly in athletes (Marian & Braunwald, 2017). 12-lead ECGs, physical examinations, and echocardiograms are used to screen for the disease.



**Figure 24-8 •** Cardiomyopathies that lead to congestive heart failure. Adapted from Anatomical Chart Company. (2010). *Atlas of pathophysiology* (3rd ed.). Ambler, PA: Lippincott Williams & Wilkins.

Patients with a suspected diagnosis of HCM should undergo genetic testing; if negative, the diagnosis is not completely ruled out. If genetic testing is positive for known HCM genetic mutations, first-degree relatives should also be tested for the genetic mutation found in the patient. In patients that have positive genetic testing, but are asymptomatic for cardiomyopathy, annual screening with an ECG, physical examination and echocardiogram should be done, as the likelihood of clinical progression increases with increasing age. The phenotype typically manifests sometime between adolescence and the fifth decade of life (Marian & Braunwald, 2017).

Cardiac muscle cells normally lie parallel to and end to end with each other. The hypertrophied cardiac muscle cells are disorganized, oblique, and perpendicular to each other, decreasing the effectiveness of contractions. In HCM, the coronary arteriole walls are thickened, which decrease the internal diameter of the arterioles. The narrow arterioles restrict the blood supply to the myocardium, causing numerous small areas of ischemia and necrosis. The necrotic areas of the myocardium ultimately fibrose and scar, further impeding ventricular contraction and possibly increasing the risk of arrhythmias such as ventricular tachycardia and ventricular fibrillation (see Chapter 22).

Increased thickness of the heart muscle reduces the size of the ventricular cavities and causes the ventricles to take a longer time to relax after systole. During the first part of diastole, it is more difficult for the ventricles to fill with blood. The atrial contraction at the end of diastole becomes critical for ventricular filling and systolic contraction.

HCM can lead to obstruction of the left ventricular outflow tract (LVOT) if there is systolic anterior motion of the mitral valve that abuts the mitral valve against the hypertrophied septum during systole (Marian & Braunwald, 2017; Nishimura, Seggewiss, & Schaff, 2017). LVOT obstruction is a dynamic process that is dependent on both the volume of blood in the left ventricle as well as the ability of the myocytes to contract. Approximately one third of patients with HCM have LVOT obstruction at rest that worsens with provocation, another one third do not have obstruction at rest, but can get obstruction with provocation (e.g., exercise or the Valsalva maneuver), and roughly one third of patients have no LVOT obstruction even with provocation (Marian & Braunwald, 2017). Obstruction of the LVOT can lead to syncope, ventricular arrhythmias, dyspnea, and heart failure. The presence of a systolic ejection murmur can be indicative of LVOT, and echocardiography is then indicated to confirm its presence. Hydration, beta-blockers, calcium channel blockers, and lifestyle modification can be used to minimize LVOT obstruction. In particular, patients should avoid activities that can cause rapid alterations to preload (e.g., hot tubs, saunas, prolonged hot showers) (Nishimura et al., 2017). However, patients that do not respond to medical therapy should be considered for surgical myectomy or alcohol septal ablation to decrease the size of the hypertrophied septum and thereby eliminate LVOT obstruction.

## Restrictive Cardiomyopathy

RCM is the least common type of cardiomyopathy (Muchtar, Blauwet, & Gertz, 2017; Pereira, Grogan, & Dec, 2018). RCM is characterized by diastolic dysfunction caused by rigid ventricular walls that impair diastolic filling and ventricular stretch (see [Fig. 24-8](#)). A rigid ventricle alters the curve in the Frank–Starling law (see [Chapter 21](#)) and leads to the rapid rise of filling pressures despite only small increases in blood volume. However, chamber size and systolic function are usually normal. Arrhythmias and conduction disturbances are common. Signs and symptoms are similar to constrictive pericarditis (see later discussion) and include dyspnea, nonproductive cough, and chest pain. Echocardiography may be useful in differentiating between these two conditions.

Generally, RCM is either due to an inherited or acquired disease that may be systemic. There are four general categories for the causes of RCM: infiltrative disease, storage disease, noninfiltrative, and endomyocardial.

An example of an infiltrative disease that may cause RCM is amyloidosis, in which amyloid, a misfolded protein, is deposited between cardiomyocytes. An inherited storage disease that can lead to RCM is hemochromatosis, in which iron deposits in the heart lead to cardiac stiffness. Scleroderma is a noninfiltrative connective tissue disorder that can cause RCM. Certain cancer treatments, such as radiation and use of various chemotherapeutic agents (e.g., anthracyclines) can cause endomyocardial damage that leads to RCM (Muchtar et al., 2017). Often, endomyocardial biopsy is needed to determine the etiology; treatment is then directed at the underlying cause.

### **Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia**

ARVC/D is an uncommon form of inherited heart muscle disease. The prevalence is estimated to be between 1 in 2000 and 1 in 5000 people in the general population (Bennett, Haqqani, Berruezo, et al., 2019). ARVC/D occurs when the myocardium is progressively infiltrated and replaced by fibrous scar and adipose tissue. Infiltration of fibrous and adipose tissue leads to ventricle dilatation, poor contractility, and arrhythmias.

Initially, only localized areas of the right ventricle are affected, but as the disease progresses, the entire heart is affected. Because of this typical pathologic progression, there is a move to change the name of this cardiomyopathy to a more general term of arrhythmogenic cardiomyopathy (ACM) to recognize the left ventricular involvement (Bennett et al., 2019).

In patients with ARVC/D, palpitations or syncope may develop between 15 and 40 years of age. Sudden cardiac death may also be the first presentation. Diagnosis is made based on the ECG, echocardiogram, cardiac MRI, and family history. Since this is a genetic disorder, patients that are diagnosed are referred to a genetic counselor for testing. However, genetic testing can be negative in up to 50% of patients (Bennett et al., 2019). If a genetic mutation is identified, first-degree relatives of the patient should undergo genetic testing. Patients affected by arrhythmias may benefit from having an implantable cardioverter defibrillator (ICD) placed (see [Chapter 22](#)).

### **Unclassified Cardiomyopathies**

Unclassified cardiomyopathies are different from or have characteristics of more than one of the previously described types and are caused by fibroelastosis, noncompacted myocardium, systolic dysfunction with minimal dilation, and mitochondrial diseases. Examples of unclassified cardiomyopathies can include left ventricular noncompaction and stress-induced (Takotsubo) cardiomyopathy (Elliott et al., 2008).

## **Clinical Manifestations**

Patients with cardiomyopathy may remain stable and without symptoms for many years. As the disease progresses, so do the symptoms. Frequently, dilated or restrictive cardiomyopathy is first diagnosed when the patient presents with signs and symptoms of heart failure (e.g., DOE, fatigue, PND, cough [especially with exertion or at night], orthopnea, peripheral edema, early satiety, nausea; see [Chapter 25](#)). The patient also may experience chest pain, palpitations, dizziness, nausea, and syncope with exertion.

## Assessment and Diagnostic Findings

Physical examination at early stages may reveal tachycardia and extra heart sounds (e.g., S<sub>3</sub>, S<sub>4</sub>). Patients with DCM may have diastolic murmurs, and patients with DCM and HCM may have systolic murmurs. With disease progression, examination also reveals signs and symptoms of heart failure (e.g., crackles on pulmonary auscultation, jugular vein distention, pitting edema of dependent body parts, hepatomegaly [i.e., enlarged liver]).

Diagnosis is usually made from findings disclosed by the patient history and by ruling out other causes of heart failure such as myocardial infarction. The echocardiogram is one of the most helpful diagnostic tools because the structure and function of the ventricles can be observed easily. Cardiac MRI may also be used, particularly to assist with the diagnosis of HCM and ARVC/D. ECG may demonstrate arrhythmias (atrial fibrillation, ventricular arrhythmias) and changes consistent with left ventricular hypertrophy (left axis deviation, wide QRS, ST changes, inverted T waves). In ARVC/D, the ECG may show QRS widening, T-wave inversions in leads V<sub>1</sub>–V<sub>4</sub>, and ventricular ectopy. Additionally, there is often a small epsilon wave at the end of the QRS (Bennett et al., 2019). The chest x-ray reveals heart enlargement and possibly pulmonary congestion. Cardiac catheterization, coronary CT, or stress testing is often used to rule out coronary artery disease as a causative factor. Endomyocardial biopsy may be performed to analyze myocardial cells, particularly in RCM.

## Medical Management

Medical management is directed toward identifying and managing possible underlying or precipitating causes; correcting the heart failure with medications, a low sodium diet, and an exercise/rest regimen (see [Chapter 25](#)); and controlling arrhythmias with antiarrhythmic medications and possibly with an implanted electronic device, such as an ICD (see [Chapter 22](#)). If the patient has signs and symptoms of congestion, fluid intake may be limited to 2 L each day. However, patients with HCM should avoid dehydration and may need beta-blockers to maintain cardiac output and minimize the risk of LVOT obstruction during systole. Anticoagulants are no longer routinely prescribed.

For some patients with DCM, biventricular pacing (also known as cardiac resynchronization therapy or CRT) increases the ejection fraction and reverses some of the structural changes in the myocardium (see [Chapter 22](#)).

As noted previously, some forms of cardiomyopathy are inherited/genetic. Any family history of cardiomyopathy should be noted when the nurse is taking a family health history. In appropriate cases, the patient should be referred to a genetic counselor for testing. If genetic mutations are found, it is recommended that first-degree relatives also be tested. Unfortunately, there are currently some problems with patients receiving insurance reimbursement for genetic testing in the United States.

## Surgical Management

When heart failure progresses and medical treatment is no longer effective, surgical intervention, including heart transplantation, is considered. However, because of the limited number of organ donors, many patients die waiting for transplantation. In some cases, a VAD is implanted to support the failing heart until a suitable donor heart becomes available (see later discussion).

### Left Ventricular Outflow Tract Surgery

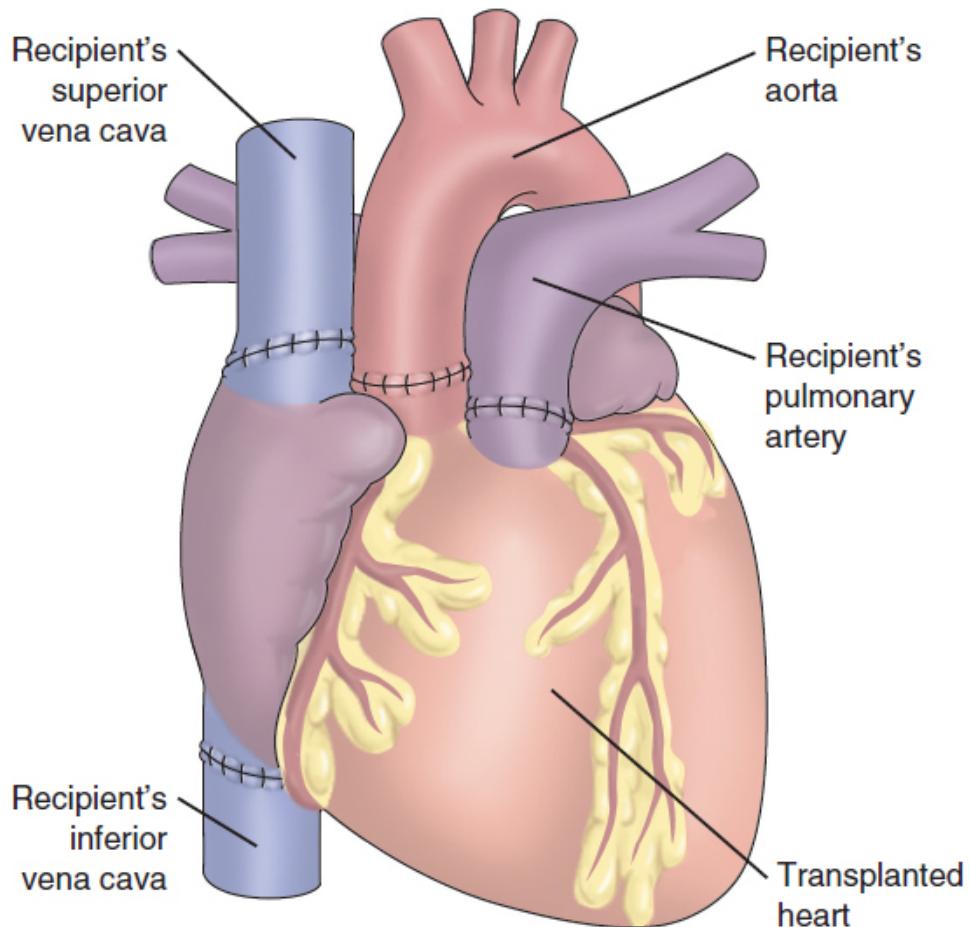
When patients with HCM and LVOT obstruction become symptomatic despite optimal medical therapy or cannot tolerate medical therapy, surgery is considered. The most common procedure done is a myectomy (sometimes referred to as a myotomy–myectomy or the Morrow procedure), in which some of the heart tissue is excised. Septal tissue approximately 1 cm wide and deep is cut from the enlarged septum below the aortic valve. The length of septum removed typically extends to the papillary muscles. Possible complications include complete heart block and subsequent pacemaker dependence, ventricular septal defects, or failure to adequately alleviate the obstruction. Surgical mortality rates are reportedly between less than 1% and as high as 16% (Nishimura et al., 2017).

### Heart Transplantation

Because of advances in surgical techniques and immunosuppressive therapies, heart transplantation is now a therapeutic option for patients with end-stage heart disease. Cyclosporine and tacrolimus are some of the more common immunosuppressants that decrease the body's rejection of foreign proteins, such as transplanted organs. Unfortunately, these drugs also decrease the body's ability to resist infections and increase the risk of various cancers, and a satisfactory balance must be achieved between suppressing rejection and avoiding infection. In 2017, there were 3273 heart transplants performed in the United States (Scientific Registry of Transplant Recipients, 2017).

Cardiomyopathy, ischemic heart disease, valvular disease, rejection of previously transplanted hearts, and congenital heart disease are the most common indications for transplantation. Typical candidates have severe symptoms uncontrolled by medical therapy, no other surgical options, and a prognosis of less than 1 to 2 years to live. A multidisciplinary team screens the candidate before recommending the transplantation procedure. The person's age, pulmonary status, other chronic health conditions, psychosocial status, family support, infections, history of other transplantations, adherence to therapeutic regimens, and current health status are considered. The United Network for Organ Sharing (UNOS), a national organization that is regulated by the U.S. government, is charged with maintaining organ transplant waiting lists and allocating donor organs. When a donor heart becomes available, UNOS generates a list of potential recipients on the basis of ABO blood group compatibility, the body sizes of the donor and the potential recipient, age, severity of illness, length of time on the waiting list, and the geographic locations of the donor and potential recipient (Organ Procurement and Transplant Network [OPTN], 2019). Some patients are candidates for more than one organ transplant (e.g., heart-lung, heart-kidney, heart-liver).

**Orthotopic transplantation** is the most common surgical procedure for cardiac transplantation. Some surgeons prefer to remove the recipient's heart but leave a portion of the recipient's atria (with the vena cava and pulmonary veins) in place, which is known as the biatrial technique. However, this technique has been modified to a more common approach called the *bicaval technique*. This technique includes removal of the recipient's heart, and the implantation of the donor heart with intact atria at the vena cava and pulmonary veins ([Fig. 24-9](#)) (Kittleson, Patel, & Kobashigawa, 2017). This newer approach is associated with decreased AV valve regurgitation, arrhythmias, and conduction abnormalities.



**Figure 24-9 •** Orthotopic method of heart transplantation.

Patients who have had heart transplantations are constantly balancing the risk of rejection with the risk of infection and diseases such as cancer. They must adhere to a complex regimen of diet, medications, activity, follow-up laboratory studies, biopsies of the transplanted heart (to diagnose rejection), and clinic visits. There are three classes of medications that are prescribed for a transplant patient to help minimize rejection: corticosteroids (e.g., prednisone), calcineurin inhibitors (tacrolimus, cyclosporine), and antiproliferative agents (mycophenolate mofetil, azathioprine, or sirolimus).

The transplanted heart has no nerve connections (i.e., denervated heart) to the recipient's body, so the sympathetic and vagus nerves do not affect the transplanted heart. The resting rate of the transplanted heart is approximately 90 to 110 bpm, but it increases gradually if catecholamines are in the circulation. Patients must gradually increase and decrease their exercise (i.e., extended warm-up and cool-down periods), because 20 to 30 minutes may be required to achieve the desired heart rate. Atropine does not increase the heart rate of transplanted hearts, and digoxin does not decrease the heart rate in atrial fibrillation. Additionally, many patients who have had heart transplantations do

not experience angina with ischemia and may present with congestive heart failure, silent myocardial infarction, or sudden death without a prior history of coronary artery disease (Kittleson et al., 2017).

In addition to rejection and infection, complications may include accelerated atherosclerosis of the coronary arteries (i.e., cardiac allograft vasculopathy, accelerated graft atherosclerosis, transplant coronary artery disease). Both immunologic and nonimmunologic factors cause arterial injury and inflammation of the coronary arteries. The arterial smooth muscle proliferates, and there is hyperplasia of the coronary artery intima, accelerating atherosclerosis along the entire length of the coronary arteries (Stehlik, Kobashigawa, Hunt, et al., 2018). Hypertension may occur in patients taking cyclosporine or tacrolimus due to the effect these medications have on the kidneys. Osteoporosis is a frequent side effect of the antirejection medications as well as pretransplantation dietary insufficiency and medications. Patients with a long-term sedentary lifestyle are at greater risk for osteoporosis. Posttransplantation lymphoproliferative disease and cancer of the skin and lips are the most common malignancies after transplantation, possibly caused by immunosuppression. Weight gain, obesity, diabetes, dyslipidemias (e.g., hypercholesterolemia), hypertension, and kidney failure, as well as central nervous system, respiratory, and gastrointestinal disturbances, may be adverse effects of corticosteroids or other immunosuppressants. Toxicity from immunosuppressant medications may occur as well. For patients receiving their transplanted hearts after the year 2000, the median survival is more than 12 years (Stehlik et al., 2018).

In the first year after transplantation, patients respond to the psychosocial stresses imposed by organ transplant in various ways. Most report a higher quality of life and can return to activities of daily living and to work with little to no functional limitations (Stehlik et al., 2018). Some have significant feelings of indebtedness to the donor, or experience guilt that someone had to die for them to be able to live, which can negatively affect their adherence to the medical regimen (Shemesh, Peles-Bortz, Peled, et al., 2017) (see [Chart 24-2](#) Nursing Research Profile: Factors Affecting Nonadherence After Heart Transplant).

## Mechanical Assist Devices and Total Artificial Hearts

The use of cardiopulmonary bypass in cardiovascular surgery and the possibility of performing heart transplantation in patients with end-stage cardiac disease, as well as the desire for a treatment option for patients who are not transplant candidates, have increased the need for mechanical assist devices. Patients who cannot be weaned from cardiopulmonary bypass and patients in cardiogenic shock may benefit from a period of mechanical heart assistance. The most commonly used device is the intra-aortic balloon pump

(see [Chapter 11](#)). This pump decreases the work of the heart during contraction but does not perform the actual work of the heart.

### Ventricular Assist Devices

More complex devices that perform some or all of the pumping function for the heart are now being used. These more sophisticated **ventricular assist devices** can circulate as much blood per minute as the heart, if not more ([Fig. 24-10](#)). There are short- and long-term devices available, depending on the indication. Each VAD is used to support one ventricle, although in some instances, two VAD pumps may be used for biventricular support. Additionally, some VADs can be combined with an oxygenator; the combination is called *extracorporeal membrane oxygenation (ECMO)*. The oxygenator–VAD combination is used for the patient whose heart cannot pump adequate blood through the body or when the lungs fail to oxygenate the blood despite supplemental oxygen or ventilation.

VADs may be external, internal (i.e., implanted) with an external power source, or completely internal, and they may generate a pulsatile or continuous blood flow. There are four types of VADs: pneumatic, electric or electromagnetic, axial flow, and centrifugal. Pneumatic VADs are external or implanted pulsatile devices with a flexible reservoir housed in a rigid exterior. The reservoir usually fills with blood drained from the atrium or ventricle. The device then forces pressurized air into the rigid housing, compressing the reservoir and returning the blood to the circulation, usually into the aorta. Electric or electromagnetic VADs are similar to pneumatic VADs, but instead of using pressurized air to return the blood to the circulation, one or more flat metal plates are pushed against the reservoir. Generally, pulsatile VADs have been replaced by the newer generation of axial and centrifugal pumps. The axial and centrifugal pumps have lower rates of pump thrombus formation and are of smaller device sizes. Axial flow VADs use a rotary mechanism (an impeller) to create nonpulsatile blood flow. The impeller spins rapidly within the VAD, creating a vacuum that pulls blood into the VAD and then pushes the blood out into the systemic circulation—the process is similar to a fan spinning in a tunnel, pulling air in one end of the tunnel and pushing it out the other. Centrifugal VADs are nonpulsatile devices that consist of a single moving impeller that is suspended in the pump housing by a combination of magnetic and hydrodynamic forces. The impeller rotates and pulls blood into the pump housing and ejects the blood out to the systemic circulation (Kittleson et al., 2017).

### Chart 24-2 NURSING RESEARCH PROFILE

## Factors Affecting Nonadherence After Heart Transplant

Shemesh, Y., Peles-Bortz, A., Peled, Y., et al. (2017). Feelings of indebtedness and guilt toward donor and immunosuppressive medication adherence among heart transplant (HTx) patients, as assessed in a cross-sectional study with the Basel Assessment of Adherence to Immunosuppressive Medications Scale (BAASIS). *Clinical Transplantation*, 31(10). doi:10.1111/ctr.13053

### Purpose

The purpose of this study was to assess immunosuppression medication adherence among patients who had heart transplants and to test if adherence was affected by feelings of indebtedness and guilt toward the donor.

### Design

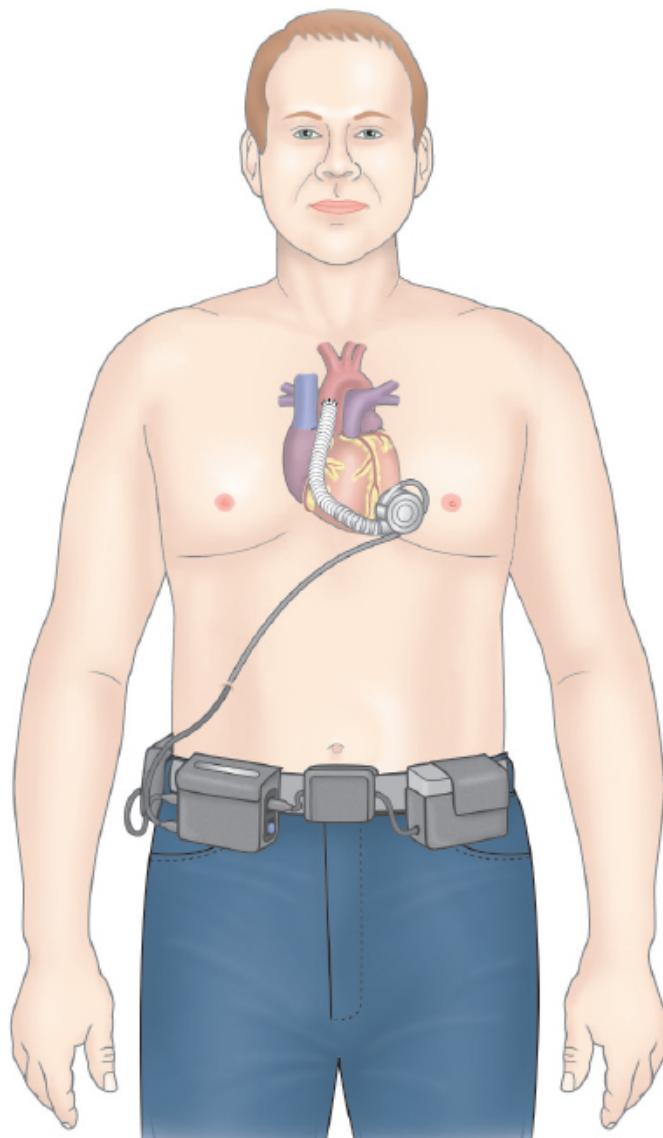
This was a descriptive correlational study with a cross-sectional design. It utilized a convenience sample consisting of 102 patients who had heart transplants and who engaged in outpatient follow up at the Sheba Medical Center in Israel. Participants were 76.5% male, 65.3% were born in Israel, and had a mean age of 56.66 y at the time of the study. The five-item BAASIS survey was used to assess adherence in the past 4 wks as it related to timing of medication intake, self-altering the prescribed dosage, and stopping or missing medication doses. Feelings of guilt and indebtedness were assessed with two survey questions via a Likert scale. Medical record review was also utilized. Data were evaluated using a variety of statistical analyses, which included *t*-tests, correlation analyses, and logistic regression analysis.

### Findings

Sixty-four percent of participants reported nonadherence with immunosuppression within the previous 4 wks. Age, time since transplant, and feelings of guilt were statistically associated with variance in adherence. An increase in age was associated with an increase in adherence, whereas an increase in time since transplant and feelings of guilt were associated with an increase in nonadherence.

### Nursing Implications

Nonadherence to immunosuppression is associated with an increase in morbidity and mortality among patients with heart transplants. Nurses should assess patients' adherence to immunosuppression and be aware of the various factors that may influence adherence. Understanding what can contribute to nonadherence, such as feelings of guilt, enables the nurse to attempt to alleviate those feelings and thereby improve patient adherence and transplant outcomes.



**Figure 24-10 •** Left ventricular assist device. Reproduced with permission of Medtronic.

VADs may be used as (1) a “bridge to recovery” for patients who require temporary assistance for reversible ventricular failure, (2) a “bridge to transplant” for patients with end-stage heart failure until a donor organ becomes available for transplant, and (3) “destination therapy” for patients with end-stage heart failure who are not candidates for or decline heart transplantation and have the VAD implanted for permanent use (Kittleson et al., 2017). As patients spend an increased length of time on the transplant list and more VADs are being implanted, destination therapy now accounts for 50% of patients on mechanical circulatory support (Han, Acker, & Atluri, 2018). Thus, the volume of patients with VADs in the community is rapidly expanding and it is important that first responders and other providers are

familiar with the basics of VAD management and equipment (Cook, Colvin, Francis, et al., 2017). In some instances, the patient may request deactivation of the VAD, which may cause ethical controversy ([Chart 24-3](#)).

**Chart 24-3**  **ETHICAL DILEMMA**

## Can an Adult with a Left Ventricular Assist Device (LVAD) Have the LVAD Deactivated?

### Case Scenario

You work on a cardiac intensive care unit (CICU) and are assigned to care for J.J., a 75-year-old man with an extensive cardiac history who received a left ventricular assist device (LVAD) 10 mo ago. When the device was implanted, J.J. knew the treatment was a destination therapy (i.e., a final treatment option) since he was not a candidate for a heart transplant. The goal was to improve his quality of life. However, this is now his 12th hospitalization since the LVAD was activated, and J.J. now questions the purported benefit of the LVAD. During morning rounds with the cardiologist, J.J. tells you, the cardiologist, his wife, and adult daughter that he feels that the quality of his life is poor and not going to improve. He pointedly asks the cardiologist to deactivate the device, saying that he realizes this will lead to his death. J.J. is mentally competent (i.e., possesses the mental capacity to understand the ramifications of his request). J.J.'s family, the cardiologist, and you are all stakeholders in this decision and each of you express reluctance at honoring his request.

### Discussion

Many ethical dilemmas are inherent in this scenario. Although the LVAD is internally implanted, the control switch to deactivate the device is external. Some bioethicists argue that since the device has an external component, deactivating the device is similar to withdrawing treatment, which is ethical. Others argue that it becomes an integrated body part once it is implanted; therefore, to remove it is consistent with euthanasia. There is an obligation to respect J.J.'s autonomy and self-determination, especially since he has the capacity to make these decisions (i.e., he is mentally competent). Yet, there is a "slippery slope" at play here in terms of the principle of nonmaleficence.

### Analysis

- Identify the ethical principles that are in conflict in this case (see [Chapter 1, Chart 1-7](#)). Which principle do you think should have preeminence as you work to resolve the conflicting emotions between J.J., his family, and the health care team?
- What arguments would you offer *in favor of* removal of the LVAD?
- What arguments would you offer *against* removal of the LVAD?
- Do you believe deactivation of an LVAD is the same as withdrawal of other life-sustaining treatments (e.g., extubation)? Do you believe deactivation of an LVAD constitutes euthanasia?
- Assume that J.J. is determined competent to make this decision and the deactivation is scheduled. Discuss the implications of this procedure on the stakeholders including you, the cardiologist, and the patient's wife and daughter. Do you have the right to refuse to

participate in the procedure? Identify what resources might be of help to you as you grapple with making the best decision for all stakeholders.

## References

- Shinall, M. C. (2018). The evolving moral landscape of palliative care. *Health Affairs*, 37(4), 670–673.
- Slavin, S. D., Allen, L. A., McIlvennan, C. K., et al. (2020). Left ventricular assist device withdrawal: Ethical, psychological, and logistical challenges. *Journal of Palliative Medicine*, 23(4), 456–458.

## Resources

See [Chapter 1, Chart 1-10](#) for Steps of an Ethical Analysis and Ethics Resources.

## Total Artificial Hearts

**Total artificial hearts** are designed to replace both ventricles. Only one TAH has been approved by the U.S. Food and Drug Administration (FDA) as a bridge to transplant, the SynCardia TAH, and it requires the removal of the patient's heart for implant. Although there has been some short-term success, the long-term results have been disappointing. Researchers hope to develop a device that can be permanently implanted and will eliminate the need for donated human heart transplantation for end-stage cardiac disease. The CARMMAT TAH is undergoing early clinical trials in Europe; it features biocompatible materials (Ewald, Milano, & Rogers, 2019).

Complications of VADs and TAHs include bleeding disorders, hemorrhage, thromboemboli, hemolysis, infection, kidney injury, right-sided heart failure, multisystem failure, and mechanical failure. Nursing care of patients with these mechanical assist devices focuses on assessment for and minimization of these complications as well as providing emotional support and education about the device and the underlying cardiac disease.

## NURSING PROCESS

### The Patient with Cardiomyopathy

#### Assessment

Nursing assessment for the patient with cardiomyopathy begins with a detailed history of the presenting signs and symptoms. The nurse identifies possible etiologic factors, such as heavy alcohol intake, recent illness or pregnancy, family history of sudden death or history of the disease in immediate family members. If the patient reports chest pain, a thorough review of the pain, including its precipitating factors, is warranted. The review of systems includes the presence of orthopnea, PND, and syncope or dyspnea with exertion. The number of pillows needed to sleep, usual weight, any weight change, and limitations on activities of daily living are assessed. The American College of Cardiology and American Heart Association Stages of Heart Failure classification is utilized to help identify disease progression, and the New York Heart Association Functional Classification for heart failure is determined based on the severity of the patient's symptoms (see [Chapter 25](#), Tables 25-1 and 25-2). The patient's usual diet is evaluated to determine the need to reduce sodium intake, optimize nutrition, or supplement with vitamins.

Because of the chronicity of cardiomyopathy, the nurse conducts a careful psychosocial history, exploring the impact of the disease on the patient's role within the family and community. Identification of perceived stressors helps the patient and the health care team to implement activities that relieve anxiety associated with changes in health status. Very early on, the patient's support systems are identified, and members are encouraged to become involved in the patient's care and therapeutic regimen. The assessment addresses the effect the diagnosis has had on the patient and members of their support system and the patient's emotional status. Findings from a recent meta-analysis suggest that 19% of patients with heart failure meet criteria for depressive disorders and 21.5% have significant symptoms of depression (Celano, Villegas, Albanese, et al., 2018). Various screening tools are available to assess symptoms associated with heart failure, including depression, though no one screening tool has emerged as the generally accepted standard.

The physical assessment focuses on signs and symptoms of heart failure. The baseline assessment includes key components such as:

- Vital signs
- Calculation of pulse pressure and identification of pulsus paradoxus
- Current weight and any weight gain or loss
- Detection by palpation of the point of maximal impulse, often shifted to the left

- Cardiac auscultation for a systolic murmur and S<sub>3</sub> and S<sub>4</sub> heart sounds
- Pulmonary auscultation for crackles
- Measurement of jugular vein distention
- Assessment of edema and its severity

## **Diagnosis**

### **NURSING DIAGNOSES**

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired cardiac output associated with structural disorders caused by cardiomyopathy or to arrhythmia from the disease process and medical treatments
- Risk for impaired cardiac function, ineffective tissue perfusion, and impaired peripheral tissue perfusion associated with decreased peripheral blood flow (resulting from impaired cardiac output)
- Impaired gas exchange associated with pulmonary congestion caused by myocardial failure (resulting from impaired cardiac output)
- Activity intolerance associated with impaired cardiac output or excessive fluid volume, or both
- Anxiety associated with the change in health status and in role functioning
- Powerlessness associated with disease process
- Able to perform self care associated with medication and diet therapies

### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Heart failure
- Ventricular arrhythmias
- Atrial arrhythmias
- Cardiac conduction defects
- Pulmonary or cerebral embolism
- Valvular dysfunction

These complications are discussed earlier in this chapter and in Chapters 22 and 25.

## **Planning and Goals**

The major goals for patients include improvement or maintenance of cardiac output, increased activity tolerance, reduction of anxiety, effective management of the self-care program, increased sense of power with decision making, and absence of complications.

## Nursing Interventions

### IMPROVING CARDIAC OUTPUT AND PERIPHERAL BLOOD FLOW

During a symptomatic episode, rest is indicated. Many patients with DCM find that sitting up with their legs down is more comfortable than lying down in a bed. This position is helpful in pooling venous blood in the periphery and reducing preload. Assessing the patient's oxygen saturation at rest and during activity may assist with determining a need for supplemental oxygen. Oxygen usually is given through a nasal cannula when indicated.

Ensuring that medications are taken as prescribed is important to preserving adequate cardiac output. The nurse may assist the patient with planning a schedule for taking medications and identifying methods to remember to follow it, such as associating the time to take a medication with an activity (e.g., eating a meal, brushing teeth) or obtaining a pillbox.

It is also important to ensure that the patient receives or chooses food selections that are appropriate for a low sodium diet. One way to monitor a patient's response to treatment is to determine the patient's weight every day and identify any significant change. Another indication of the effect of treatment involves assessment of shortness of breath after activity and comparison to before treatment, as well as a change in the number of pillows needed to comfortably sleep. Patients with low cardiac output may need assistance keeping warm and frequently changing position to stimulate circulation and reduce the possibility of skin breakdown.

### INCREASING ACTIVITY TOLERANCE AND IMPROVING GAS EXCHANGE

The nurse plans the patient's activities so that they occur in cycles, alternating rest with activity periods. This benefits the patient's physiologic status, and it helps educate the patient about the need for planned cycles of rest and activity. For example, after taking a bath or shower, the patient should plan to sit and read a newspaper or engage in other relaxing activities. Suggesting that the patient sit while chopping vegetables, drying their hair, or shaving helps the patient learn to balance rest with activity. The nurse also makes sure that the patient recognizes the symptoms indicating the need for rest and actions to take when the symptoms occur. Patients with HCM, ARVC/D, or RCM must avoid strenuous activity, isometric exercises, and competitive sports.

### REDUCING ANXIETY

Spiritual, psychological, and emotional support may be indicated for patients, families, and significant others. Interventions are directed toward eradicating or alleviating perceived stressors. Patients receive appropriate information about cardiomyopathy and self-management activities. It is important to provide an atmosphere in which patients feel free to verbalize concerns and receive assurance that their concerns are legitimate. If the

patient is awaiting transplantation or facing death, it is necessary to allow time to discuss these issues. Providing the patient with realistic hope helps reduce anxiety while they await a donor heart. The nurse helps the patient, family, and significant others with anticipatory grieving.

#### **DECREASING THE SENSE OF POWERLESSNESS**

Patients often go through a grieving process when cardiomyopathy is diagnosed. The patient is assisted in identifying the things in life that they have lost (e.g., foods that the patient enjoyed eating but are high in sodium, the ability to engage in an active lifestyle, the ability to play sports, the ability to lift grandchildren) and their emotional responses to the loss (e.g., anger, feelings of sadness). The nurse assists the patient in identifying the amount of control that they still have over life, such as making food choices, managing medications, and working with the patient's primary provider to achieve the best possible outcomes. A diary in which the patient records food selections and weight may help with understanding the relationship between sodium intake and weight gain and give patients a sense of control over their disease. Some patients can manage a self-titrating diuretic regimen in which they adjust the dose of diuretic to their symptoms.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**



**Educating Patients About Self-Care.** A key part of the plan of nursing care involves educating patients about the medication regimen, symptom monitoring, and symptom management ([Chart 24-4](#)). The nurse plays an integral role as the patient learns to balance lifestyle and work while accomplishing therapeutic activities. Helping patients cope with their disease status helps them adjust their lifestyles and implement a self-care program at home. Attainment of a goal, no matter how small, also promotes the patient's sense of well-being.

**Continuing and Transitional Care.** The nurse reinforces previous education and performs ongoing assessment of the patient's symptoms and progress. The nurse also assists the patient and family to adjust to lifestyle changes. Patients are taught to read nutrition labels, to maintain a record of daily weights and symptoms, and to organize daily activities to increase activity tolerance. In addition, the nurse assesses the patient's response to recommendations about diet and fluid intake and to the medication regimen and stresses the signs and symptoms that should be reported to the primary provider. Because of the risk of arrhythmia, it may be necessary to educate the patient's family about cardiopulmonary resuscitation and the use of an automated external defibrillator (see [Chapter 25](#)). Women are often advised to avoid pregnancy, but each case is assessed individually. The nurse assesses the psychosocial needs of the patient and family on an ongoing

basis. There may be concerns and fears about the prognosis, changes in lifestyle, effects of medications, and the possibility of others in the family having the same condition; these concerns often increase the patient's anxiety and interfere with effective coping strategies. Establishing trust is vital to the nurse's relationship with patients who are chronically ill and with these patients' families. This is particularly significant when the nurse is involved with a patient and family in discussions about end-of-life decisions. Patients who have significant symptoms of heart failure or other complications of cardiomyopathy may benefit from transitional or home care.

### Evaluation

Expected patient outcomes may include:

1. Maintains or improves cardiac function
  - a. Exhibits heart and respiratory rates within normal limits

Chart 24-4



### HOME CARE CHECKLIST

## The Patient with Cardiomyopathy

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of cardiomyopathy on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- Identify interventions and strategies (e.g., durable medical equipment, adaptive equipment) used in adapting to any permanent or temporary changes in structure or function.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Take or administer medications daily, exactly as prescribed.
- Monitor effects of medication such as changes in breathing and edema.
- Know signs and symptoms of arrhythmia formation (e.g., lightheadedness, dizziness, orthostatic hypotension, racing heartbeats, diaphoresis, confusion):
  - Identify how to initiate emergency response.
  - Demonstrate skill set in cardiopulmonary resuscitation.
  - Demonstrate use of an automated external defibrillator.
- Weigh self daily at the same time with same clothes.
- Restrict sodium intake to no more than 2 g/day:
  - Adapt diet by examining nutrition labels to check sodium content per serving.
  - Avoid canned or processed foods, eating fresh or frozen foods.
  - Consult the written diet plan and the list of permitted and restricted foods.
  - Avoid salt use.
  - Avoid excesses in eating and drinking.
- Participate in prescribed activity program:
  - Participate in a daily exercise program, with walking and other activities, provided they do not cause unusual fatigue or dyspnea.
  - Conserve energy by balancing activity with rest periods.
  - Avoid strenuous activity, isometric exercises, and competitive sports.
- Develop methods to manage and prevent stress:
  - Avoid tobacco.
  - Avoid alcohol.
  - Engage in social and diversional activities.
- Identify community resources for peer and caregiver/family support:

- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- State meeting locations and times.
- Report immediately to the primary provider or clinic any of the following:
  - Gain in weight of 2–3 lb (0.9–1.4 kg) in 1 d, or 5 lb (2.3 kg) in 1 wk
  - Unusual shortness of breath with activity or at rest
  - Increased swelling of ankles, feet, or abdomen
  - Persistent cough
  - Loss of appetite
  - Development of restless sleep; increase in number of pillows needed to sleep
  - Profound fatigue
- State how to reach primary provider with questions or complications.
- State time and date of follow-up appointments and diagnostic tests.
- Identify the need for health promotion, disease prevention, and screening activities.

ADL, activities of daily living; IADL, independent activities of daily living.

- b. Reports decreased dyspnea and increased comfort; maintains or improves gas exchange
  - c. Reports no weight gain; appropriate weight for height
  - d. Maintains or improves peripheral blood flow
2. Maintains or increases activity tolerance
    - a. Carries out activities of daily living (e.g., brushes teeth, feeds self)
    - b. Reports increased tolerance to activity
  3. Is less anxious
    - a. Discusses prognosis freely
    - b. Verbalizes fears and concerns
    - c. Participates in support groups if appropriate
    - d. Demonstrates appropriate coping mechanisms
  4. Decreases sense of powerlessness
    - a. Identifies emotional response to diagnosis
    - b. Discusses control that they have
  5. Effectively manages self-care program
    - a. Takes medications according to prescribed schedule
    - b. Modifies diet to accommodate sodium and fluid recommendations

- c. Modifies lifestyle to accommodate activity and rest behavior recommendations
- d. Identifies signs and symptoms to be reported to health care professionals

## INFECTIOUS DISEASES OF THE HEART

Any of the heart's three layers may be affected by an infectious process. Infections are named for the layer of the heart most involved in the infectious process: infective endocarditis (endocardium), myocarditis (myocardium), and pericarditis (pericardium) (see [Chapter 21, Fig. 21-1](#)). Rheumatic endocarditis is a unique infective endocarditis syndrome. Diagnosis of infection is made primarily on the basis of the patient's symptoms and echocardiography. Ideal management for all infectious diseases is prevention. IV antibiotics usually are necessary once an infection has developed in the heart.

### Rheumatic Endocarditis

Acute rheumatic fever, which occurs most often in school-age children, may develop after an episode of group A beta-hemolytic streptococcal pharyngitis ([Chart 24-5](#)). Patients with rheumatic fever may develop rheumatic heart disease as evidenced by a new heart murmur, cardiomegaly, pericarditis, and heart failure. Prompt and effective treatment of "strep" throat with antibiotics can prevent development of rheumatic fever. Streptococcus is spread by direct contact with oral or respiratory secretions. Although bacteria are the causative agents, malnutrition, overcrowding, poor hygiene, and lower socioeconomic status may predispose individuals to rheumatic fever. The incidence of rheumatic fever in developed countries has decreased and it is now primarily a disease of the developing world (Cannon, Roberts, Milne, et al., 2017). As noted earlier in the chapter, rheumatic heart disease may lead to mitral valve stenosis or regurgitation that remains an issue in adulthood. Further information about rheumatic fever and rheumatic endocarditis can be found in pediatric nursing books.

#### Chart 24-5

## Rheumatic Fever

Rheumatic fever is a preventable disease. Diagnosing and effectively treating streptococcal pharyngitis can prevent rheumatic fever and, therefore, rheumatic heart disease. Signs and symptoms of streptococcal pharyngitis include:

- Sore throat that can start very quickly
- Pain when swallowing
- Fever
- Red and swollen tonsils, sometimes with white patches or streaks of pus
- Petechiae (tiny, red spots) on the roof of the mouth (the soft or hard palate)
- Swollen lymph nodes in the front of the neck

If signs and symptoms of streptococcal pharyngitis are present, a rapid strep test is necessary to make an accurate diagnosis. If the test is negative but strep throat is still suspected, then a throat culture can be done. For adults, it is usually not necessary to do a throat culture following a negative rapid strep test. Adults are generally not at risk for getting rheumatic fever following a strep throat infection. All patients with a positive rapid strep test or throat cultures positive for streptococcal pharyngitis must adhere to the prescribed antibiotic treatment. Penicillin or amoxicillin are typical first-line antibiotics used. Completing the course of prescribed antibiotics minimizes the risk of developing rheumatic fever (and subsequent rheumatic heart disease).

Adapted from Centers for Disease Control and Prevention (CDC). (2018).

*Strep throat: All you need to know.* Retrieved on 12/3/2019 at:

[www.cdc.gov/groupastrep/diseases-public/strep-throat.html](http://www.cdc.gov/groupastrep/diseases-public/strep-throat.html)

## Infective Endocarditis

Infective endocarditis is a microbial infection of the endothelial surface of the heart. The disease is rare, but it has a high mortality rate; approximately 14% to 22% of patients die during their hospital stay, and up to 40% of patients die within 1 year of diagnosis (Kaura, Byrne, Fife, et al., 2017). It usually develops in older adults, or in people with prosthetic heart valves or cardiac devices. Staphylococcal endocarditis infections of valves in the right side of the heart are common among adults who use illicit IV drugs (Baddour, Wilson, Bayer, et al., 2015). Hospital-acquired infective endocarditis occurs most often in patients with debilitating disease or indwelling catheters and in patients who are receiving hemodialysis or prolonged IV fluid or antibiotic therapy ([Chart 24-6](#)).

## Pathophysiology

A deformity or injury of the endocardium leads to accumulation of fibrin and platelets (clot formation) on the endocardium. Infectious organisms, usually staphylococci or streptococci, invade the clot and endocardial lesion. Infection most frequently results in platelets, fibrin, blood cells, and microorganisms that cluster as vegetations on the endocardium. Vegetations may embolize to other vessels throughout the body. As the clot on the endocardium continues to expand, the infecting organism is covered by new clot and concealed from the body's normal defenses. Infection may erode through the endocardium into underlying structures (e.g., valve leaflets), causing tears or other deformities of valve leaflets, dehiscence of prosthetic valves, deformity of chordae tendineae, or mural abscesses.

### Chart 24-6 RISK FACTORS

#### Infective Endocarditis

- Prosthetic cardiac valves or prosthetic material used for cardiac valve repair
- Implanted cardiac devices (e.g., pacemaker, implanted cardioverter defibrillator)
- History of bacterial endocarditis (even without heart disease)
- Congenital heart disease:
  - Unrepaired cyanotic disease, including patients with palliative shunts and conduits
  - Repaired with prosthetic material or device either by surgery or catheter intervention during the first 6 mo after the procedure
  - Repaired with residual defects at the site or adjacent to the site of a prosthetic patch or device
- Cardiac transplant recipients with valvulopathy
- IV drug abuse
- Body piercing (especially oral, nasal, and nipple), branding, and tattooing
- Hemodialysis

Adapted from Nishimura, R. A., Otto, C. M., Bonow, R. O., et al. (2014). 2014 AHA/ACC guidelines for the management of patients with valvular heart disease. *Journal of the American College of Cardiology*, 63(22), e57–e185.

## Clinical Manifestations

Onset of infective endocarditis usually is insidious. Signs and symptoms develop from toxic effects of the infection, destruction of heart valves, and embolization of fragments of vegetative growths on the endocardium. Primary presenting symptoms of infective endocarditis are fever and a heart murmur. Fever may be intermittent or absent, especially in patients who are receiving antibiotics or corticosteroids, in older adults, and in those who have heart failure or kidney injury. A heart murmur may be absent initially but develops in 85% of patients (Karchmer, 2018). Murmurs that worsen over time may indicate progressive damage and extension of the infectious vegetation (Baddour et al., 2015).

In addition to fever and heart murmur, clusters of petechiae may be found on the body. Small, painful nodules (Osler nodes) may be present in pads of fingers or toes. Irregular, red or purple, painless flat macules (Janeway lesions) may be present on palms, fingers, hands, soles, and toes. Hemorrhages with pale centers (Roth spots) caused by emboli may be observed in fundi of the eyes. Splinter hemorrhages (i.e., reddish-brown lines and streaks) may be seen under the proximal half of fingernails and toenails. Petechiae may appear in conjunctiva and mucous membranes.

Systemic embolization occurs in 22% to 50% of patients. It can occur at any time and may even be a presenting symptom. Up to 65% of emboli target the central nervous system and the majority of those are to the middle cerebral artery, effectively causing an embolic stroke. Metastatic foci of infection can also occur from embolization; for example, patients may develop a splenic abscess that requires splenectomy (Baddour et al., 2015).

Heart failure is the most frequent complication of infective endocarditis and may result from perforation of a valve leaflet, rupture of chordae, blood flow obstruction due to vegetations, or intracardiac shunts from dehiscence of prosthetic valves. It indicates a poor prognosis with medical therapy alone and is an indication for surgery (Alpert & Klotz, 2017).

## Assessment and Diagnostic Findings

Although characteristics described previously may indicate infective endocarditis, signs and symptoms may indicate other diseases as well. Vague complaints of malaise, anorexia, weight loss, cough, and back and joint pain may be mistaken for influenza. Virulence of the causative organism usually correlates with the speed and degree of symptom development. A definitive diagnosis is made when a microorganism is found in two separate blood cultures and there is evidence of vegetation on imaging of the heart (e.g., echocardiogram). At least two sets of blood cultures (with each set including one aerobic and one anaerobic culture) drawn from different venipuncture sites

over a 24-hour period (each set at least 2 hours apart), should be obtained before administration of any antibiotics (Karchmer, 2018). Negative blood cultures do not definitely rule out infective endocarditis. Patients may have elevated white blood cell (WBC) counts. In addition, patients may be anemic, have a positive rheumatoid factor, and an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein.

Echocardiography may assist in diagnosis by demonstrating a mass on a valve, prosthetic valve, or supporting structures and by identifying vegetations, abscesses, new prosthetic valve dehiscence, or new regurgitation. An echocardiogram may reveal development of heart failure. TEE may provide additional data when transthoracic imaging is nondiagnostic; this method of echocardiography is superior in assessing vegetations and perivalvular complications (Karchmer, 2018).

## Prevention

Antibiotic prophylaxis had been traditionally recommended in patients at high risk (e.g., those with previous infective endocarditis, prosthetic heart valves, patients with heart transplant and valve regurgitation, some patients with congenital heart disease) before and sometimes after dental procedures that involved manipulation of gingival tissue or periapical area of teeth or perforation of oral mucosa. Antibiotic prophylaxis was also indicated for patients at high risk having procedures which involved the airway, and procedures which involved manipulation of infected tissue (e.g., wound débridement). The latest U.S. guidelines assert that prophylaxis in patients at high risk is reasonable but admit that the data are mixed regarding whether prophylaxis really decreases rates of infective endocarditis (Nishimura et al., 2017).

Patients at high risk should practice good oral hygiene. Poor dental hygiene can lead to bacteremia, particularly in the setting of a dental procedure. Severity of oral inflammation and infection is a significant factor in the incidence and degree of bacteremia. Regular professional oral care combined with personal oral care may reduce the risk of bacteremia. Recommended ongoing personal oral care includes using a manual or electronic toothbrush, dental floss, and other plaque removing devices (Nishimura et al., 2017).

Any patient at risk with a fever of more than 7 days' duration should report that finding to a primary provider; patients should not self-medicate with antibiotics or stop taking them before the prescribed dosage has been completed (Nishimura et al., 2017).

Increased vigilance is also required in patients with IV catheters and during invasive procedures. To minimize the risk of infection, nurses must ensure meticulous hand hygiene, site preparation, and aseptic technique during insertion and maintenance procedures. All catheters, tubes, drains, and other

devices are removed as soon as they are no longer needed or no longer function.

## **Medical Management**

The objective of treatment is to eradicate invading organisms through adequate doses of an appropriate antibiotic. Antibiotic therapy usually is given intravenously for 2 to 6 weeks. Parenteral therapy is given in doses that produce a high serum concentration for a significant period to ensure eradication of the dormant bacteria within dense vegetations. This therapy is often delivered in the patient's home and is monitored by a home health nurse. Serum levels of the antibiotic and blood cultures are monitored to gauge effectiveness of therapy. If there is insufficient bactericidal activity, increased dosages of the antibiotic are prescribed or a different antibiotic is used. After adequate antibiotic therapy is initiated, the infective organism is usually eliminated. The patient should begin to feel better, regain an appetite, and have less fatigue. During this time, patients require psychosocial support because although they feel well, they may find themselves confined to the hospital or home with restrictive IV therapy.

## **Surgical Management**

Surgical intervention may be required if the patient develops heart failure or an intracardiac abscess, or the patient has recurrent systemic embolizations, or the infection does not respond to medications. Surgical interventions include valve repair and replacement, débridement of vegetations, débridement and closure of an abscess, and closure of a fistula. Surgical valve replacement greatly improves the prognosis for patients with severe symptoms from damaged heart valves. Most patients who have prosthetic valve endocarditis require repeat valve replacement (Alpert & Klotz, 2017).

## **Nursing Management**

The nurse monitors the patient's temperature at regular intervals, because the course of fever is one indication of treatment effectiveness. However, febrile reactions also may occur as a result of medication. The nurse administers antibiotic, antifungal, or antiviral medication as prescribed and educates the patient to take them as prescribed. Timing of antimicrobial medication administration is critical to maintain therapeutic drug levels. Fever often increases fatigue; rest periods should be planned and activities spaced to provide rest between activities. Good infection control and prevention practices include appropriate hand hygiene by both patients and caregivers. Nonsteroidal anti-inflammatory drugs (NSAIDs) may be prescribed as

antipyretics or to decrease the discomfort of fever. Patients may be more comfortable with a light layer of linens and exposure of their skin to air. They may be cooled with a fan, tepid water baths, or cloth compresses; if shivering or piloerection occurs, these interventions should be discontinued due to increased oxygen consumption and potential to further increase of body temperature.

Heart sounds are assessed. A new or worsening murmur may indicate dehiscence of a prosthetic valve, rupture of an abscess, or injury to valve leaflets or chordae tendineae (Baddour et al., 2015). The nurse monitors for signs and symptoms of systemic embolization, or, for patients with right-sided heart endocarditis, for signs and symptoms of pulmonary infarction and infiltrates. In addition, the nurse assesses signs and symptoms of organ damage such as stroke, meningitis, heart failure, myocardial infarction, glomerulonephritis, and splenomegaly.

Patient care is directed toward management of infection. Long-term IV antimicrobial therapy often is necessary; therefore, many patients have peripherally inserted central catheters or other long-term IV access. All invasive lines and wounds must be assessed daily for redness, tenderness, warmth, swelling, drainage, or other signs of infection. The patient and family are educated about activity restrictions, medications, and signs and symptoms of infection. Patients with infective endocarditis are at high risk for another episode of infective endocarditis. If the patient has undergone surgical treatment, the nurse provides postoperative care and instructions (see Chapters 16 and 23).

As appropriate, the home health nurse supervises and monitors IV antibiotic therapy delivered in the home setting and educates the patient and family about prevention and health promotion. The nurse provides the patient and family with emotional support and facilitates coping strategies during the prolonged course of infection and antibiotic treatment.

## Myocarditis

Myocarditis, an inflammatory process involving the myocardium, can cause heart dilation, thrombi on the heart wall (mural thrombi), infiltration of circulating blood cells around the coronary vessels and between the muscle fibers, and degeneration of the muscle fibers themselves. Mortality varies with the severity of symptoms. Most patients with mild symptoms recover completely; however, some patients develop cardiomyopathy and heart failure.

## Pathophysiology

Myocarditis usually results from an infectious source, be it viral (e.g., coxsackieviruses A and B, human immune deficiency virus, influenza A), bacterial, rickettsial, fungal, parasitic, metazoal, protozoal (e.g., Chagas disease), or spirochetal. It also may be immune related, occurring after acute systemic infections such as rheumatic fever, or it may be related to an autoimmune disorder. It may also result from an inflammatory reaction to toxins such as pharmacologic agents used in the treatment of other diseases (Arbustini, Agozzino, Favalli, et al., 2017). It may begin in one small area of the myocardium and then spread throughout the myocardium. The degree of myocardial inflammation and necrosis determines the degree of interstitial collagen and elastin destruction. The greater the destruction, the greater is the hemodynamic effect and resulting signs and symptoms. It is thought that DCM and HCM are latent manifestations of myocarditis.

## Clinical Manifestations

The symptoms of acute myocarditis depend on the type of infection, the degree of myocardial damage, and the capacity of the myocardium to recover. Patients may be asymptomatic, with an infection that resolves on its own. However, they may develop mild to moderate symptoms and seek medical attention, often reporting fatigue and dyspnea, syncope, palpitations, and occasional discomfort in the chest and upper abdomen. The most common symptoms are flulike. Patients may also sustain sudden cardiac death or quickly develop severe congestive heart failure in fulminant myocarditis.

## Assessment and Diagnostic Findings

Assessment of the patient may reveal no detectable abnormalities; as a result, the illness can go undiagnosed. Patients may be tachycardic or may report chest pain. An endomyocardial biopsy can provide the definitive diagnosis (Arbustini et al., 2017), but cardiac MRI is being used more often as a diagnostic tool because of its noninvasive approach (Lakdawala, Stevenson, & Loscalzo, 2018). With contrast, cardiac MRI may be diagnostic and can guide clinicians to sites for endomyocardial biopsies, which may be also indicated to find an organism or its genome, an immune process, or a radiation reaction causing the myocarditis. Patients without any abnormal heart structure (at least initially) may suddenly develop arrhythmias or ST-T-wave changes. If the patient has structural heart abnormalities (e.g., systolic dysfunction), a clinical assessment may disclose cardiac enlargement, faint heart sounds (especially S<sub>1</sub>), pericardial friction rub, a gallop rhythm, or a systolic murmur. The WBC count, C-reactive protein, leukocyte count, and ESR may be elevated.

## Medical Management

Patients are given specific treatment for the underlying cause if it is known (e.g., penicillin for hemolytic streptococci) and are placed on bed rest to decrease cardiac workload. Bed rest also helps decrease myocardial damage and the complications of myocarditis. In young patients with myocarditis, activities, especially athletics, should be limited for a 6-month period or at least until heart size and function have returned to normal. Physical activity is increased slowly, and the patient is instructed to report any symptoms that occur with increasing activity, such as a rapidly beating heart. If heart failure or arrhythmia develops, management is essentially the same as for all causes of heart failure and arrhythmias (see Chapters 25 and 22, respectively). Although they are known for their anti-inflammatory effects, NSAIDs should not be used for pain control; they have been implicated in increased cardiac injury and viral replication in animal studies (Lakdawala et al., 2018).

## Nursing Management

The nurse assesses for resolution of tachycardia, fever, and any other clinical manifestations. The cardiovascular assessment focuses on signs and symptoms of heart failure and arrhythmias. Patients with arrhythmias should have continuous cardiac monitoring with personnel and equipment readily available to treat life-threatening arrhythmias.



### Quality and Safety Nursing Alert

*Patients with myocarditis are sensitive to digitalis. Nurses must closely monitor these patients for digitalis toxicity, which is evidenced by a new onset of arrhythmia, anorexia, nausea, vomiting, headache, and malaise. The primary provider should be notified immediately if this is suspected.*

Anti-embolism stockings and passive and active exercises should be used because embolization from venous thrombosis and mural thrombi can occur, especially in patients on bed rest. In some patients, pharmacologic prophylaxis may also be indicated (see [Chapter 26](#)).

## Pericarditis

Pericarditis refers to an inflammation of the pericardium, which is the membranous sac enveloping the heart. It accounts for 5% of emergency room visits for chest pain (Adler, Charron, Imazio, et al., 2015; Imazio, Gaita, &

LeWinter, 2015). Classification of pericarditis may be acute, chronic, or recurrent. The etiology of pericarditis can be infectious or noninfectious (Imazio & Gaita, 2015). For example, pericarditis may occur after pericardectomy (opening of the pericardium) following cardiac surgery. Pericarditis also may occur 10 days to 2 months after acute myocardial infarction (Dressler syndrome).

## Pathophysiology

Causes underlying or associated with pericarditis are listed in [Chart 24-7](#). The inflammatory process of pericarditis may lead to an accumulation of fluid in the pericardial sac (pericardial effusion) and increased pressure on the heart, leading to cardiac tamponade (see [Chapter 25](#)). Frequent or prolonged episodes of pericarditis also may lead to thickening and decreased elasticity of the pericardium, or scarring may fuse the visceral and parietal pericardium. These conditions restrict the heart's ability to fill with blood (constrictive pericarditis). The pericardium may become calcified, further restricting ventricular expansion during ventricular filling (diastole). With less filling, the ventricles pump less blood, leading to decreased cardiac output and signs and symptoms of heart failure. Restricted diastolic filling may result in increased systemic venous pressure, causing peripheral edema and hepatic failure.

## Clinical Manifestations

Pericarditis may be asymptomatic. The most characteristic symptom of pericarditis is chest pain, although pain also may be located beneath the clavicle, in the neck, or in the left trapezius (scapula) region. Pain or discomfort usually remains fairly constant, but it may worsen with deep inspiration and when lying down or turning. The most characteristic clinical manifestation of pericarditis is a creaky or scratchy friction rub heard most clearly at the left lower sternal border. Other signs may include a mild fever, increased WBC count, anemia, and an elevated ESR or C-reactive protein level. Patients may have a nonproductive cough or hiccup. Dyspnea, as well as respiratory splinting because of pain upon inspiration, and other signs and symptoms of heart failure may occur as a result of pericardial compression due to constrictive pericarditis or cardiac tamponade. The heart rate may increase to maintain cardiac output.

### Chart 24-7

## Causes of Pericarditis

- Idiopathic or nonspecific causes
- Infection:
  - Viral: most commonly infectious cause (e.g., enteroviruses, herpes viruses, adenoviruses, parvoviruses)
  - Bacterial: rare (but if bacterial cause, most commonly *Mycobacterium tuberculosis* is implicated)
  - Fungal: rare (e.g., *Histoplasma*, *Aspergillus*, *Candida*)
  - Parasitic: rare (e.g., *Echinococcus*, *Toxoplasma*)
- Autoimmune disorders (e.g., systemic lupus erythematosus, rheumatic fever, rheumatoid arthritis, polyarteritis, sarcoidosis, scleroderma)
- Immune-mediated drug reactions:
  - Lupuslike reaction
  - Antineoplastic drugs
  - Hypersensitivity eosinophilia
- Disorders of adjacent structures (e.g., myocardial infarction, dissecting aneurysm, pneumonia)
- Neoplastic disease:
  - Metastasis from lung or breast cancer
  - Primary neoplasms (e.g., mesothelioma)
- Radiation therapy of chest and upper torso (peak occurrence 5–9 mo after treatment)
- Intentional or unintentional chest trauma (e.g., chest injury, cardiac surgery, cardiac catheterization, implantation of pacemaker, or implantation of a cardiac implantable electronic device)
- Metabolic:
  - Uremia
  - Anorexia
  - Myxedema

Adapted from Miranda, W. R., Imazio, M., Greason, K. L., et al. (2017). Pericardial diseases. In V. Fuster, R. A. Harrington, J. Narula, et al. (Eds.). *Hurst's the heart* (14th ed.). New York: McGraw-Hill.

## Assessment and Diagnostic Findings

The diagnosis most often is made on the basis of history, signs, and symptoms. An echocardiogram may detect inflammation, pericardial effusion or tamponade, and heart failure. It may help confirm the diagnosis and may be used to guide pericardiocentesis (needle or catheter drainage of the pericardium). TEE may be useful in diagnosis but may underestimate the extent of pericardial effusions. CT imaging may be the best diagnostic tool for determining size, shape, and location of pericardial effusions and may be used

to guide pericardiocentesis. Cardiac MRI may assist with detection of inflammation and adhesions. Occasionally, a video-assisted pericardioscope-guided biopsy of the pericardium or epicardium is performed to obtain tissue samples for culture and microscopic examination. Because the pericardial sac surrounds the heart, a 12-lead ECG may show concave ST elevations in many, if not all, leads (with no reciprocal changes) and may show depressed PR segments or atrial arrhythmias (Imazio & Gaita, 2015).

## Medical Management

Objectives of pericarditis management are to determine the cause, administer therapy for treatment and symptom relief, and detect signs and symptoms of cardiac tamponade. When cardiac output is impaired, the patient is placed on bed rest until fever, chest pain, and friction rub have subsided.

Analgesic medications and NSAIDs such as aspirin, indomethacin, or ibuprofen may be prescribed for pain relief during the acute phase. Corticosteroids (e.g., prednisone) can be used as an alternative when NSAIDs are contraindicated (e.g., kidney disease). Colchicine may be prescribed if the pericarditis is severe as an additive therapy to NSAIDs (Adler et al., 2015).

Pericardiocentesis, a procedure in which some pericardial fluid is removed, rarely is necessary. It may be performed to assist in identification of the cause or relieve symptoms, especially if there are signs and symptoms of heart failure or tamponade. Pericardial fluid is cultured if bacterial, tubercular, or fungal disease is suspected; a sample is sent for cytology if neoplastic disease is suspected. A pericardial window, a small opening made in the pericardium, may be performed to allow continuous drainage into the chest cavity. Surgical removal of tough encasing pericardium (pericardectomy) may be necessary to release both ventricles from constrictive and restrictive inflammation and scarring.

## Nursing Management

Patients with acute pericarditis require pain management with antispasmodic agents, assistance with positioning, and psychological support. Patients with chest pain often benefit from education and reassurance that the pain is not due to a heart attack. Pain may be relieved with a forward-leaning or sitting position. To minimize complications, the nurse helps the patient with activity restrictions until pain and fever subside. As the patient's condition improves, the nurse encourages gradual increases of activity. However, if pain, fever, or friction rub recurs, activity restrictions must be resumed. The nurse educates the patient and family about a healthy lifestyle to enhance the patient's immune system.

Nurses caring for patients with pericarditis must be alert to signs and symptoms of cardiac tamponade (see [Chapter 25](#)). The nurse monitors the patient for heart failure. Patients with hemodynamic instability or pulmonary congestion are treated as if they had heart failure (see [Chapter 25](#)).

## NURSING PROCESS

### The Patient with Pericarditis

#### Assessment

The primary symptom of pericarditis is pain, which is assessed by evaluating the patient in various positions. The nurse tries to identify whether pain is influenced by respiratory movements, while holding an inhaled breath or holding an exhaled breath; by flexion, extension, or rotation of the spine, including the neck; by movements of shoulders and arms; by coughing; or by swallowing. Recognizing events that precipitate or intensify pain may help establish a diagnosis and differentiate pain of pericarditis from pain of myocardial infarction.

When pericardial surfaces lose their lubricating fluid because of inflammation, a pericardial friction rub occurs. The rub is audible on auscultation and is synchronous with the heartbeat. However, it may be elusive and difficult to detect.



#### Quality and Safety Nursing Alert

*A pericardial friction rub is diagnostic of pericarditis. It is a creaky or scratchy sound and is louder at the end of exhalation. Nurses should monitor for pericardial friction rub by placing the diaphragm of the stethoscope tightly against the patient's thorax and auscultating the left sternal edge in the fourth intercostal space, which is the site where the pericardium comes into closest contact with the left chest wall. The rub may be heard best when a patient is sitting and leaning forward.*

If there is difficulty in distinguishing a pericardial friction rub from a pleural friction rub, the patient is asked to hold their breath; a pericardial friction rub will continue to be heard.

The patient's temperature is monitored frequently. Pericarditis may cause an abrupt onset of fever in a patient who has been afebrile.

#### Diagnosis

#### NURSING DIAGNOSES

Based on the assessment data, the major nursing diagnoses may be:

- Acute pain associated with inflammation of the pericardium
- Lack of knowledge of diagnosis and therapeutic self-care management

#### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Pericardial effusion
- Cardiac tamponade

### Planning and Goals

The patient's major goals may include relief of pain and absence of complications.

### Nursing Interventions

#### RELIEVING PAIN

Relief of pain is achieved by rest. Because sitting upright and leaning forward is the posture that tends to relieve pain, chair rest may be more comfortable. The nurse instructs the patient to restrict activity until pain subsides. As chest pain and friction rub abate, activities of daily living may be resumed gradually. If the patient is taking antispasmodic agents, antibiotics, or corticosteroids for pericarditis, responses to these medications are monitored and recorded. Patients taking NSAIDs or colchicine are assessed for gastrointestinal adverse effects. If chest pain and friction rub recur, bed rest or chair rest is resumed.

#### ENHANCING KNOWLEDGE ABOUT PERICARDITIS AND SELF-CARE MANAGEMENT

There are a multitude of possible causes for pericarditis (see [Chart 24-7](#)). Some of these are life-threatening (e.g., metastatic cancers) while others are not; regardless, the patient is likely to be fearful and anxious because they are experiencing chest pain. Educating the patient about the cause of the pericarditis, and medications prescribed to treat it (e.g., NSAIDs), and methods to improve breathing patterns and alleviate pain (e.g., sitting up and leaning forward upon a pillow) can allay anxieties. The patient should verbalize when it is appropriate to follow up with the primary provider (i.e., for routine follow ups, with relapse of symptoms, with symptoms suggestive of cardiac tamponade, including lightheadedness, orthostasis and tachycardia [see following]).

#### MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Abnormal accumulation of fluid between the pericardial linings (i.e., in the pericardial sac) is called *pericardial effusion*. Most patients have no effects or symptoms. However, enough fluid can accumulate to constrict the myocardium, impairing ventricular filling and the myocardium's ability to pump, a condition known as *cardiac tamponade* (discussed below) (Imazio et al., 2015). Failure to identify and treat this problem can lead to death.

Signs and symptoms of cardiac tamponade may begin with the patient reporting shortness of breath, chest tightness, or dizziness. The nurse may observe that the patient is becoming progressively more restless. Assessment of blood pressure may reveal a decrease of 10 mm Hg or more

in systolic blood pressure during inspiration (pulsus paradoxus). Usually, the systolic pressure decreases and the diastolic pressure remains stable; hence, the pulse pressure narrows. The patient usually has tachycardia, and ECG voltage may be decreased or QRS complexes may alternate in height (electrical alternans). Heart sounds may progress from distant to imperceptible. Blood continues to return to the heart from the periphery but cannot flow into the heart to be pumped back into the circulation. The patient develops jugular vein distention and other signs of rising central venous pressure. The Beck triad (hypotension, muffled heart sounds, and an elevated jugular venous pressure) is a useful diagnostic parameter of severe tamponade.

In such situations, the nurse notifies the primary provider immediately and prepares to assist with diagnostic echocardiography and pericardiocentesis (see [Chapter 25](#)). The nurse stays with the patient and continues to assess and record signs and symptoms while intervening to decrease patient anxiety.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**

Because patients, their family members, and health care providers tend to focus on the most obvious needs and issues related to pericarditis, the nurse reminds them about the importance of continuing health promotion and screening practices. The nurse educates patients who have not been involved in these practices in the past about their importance and refers them to appropriate health care providers.

#### **Evaluation**

Expected patient outcomes may include:

1. Freedom from pain
  - a. Performs activities of daily living without pain, fatigue, or shortness of breath
  - b. Temperature returns to normal range
  - c. Exhibits no pericardial friction rub
2. Effectively manages self-care
  - a. Identifies cause(s) of pericarditis and rationale for prescribed therapeutic regimen
  - b. Follows-up with primary provider for ongoing appointments and as needed
3. Absence of complications
  - a. Sustains blood pressure in normal range
  - b. Heart sounds strong and can be auscultated
  - c. Absence of jugular vein distention

## CRITICAL THINKING EXERCISES

**1 -ebp-** A 67-year-old woman presents to the emergency department (ED) where you work as a staff nurse with reports of fevers, malaise, and painful nodules on the pads of her fingers. Upon review of her medical history, the patient reports an aortic valve replacement (prosthetic) as a child, but no other significant medical or surgical history. Based on the history and physical examination findings, the primary provider thinks that the patient may have infective endocarditis and prescribes antibiotics. What should you ensure happens before the antibiotics are given? What are the care priorities for this patient?

**2 -ipc-** A 57-year-old man presents to the cardiology clinic where you work with a complaint of ankle swelling and trouble breathing when he walks more than 20 feet. The cardiologist does an echocardiogram and diagnoses the patient with cardiomyopathy. The patient expresses anxiety about what this means. Based on what you know about cardiomyopathy, what would you focus on when educating this patient? What are the key roles of other members of the health care team in appropriately assisting the patient in managing a new diagnosis of cardiomyopathy?

**3 -pq-** A 47-year-old woman with HCM presents to the ED where you work after fainting. It is the middle of summer and the primary provider thinks the patient was dehydrated. Explain the pathophysiology of why dehydration in a patient with HCM might precipitate a syncopal episode. Describe your prioritized focused assessment of this patient in the ED.

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

American Heart Association, National Center, [www.heart.org](http://www.heart.org)

Cardiomyopathy UK, [www.cardiomyopathy.org](http://www.cardiomyopathy.org)

MyLVAD, [www.mylvad.com](http://www.mylvad.com)

# 25 Management of Patients with Complications from Heart Disease

## LEARNING OUTCOMES

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

1. Recognize the etiology, pathophysiology, and clinical manifestations of the different classifications of heart failure.
2. Describe the medical management, including recommended pharmacologic treatments, for patients with heart failure.
3. Use the nursing process as a framework for care of the patient with heart failure.
4. Identify additional heart disease disorders and medical and nursing management of patients with complications from heart disease.

## NURSING CONCEPT

Perfusion

## GLOSSARY

**anuria:** urine output of less than 50 mL/24 h

**ascites:** an accumulation of serous fluid in the peritoneal cavity

**cardiac resynchronization therapy (CRT):** a treatment for heart failure in which a device paces both ventricles to synchronize contractions

**congestive heart failure (CHF):** a fluid overload condition (congestion) associated with heart failure

**diastolic heart failure:** the inability of the left ventricle of the heart to fill and pump sufficiently; term used to define a type of heart failure (*synonym:* Heart Failure with preserved Ejection Fraction [HFpEF])

**ejection fraction (EF):** percentage of blood volume in the ventricles at the end of diastole that is ejected during systole; a measurement of contractility

**heart failure (HF):** a clinical syndrome resulting from structural or functional cardiac disorders that impair the ability of a ventricle to fill or eject blood

**Heart Failure with midrange Ejection Fraction (HFmrEF):** clinical heart failure syndrome with left ventricular ejection fraction that is 40% to 49%

**Heart Failure with preserved Ejection Fraction (HFpEF):** clinical heart failure syndrome with left ventricular ejection fraction greater than or equal to 50% (*synonym:* diastolic heart failure)

**Heart Failure with reduced Ejection Fraction (HFrEF):** clinical heart failure syndrome with left ventricular ejection fraction less than or equal to 40% (*synonym:* systolic heart failure)

**left-sided heart failure:** inability of the left ventricle to fill or eject sufficient blood into the systemic circulation (*synonym:* left ventricular failure)

**oliguria:** diminished urine output; less than 0.5 mL/kg/h over at least 6 hours, or less than 400 mL in 24 hours

**orthopnea:** shortness of breath when lying flat

**paroxysmal nocturnal dyspnea (PND):** shortness of breath that occurs suddenly during sleep

**pericardiocentesis:** procedure that involves aspiration of fluid from the pericardial sac

**pericardiotomy:** surgically created opening of the pericardium

**pulmonary edema:** pathologic accumulation of fluid in the interstitial spaces and alveoli of the lungs causing severe respiratory distress

**pulseless electrical activity (PEA):** condition in which electrical activity is present on an electrocardiogram, but there is not a physiologically adequate pulse or blood pressure

**pulsus paradoxus:** systolic blood pressure that is more than 10 mm Hg lower during inhalation than during exhalation; difference is normally less than 10 mm Hg

**right-sided heart failure:** inability of the right ventricle to fill or eject sufficient blood into the pulmonary circulation (*synonym:* right ventricular failure)

**systolic heart failure:** inability of the heart to pump sufficiently because of an alteration in the ability of the heart to contract; term used to describe a type of heart failure (*synonym:* Heart Failure with reduced Ejection Fraction [HFrEF])

Cardiovascular disease is the leading cause of death in the United States (Centers for Disease Control and Prevention [CDC], 2017). Because of advancements in diagnostic and screening procedures, greater recognition of the importance of diligent self-care practices, and new discoveries in pharmacotherapies, it is now possible for a person diagnosed with heart disease to continue to live with a high quality of life years after being diagnosed. Despite this progress, heart disease remains a chronic and often progressive condition, associated with serious comorbidities, such as heart failure (Benjamin, Muntner, Alonso, et al., 2019). This chapter presents the complications most often associated with heart disease, including the medical management and nursing processes for managing patients with complications of cardiovascular disease.

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## HEART FAILURE

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**Heart failure (HF)** is a clinical syndrome resulting from structural or functional cardiac disorders so that the heart is unable to pump enough blood to meet the body's metabolic demands or needs (American Heart Association [AHA], 2019a). The term *heart failure* indicates myocardial disease in which impaired contraction of the heart (systolic dysfunction) or filling of the heart (diastolic dysfunction) may cause pulmonary or systemic congestion. Some cases of HF are reversible, depending on the cause. Most often, HF is a chronic, progressive condition that is managed with lifestyle changes and medications to prevent episodes of acute decompensated heart failure. These episodes are characterized by increased symptoms of respiratory distress, decreased cardiac output (CO), and poor perfusion. These episodes are also associated with increased hospitalizations, increased health care costs, and decreased quality of life (Benjamin et al., 2019).

Approximately six million people in the United States have HF, and 870,000 new cases are diagnosed each year (AHA, 2019a). As more people

live longer with chronic heart diseases, HF has become an epidemic that challenges the country's health care resources. HF is the most common reason for hospitalization of people older than 65 years and is the second most common reason for visits to a provider's office. Emergency department (ED) visits and hospital readmissions for this disorder are very common, despite efforts to prevent rehospitalizations. Over 20% of patients discharged after treatment for HF are readmitted to the hospital within 30 days, and nearly 50% are readmitted to the hospital within 6 months (O'Connor, 2017). The estimated economic burden caused by HF in the United States is more than \$30 billion annually in direct and indirect costs and is expected to continue to increase over time (CDC, 2017).

HF is more prevalent among African Americans and Hispanics than among Caucasians. The risk for having HF increases with advancing age. For adults over 60 years of age, HF is more prevalent among men than women (Benjamin et al., 2019). As typical for other major cardiovascular diseases and disorders, cigarette smoking, obesity, poorly managed diabetes, and metabolic syndrome are all risks for HF (Benjamin et al., 2019). The onset of HF is typically a morbid consequence of another disease or disorder, including coronary artery disease (CAD), hypertension, cardiomyopathy, valvular disorders, and renal dysfunction with volume overload (McCance, Huether, Brashers, et al., 2019).

Atherosclerosis of the coronary arteries is a primary cause of HF, and CAD is found in the majority of patients with HF. Ischemia causes myocardial dysfunction because it deprives heart cells of oxygen and causes cellular damage. Myocardial infarction (MI) causes focal heart muscle necrosis, the death of myocardial cells, and a loss of contractility; the extent of the infarction correlates with the severity of HF. Revascularization of the coronary artery by a percutaneous coronary intervention (PCI) or by coronary artery bypass surgery (coronary artery bypass graft [CABG]) may improve myocardial oxygenation and ventricular function and prevent more extensive myocardial necrosis that can lead to HF (see Chapter 23).

Systemic or pulmonary hypertension increases afterload (resistance to ejection), increasing the cardiac workload and leading to the hypertrophy of myocardial muscle fibers. This can be considered a compensatory mechanism because it initially increases contractility. However, sustained hypertension eventually leads to changes that impair the heart's ability to fill properly during diastole, and the hypertrophied ventricles may dilate and fail (Norris, 2019; Yancy, Jessup, Bozkurt, et al., 2017).

Cardiomyopathy is a disease of the myocardium. The various types of cardiomyopathy lead to HF and arrhythmias. Dilated cardiomyopathy (DCM), the most common type of cardiomyopathy, causes diffuse myocyte necrosis and fibrosis, and commonly leads to progressive HF (Norris, 2019). DCM can be idiopathic (unknown cause), or it can result from an inflammatory process, such as myocarditis, or from a cytotoxic agent, such as alcohol or certain

antineoplastic drugs. Usually, HF due to cardiomyopathy is chronic and progressive. However, cardiomyopathy and HF may resolve following removal of the causative agent. Genetic testing may be recommended for idiopathic cardiomyopathy (van der Meer, Gaggin, & Dec, 2019) (see Chapter 24).

Valvular heart disease is also a cause of HF. The valves ensure that blood flows in one direction. With valvular dysfunction, it becomes increasingly difficult for blood to move forward, increasing pressure within the heart and increasing cardiac workload, leading to HF (see Chapter 24).

Several systemic conditions, including progressive kidney failure, contribute to the development and severity of HF. Nearly 30% of patients with chronic HF also have chronic kidney disease (Benjamin et al., 2019). In addition, cardiac arrhythmias such as atrial fibrillation may either cause or result from HF; in both instances, the altered electrical stimulation impairs myocardial contraction and decreases the overall efficiency of myocardial function. Other factors, such as hypoxia, acidosis, and electrolyte abnormalities, can worsen myocardial function (Yancy et al., 2017).

## Pathophysiology

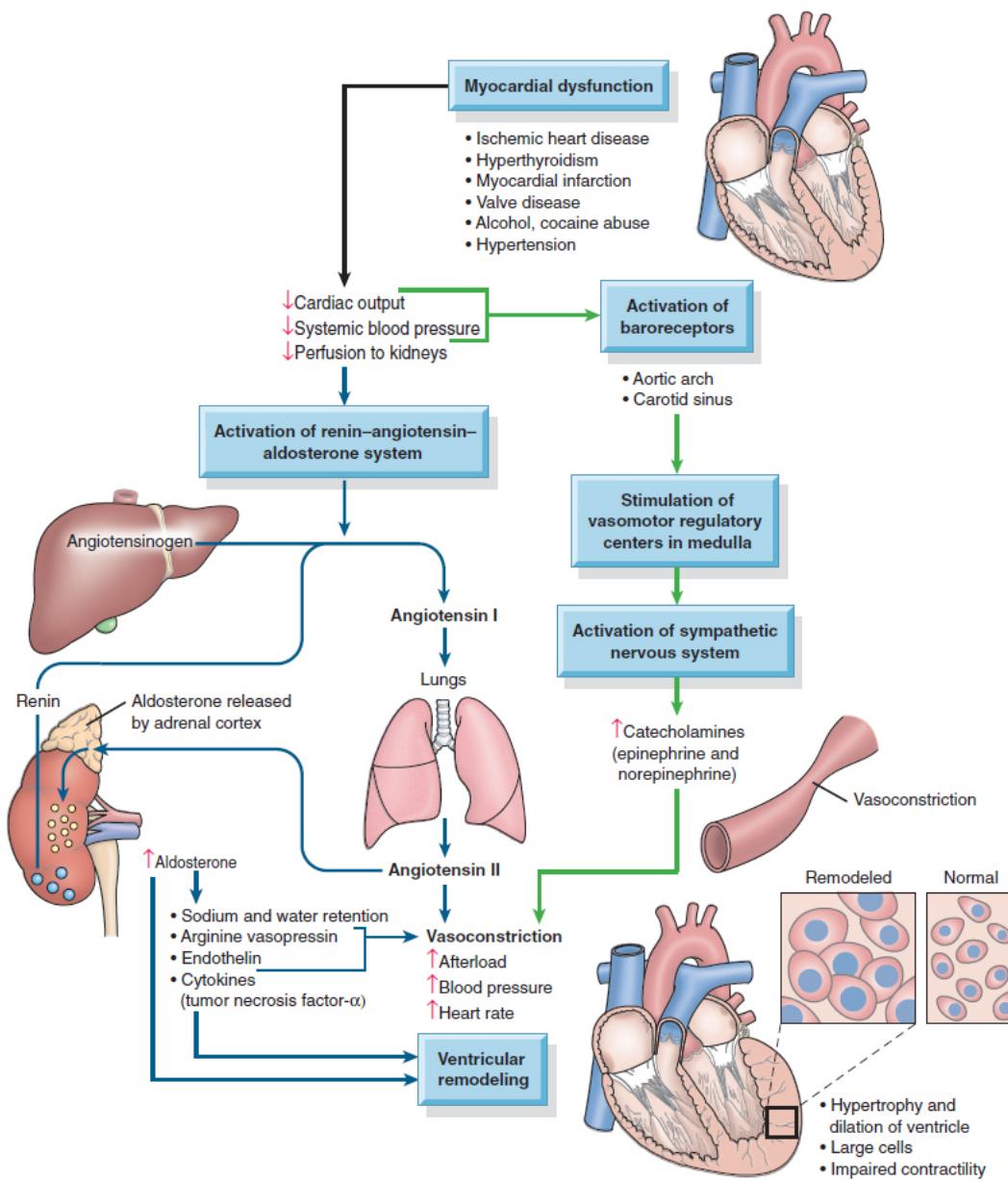
Regardless of the etiology, the pathophysiology of HF results in similar changes and clinical manifestations. Significant myocardial dysfunction usually occurs before the patient experiences signs and symptoms of HF such as shortness of breath, edema, or fatigue.

As HF develops, the body activates neurohormonal compensatory mechanisms. These mechanisms represent the body's attempt to cope with the HF and are responsible for the signs and symptoms that develop (Norris, 2019). Understanding these mechanisms is important because the treatment for HF is aimed at correcting them and relieving symptoms.

The most common type of HF is systolic HF, also called Heart Failure with reduced Ejection Fraction (HFrEF; see later discussion in Assessment and Diagnostic Findings). **Systolic heart failure** results in decreased blood ejected from the ventricle. The decreased blood flow is sensed by baroreceptors in the aortic and carotid bodies, and the sympathetic nervous system is then stimulated to release epinephrine and norepinephrine (Fig. 25-1). The purpose of this initial response is to increase heart rate and contractility and support the failing myocardium, but the continued response has multiple negative effects. Sympathetic stimulation causes vasoconstriction in the skin, gastrointestinal tract, and kidneys. A decrease in renal perfusion due to low CO and vasoconstriction then causes the release of renin by the kidneys. Renin converts the plasma protein angiotensinogen to angiotensin I, which then circulates to the lungs. Angiotensin-converting enzyme (ACE) in the lumen of pulmonary blood vessels converts angiotensin I to angiotensin II, a potent vasoconstrictor, which then increases the blood pressure and afterload.

Angiotensin II also stimulates the release of aldosterone from the adrenal cortex, resulting in sodium and fluid retention by the renal tubules and an increase in blood volume. These mechanisms lead to the fluid volume overload commonly seen in HF. Angiotensin, aldosterone, and other neurohormones (e.g., endothelin) lead to an increase in preload and afterload, which increases stress on the ventricular wall, causing an increase in cardiac workload. A counterregulatory mechanism is attempted through the release of natriuretic peptides. Atrial natriuretic peptide (ANP) and B-type natriuretic peptide (BNP; brain type) are released from the overdistended cardiac chambers. These substances promote vasodilation and diuresis. However, their effect is usually not strong enough to overcome the negative effects of the other mechanisms (Norris, 2019).

## Physiology/Pathophysiology



**Figure 25-1 •** The pathophysiology of heart failure. A decrease in cardiac output activates multiple neurohormonal mechanisms that ultimately result in the signs and symptoms of heart failure.

As the heart's workload increases, contractility of the myocardial muscle fibers decreases. Decreased contractility results in an increase in end-diastolic blood volume in the ventricle, stretching the myocardial muscle fibers and increasing the size of the ventricle (ventricular dilation). The heart compensates for the increased workload by increasing the thickness of the heart muscle (ventricular hypertrophy). Hypertrophy results in abnormal

changes in the structure and function of myocardial cells, a process known as ventricular remodeling. Under the influence of neurohormones (e.g., angiotensin II), enlarged myocardial cells become dysfunctional and die early (a process called *apoptosis*), leaving the other, functional myocardial cells struggling to maintain CO.

As cardiac cells die and the heart muscle becomes fibrotic, **diastolic heart failure**, also called Heart Failure with preserved Ejection Fraction (HFpEF) (see later discussion in Assessment and Diagnostic Findings), can develop, leading to further dysfunction. A stiff ventricle resists filling, and less blood in the ventricles causes a further decrease in CO. All of these compensatory mechanisms of HF have been referred to as the “vicious cycle of heart failure” because low CO leads to multiple mechanisms that make the heart work harder, worsening the HF.

## Clinical Manifestations

Many clinical manifestations are associated with HF ([Chart 25-1](#)). However, the cardinal manifestations of HF are dyspnea; fatigue, which may limit exercise tolerance; and fluid retention, which may lead to congestion, evidenced by pulmonary and peripheral edema (Yancy, Jessup, Bozkurt, et al., 2013). The signs and symptoms of HF are related to the ventricle that is most affected. **Left-sided heart failure**, also referred to as left ventricular failure because of the inability of the left ventricle to fill or eject sufficient blood into the systemic circulation, causes different manifestations than **right-sided heart failure**, also referred to as right ventricular failure because of the inability of the right ventricle to fill or eject sufficient blood into the pulmonary circulation. In chronic HF, particularly congestive heart failure, patients may have signs and symptoms of both left- and right-sided heart failure. The patient with pulmonary edema manifests signs and symptoms of acute decompensation, warranting expeditious treatment.

### Left-Sided Heart Failure



Pulmonary congestion occurs when the left ventricle cannot effectively pump blood out of the ventricle into the aorta and the systemic circulation. The increased left ventricular end-diastolic blood volume increases the left ventricular end-diastolic pressure, which decreases blood flow from the left atrium into the left ventricle during diastole. The blood volume and pressure build up in the left atrium, decreasing flow through the pulmonary veins into the left atrium. Pulmonary venous blood volume and pressure increase in the lungs, forcing fluid from the pulmonary capillaries into the pulmonary tissues and alveoli, causing pulmonary interstitial edema and impaired gas exchange.

The clinical manifestations of pulmonary congestion include dyspnea, cough, pulmonary crackles, and low oxygen saturation levels. An extra heart sound, the S<sub>3</sub>, or “ventricular gallop,” may be detected on auscultation. It is caused by abnormal ventricular filling (Colucci & Dunlay, 2017; Dumitru, 2018).

Chart 25-1



## ASSESSMENT

### Heart Failure

Be alert for the following signs and symptoms:

#### Congestion

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Cough (recumbent or exertional)
- Pulmonary crackles that do not clear with cough
- Weight gain (rapid)
- Dependent edema
- Abdominal bloating or discomfort
- Ascites
- Jugular venous distention
- Sleep disturbance (anxiety or air hunger)
- Fatigue

#### Poor Perfusion/Low Cardiac Output

- Decreased exercise tolerance
- Muscle wasting or weakness
- Anorexia or nausea
- Unexplained weight loss
- Lightheadedness or dizziness
- Unexplained confusion or altered mental status
- Resting tachycardia
- Daytime oliguria with recumbent nocturia
- Cool or vasoconstricted extremities
- Pallor or cyanosis

Adapted from Colucci, W. S., & Dunlay, S. M. (2017). Clinical manifestations and diagnosis of advanced heart failure. *UpToDate*. Retrieved on 12/6/2019 at:

[www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-advanced-heart-failure](http://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-advanced-heart-failure);

Dumitru, I. (2018). Heart failure. *Medscape*. Retrieved on 12/6/2019 at:

[www.emedicine.medscape.com/article/163062-overview](http://www.emedicine.medscape.com/article/163062-overview)

Dyspnea, or shortness of breath, may be precipitated by minimal to moderate activity (dyspnea on exertion [DOE]), yet dyspnea may also occur at rest. The patient may report **orthopnea**, difficulty breathing when lying flat. Patients with orthopnea may use multiple pillows to prop themselves up in bed, or they may sleep sitting up or in a high, reclined position. Some patients have sudden attacks of dyspnea at night, a condition known as **paroxysmal nocturnal dyspnea (PND)**. Fluid accumulating in the dependent extremities during the day may be reabsorbed into the circulating blood volume when the patient lies down. Because the impaired left ventricle cannot eject the increased circulating blood volume, the pressure in the pulmonary circulation increases, shifting fluid into the alveoli. The fluid-filled alveoli cannot exchange oxygen and carbon dioxide. Without sufficient oxygen, the patient experiences dyspnea and has difficulty sleeping (Colucci & Dunlay, 2017; Dumitru, 2018).

The cough associated with left ventricular failure is initially dry and nonproductive. Most often, patients complain of a dry hacking cough that may be mislabeled as asthma or chronic obstructive pulmonary disease (COPD). Over time, the cough may begin to accumulate secretions. Large quantities of frothy sputum, sometimes pink or tan, may be produced, indicating acute decompensated HF and pulmonary edema (Colucci & Dunlay, 2017; Dumitru, 2018).

Adventitious breath sounds may be heard in various areas of the lungs. Usually, bibasilar crackles that do not clear with coughing are detected in the early phase of left ventricular failure. As the failure worsens and pulmonary congestion increases, crackles may be auscultated throughout the lung fields. At this point, oxygen saturation may decrease.

In addition to pulmonary manifestations, the decreased amount of blood ejected from the left ventricle can lead to inadequate tissue perfusion. The diminished CO has widespread manifestations because not enough blood reaches all of the tissues and organs (low perfusion) to provide the necessary oxygen. The decrease in stroke volume (SV) can also stimulate the sympathetic nervous system to release catecholamines, which further impedes perfusion to many organs, including the kidneys.

As reduced CO and catecholamines decrease blood flow to the kidneys, urine output drops. Renal perfusion pressure falls, and the renin–angiotensin–aldosterone system is stimulated to increase blood pressure and intravascular volume. While the patient sleeps, the cardiac workload decreases, improving renal perfusion. This may cause **nocturia** (i.e., frequent urination at night) (Colucci & Dunlay, 2017; Dumitru, 2018).

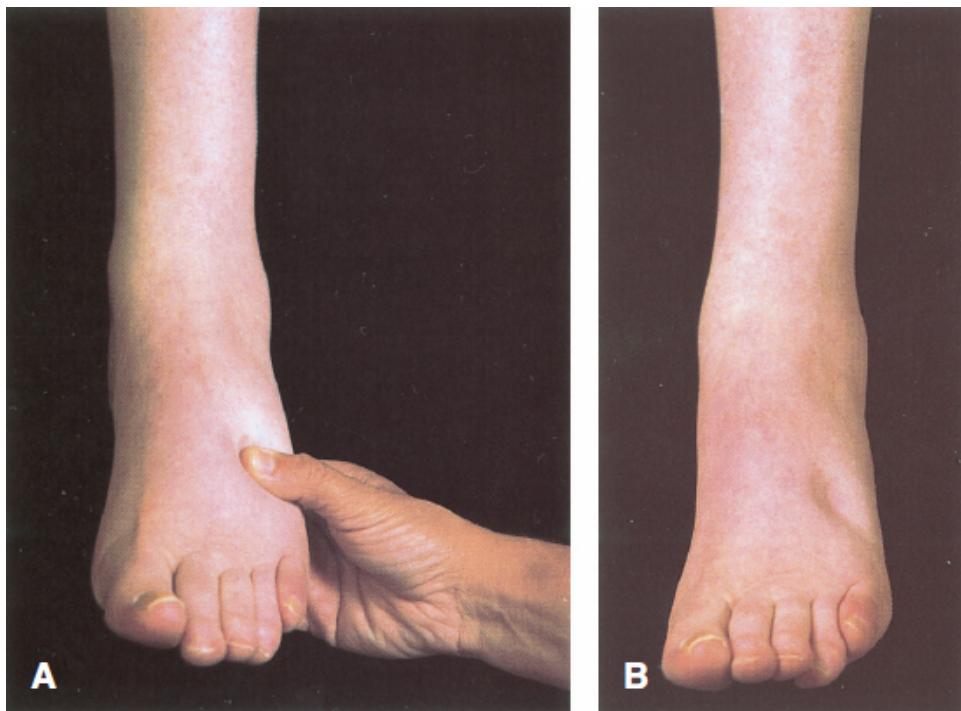
As HF progresses, decreased output from the left ventricle may cause other symptoms. Decreased gastrointestinal perfusion causes altered digestion. Decreased brain perfusion causes dizziness, lightheadedness, confusion, restlessness, and anxiety due to decreased oxygenation and blood flow. As

anxiety increases, so does dyspnea, increasing anxiety and creating a vicious cycle. Stimulation of the sympathetic system also causes the peripheral blood vessels to constrict, so the skin appears pale or ashen and feels cool and clammy.

A decrease in SV causes the sympathetic nervous system to increase the heart rate (tachycardia), often causing the patient to complain of palpitations. The peripheral pulses become weak. Without adequate CO, the body cannot respond to increased energy demands, and the patient becomes easily fatigued and has decreased activity tolerance. Fatigue also results from the increased energy expended in breathing and the insomnia that results from respiratory distress, coughing, and nocturia (Colucci & Dunlay, 2017; Dumitru, 2018).

### Right-Sided Heart Failure

When the right ventricle fails, congestion in the peripheral tissues and the viscera predominates. This occurs because the right side of the heart cannot eject blood effectively and cannot accommodate all of the blood that normally returns to it from the venous circulation. Increased venous pressure leads to jugular venous distention (JVD) and increased capillary hydrostatic pressure throughout the venous system. Systemic clinical manifestations include dependent edema (edema of the lower extremities), **hepatomegaly** (enlargement of the liver), **ascites** (accumulation of fluid in the peritoneal cavity), and weight gain due to retention of fluid. Edema usually affects the feet and ankles and worsens when the patient stands or sits for a long period. The edema may decrease when the patient elevates the legs. Edema can gradually progress up the legs and thighs and eventually into the external genitalia and lower trunk. Ascites is evidenced by increased abdominal girth and may accompany lower body edema or may be the only edema present. Sacral edema is common in patients who are on bed rest, because the sacral area is dependent. Pitting edema, in which indentations in the skin remain after even slight compression with the fingertips (Fig. 25-2), is generally obvious after retention of at least 4.5 kg (10 lb) of fluid (4.5 L).



**Figure 25-2** • Example of pitting edema. **A.** The nurse applies pressure to an area near the ankle. **B.** When the pressure is released, an indentation remains in the edematous tissue. Reprinted with permission from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Hepatomegaly and tenderness in the right upper quadrant of the abdomen result from venous engorgement of the liver. The increased pressure may interfere with the liver's ability to function (secondary liver dysfunction). As hepatic dysfunction progresses, increased pressure within the portal vessels may force fluid into the abdominal cavity, causing ascites. Ascites may increase pressure on the stomach and intestines and cause gastrointestinal distress. Hepatomegaly may also increase pressure on the diaphragm, causing respiratory distress.

Anorexia (loss of appetite), nausea, or abdominal pain may result from the venous engorgement and venous stasis within the abdominal organs. The generalized weakness that accompanies right-sided HF results from reduced CO and impaired circulation (Colucci & Dunlay, 2017; Dumitru, 2018).

### Congestive Heart Failure

Right-sided heart failure can sometimes occur as a result of left-sided failure. The failure of these dual mechanisms is sometimes referred to as **congestive heart failure**. When the left ventricle fails, increased fluid pressure is transferred back through the lungs, leading to damage of the right side of the

heart. When the right side loses pumping power, the blood backs up in the body's venous system. This may cause swelling or congestion in the legs, ankles, and swelling within the abdomen such as the GI tract and liver. Increased venous pressure may also lead to JVD and increased capillary hydrostatic pressure throughout the venous system. Edema may be present in the periphery as well as within the pulmonary vascular bed. Without appropriate treatment, this may progress to pulmonary edema.



## Pulmonary Edema

**Pulmonary edema** is an acute event, reflecting a breakdown of physiologic compensatory mechanisms; hence, it is sometimes referred to as acute decompensated heart failure. It can occur following acute MI or as an exacerbation of chronic HF. When the left ventricle begins to fail, blood backs up into the pulmonary circulation, causing pulmonary interstitial edema. This may occur quickly in some patients, a condition sometimes called *flash pulmonary edema*. Pulmonary edema can also develop slowly, especially when it is caused by noncardiac disorders such as kidney injury and other conditions that cause fluid overload. The left ventricle cannot handle the volume overload, and blood volume and pressure build up in the left atrium. The rapid increase in atrial pressure results in an acute increase in pulmonary venous pressure, which produces an increase in hydrostatic pressure that forces fluid out of the pulmonary capillaries and into the interstitial spaces and alveoli (Norris, 2019).

As a result of decreased cerebral oxygenation, the patient may become increasingly restless and anxious. Along with a sudden onset of breathlessness and a sense of suffocation, the patient may be tachypneic with low oxygen saturation levels. The skin and mucous membranes may be pale to cyanotic, and the hands may be cool and clammy. Tachycardia and JVD may be present. Incessant coughing may occur, producing increasing quantities of foamy sputum. The patient may become progressively confused. The situation demands emergent action before oxygenation and perfusion levels become critical.

## Assessment and Diagnostic Findings

For many years, the severity of HF was classified solely according to the patient's symptoms, using the New York Heart Association (NYHA) classification of HF. This classification system, which is still in widespread use, is described in [Table 25-1](#). The American College of Cardiology and the American Heart Association (ACC/AHA) have developed another HF classification system (Yancy et al., 2013). This system, described in [Table 25-2](#), takes into consideration the natural history and progressive nature of HF.

The ACC/AHA periodically issues evidence-based guidelines for patients with HF or at high-risk of having HF, using this classification system as a framework for treatment (Yancy et al., 2013; Yancy, Jessup, Bozkurt, et al., 2016; Yancy et al., 2017).

HF may go undetected until the patient presents with signs and symptoms of pulmonary and peripheral edema. Some of the physical signs that suggest HF may also occur with other diseases, such as kidney injury and COPD; therefore, diagnostic testing is essential to confirm a diagnosis of HF.

Assessment of ventricular function is an essential part of the initial diagnostic workup. An echocardiogram is performed to determine the ejection fraction (EF), identify anatomic features such as structural abnormalities and valve malfunction, and confirm the diagnosis of HF. The **ejection fraction** is a measure of ventricular contractility; it is the percentage of the end-diastolic blood volume that is ejected with each heartbeat. An expected EF is 55% to 65% of the ventricular volume; the ventricle does not completely empty between contractions (Wiegand, 2017).

**TABLE 25-1** New York Heart Association (NYHA) Classification of Heart Failure

Classification	Signs and Symptoms
I	No limitation of physical activity Ordinary activity does not cause undue fatigue, palpitation, or dyspnea.
II	Slight limitation of physical activity Comfortable at rest, but ordinary physical activity causes fatigue, palpitation, or dyspnea.
III	Marked limitation of physical activity Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
IV	Unable to carry out any physical activity without discomfort Symptoms of cardiac insufficiency at rest If any physical activity is undertaken, discomfort is increased.

Adapted from Yancy, C. W., Jessup, M., Bozkurt, B., et al. (2013). 2013 ACCF/AHA Guideline for the management of heart failure. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*, 128(16), e240–e327.

There are two recognized main types of left-sided HF, with a third, emerging category. In **heart failure with reduced ejection fraction (HFrEF)**, or systolic heart failure, the left ventricle loses the ability to contract effectively, manifesting as EFs of less than 40%, reflecting decreased CO and pump failure (Yancy et al., 2017).

**Heart failure with preserved ejection fraction (HFpEF)**, or diastolic heart failure, is diagnosed when the left ventricular function measures greater

than or equal to 50%, yet the ventricle loses its ability to relax due to myocardial stiffness. Because of the noncompliance of the ventricular wall, the chamber is unable to fill at normal capacity during the relaxation phase of diastole (Yancy et al., 2017).

**Heart failure with midrange ejection fraction (HFmrEF)** is a third and emerging classification category, with EFs typically between 40% and 49% (van der Meer et al., 2019).

Diagnosing a patient with HFpEF is more challenging than diagnosing a patient with HFrEF, because the diagnosis of HFpEF is a *diagnosis of exclusion*. That is, it is made by excluding other potential noncardiac causes suggestive of HF. The incidence of HFpEF is increasing and is becoming more commonplace among older adult women with a history of hypertension; indeed, hypertension is the most common underlying cause of HFpEF. Comorbid conditions such as obesity, CAD, diabetes, atrial fibrillation, and hyperlipidemia are also common in patients with HFpEF (Yancy et al., 2013).

In addition to the echocardiogram, a chest x-ray and a 12-lead electrocardiogram (ECG) are obtained to assist in the diagnosis. Laboratory studies usually performed during the initial workup include serum electrolytes, blood urea nitrogen (BUN), creatinine, liver function tests, thyroid-stimulating hormone, complete blood count (CBC), BNP, and routine urinalysis. The results of these laboratory studies assist in determining the underlying cause and can also be used to establish a baseline to assess effects of treatment. The BNP level is a key diagnostic indicator of HF; high levels are a sign of high cardiac filling pressure and can aid in both the diagnosis and management of HF; in particular, rising levels may suggest an acute exacerbation of HF (Yancy et al., 2013). BNP levels are best used for diagnostic purposes when there is a baseline measurement and a measurement obtained at the time of treatment (e.g., hospital discharge) to help in determining a posttreatment prognosis (Yancy et al., 2017).

**TABLE 25-2** American College of Cardiology and American Heart Association (ACC/AHA) Classification of Heart Failure

Classification Criteria	Patient Characteristics	Treatment Recommendations for Appropriate Patients	
<b>Stage A</b>	Patients at high risk for developing left ventricular dysfunction but without structural heart disease or symptoms of HF	Hypertension Atherosclerotic disease Diabetes Metabolic syndrome	Heart healthy lifestyle Risk factor control of hypertension, lipids, diabetes, obesity
<b>Stage B</b>	Patients with left ventricular dysfunction or structural heart disease who have not developed symptoms of HF	History of myocardial infarction Left ventricular hypertrophy Low ejection fraction	Implement stage A recommendations, plus: <ul style="list-style-type: none"><li>• ACE inhibitor, or ARB, or ARNI for low EF or history of MI</li><li>• Beta-blocker</li><li>• Statin</li></ul>
<b>Stage C</b>	Patients with left ventricular dysfunction or structural heart disease with current or prior symptoms of heart disease	Shortness of breath Fatigue Decreased exercise tolerance	Implement stage A and B recommendations, plus: <ul style="list-style-type: none"><li>• Diuretics</li><li>• Aldosterone antagonist</li><li>• Sodium restriction</li><li>• Implantable defibrillator</li><li>• Cardiac resynchronization therapy</li></ul>
<b>Stage D</b>	Patients with refractory end-stage HF requiring specialized interventions	Symptoms despite maximal medical therapy Recurrent hospitalizations	Implement stage A, B, and C recommendations, plus: <ul style="list-style-type: none"><li>• Fluid restriction</li><li>• End-of-life care</li><li>• Extraordinary measures:<ul style="list-style-type: none"><li>• Inotropes</li><li>• Cardiac transplantation</li><li>• Mechanical support</li></ul></li></ul>

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ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; EF, ejection fraction; HF, heart failure; MI, myocardial infarction. Adapted from Yancy, C. W., Jessup, M., Bozkurt, B., et al. (2013). 2013 ACCF/AHA guideline for the diagnosis and management of heart failure: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*, 128(16), e240–e327; Yancy, C. W., Jessup, M., Bozkurt, B., et al. (2016). ACC/AHA/HFSA focused update on new pharmacological therapy for heart failure: An update of the 2013 ACCF/AHA guideline for the management of heart failure: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *Circulation*, 134(13), e282–e293.

## Medical Management

The prognosis for patients with HF has improved with the use of evidence-based protocols for patient management. Specific interventions are based on the stage of HF (Yancy et al., 2013; Yancy et al., 2016; Yancy et al., 2017). The management goals of HF include the following (Cyrille & Patel, 2017):

- Improvement of cardiac function with optimal pharmacologic management
- Reduction of symptoms and improvement of functional status
- Stabilization of patient condition and lowering of the risk of hospitalization
- Delay of the progression of HF and extension of life expectancy
- Promotion of a lifestyle conducive to cardiac health

Treatment options vary according to the severity of the patient's condition, comorbidities, and cause of the HF, and may include oral and intravenous (IV) medications, lifestyle modifications, supplemental oxygen, and surgical interventions, including implantation of cardiac devices, and cardiac transplantation (see Chapter 24).

Managing the patient with HF begins with providing comprehensive education and counseling to the patient and family. The patient and family must understand the nature of HF and the importance of their participation in the treatment regimen, including side and adverse effects of pharmacologic therapies. Lifestyle recommendations include restriction of dietary sodium; avoidance of smoking, including secondhand smoke; avoidance of excessive fluid and alcohol intake; weight reduction when indicated; and regular exercise. The patient must also know how to recognize signs and symptoms that need to be reported to the primary provider.

## Pharmacologic Therapy

Several types of medications are routinely prescribed for patients with HF. The cornerstone of therapy for patients with HFrEF (systolic HF), which is the most common type of HF, includes a diuretic, an angiotensin system blocker,

and a beta-blocker ([Table 25-3](#)). Many of these medications, particularly angiotensin system blockers and beta-blockers, improve symptoms and extend survival. Others, such as diuretics, improve symptoms but may not affect survival (Meyer, 2019b). The patient with HFpEF (diastolic HF) may be prescribed a diuretic, most commonly an aldosterone antagonist (see [Table 25-3](#)), and may also be prescribed an angiotensin system blocker and/or a beta-blocker and find symptomatic relief; however, these drugs are not necessarily associated with improved survival in those patients (Borlaug & Colucci, 2019). Target doses for these medications and alternative medications for treating heart failure are identified in the ACC/AHA guidelines. Nurses, primary providers, and pharmacists work collaboratively toward achieving effective dosing of these medications (Yancy et al., 2013; Yancy et al., 2016; Yancy et al., 2017).

**TABLE 25-3** Select Medications Used to Treat Heart Failure

Medication	Therapeutic Effects	Key Nursing Considerations
<b>Diuretics</b>		
<i>Loop diuretics:</i> furosemide	↓ Fluid volume overload ↓ Signs and symptoms of HF	Observe for electrolyte abnormalities, renal dysfunction, diuretic resistance, and ↓ BP.
<i>Thiazide diuretics:</i> metolazone hydrochlorothiazide		Carefully monitor I&O and daily weight (see <a href="#">Chart 25-2</a> ).
<i>Aldosterone antagonists:</i> spironolactone	Improves HF symptoms in advanced HF	Observe for ↑ serum K <sup>+</sup> , ↓ serum Na <sup>+</sup> .
<b>Angiotensin System Blockers</b>		
<i>ACE Inhibitors:</i> lisinopril enalapril	↓ BP and ↓ afterload Relieves signs and symptoms of HF Prevents progression of HF	Observe for symptomatic ↓ BP, ↑ serum K <sup>+</sup> , cough, and worsening renal function.
<i>ARBs:</i> valsartan losartan	↓ BP and ↓ afterload Relieves signs and symptoms of HF Prevents progression of HF	Observe for symptomatic ↓ BP, ↑ serum K <sup>+</sup> , and worsening renal function.
<i>ARNI:</i> sacubitril-valsartan	↓ BP and ↓ afterload ↓ Fluid volume overload ↓ Signs and symptoms of HF Prevents progression of HF	Observe for symptomatic ↓ BP, ↑ serum K <sup>+</sup> , cough, dizziness, and renal failure.
<b>Beta-Adrenergic-Blocking Agents (Beta-Blockers)</b>		
carvedilol bisoprolol metoprolol	Dilates blood vessels and ↓ afterload ↓ Signs and symptoms of HF Improves exercise capacity	Observe for ↓ heart rate, symptomatic ↓ BP, dizziness, and fatigue.
<b>Ivabradine</b>	Decreases rate of conduction through the SA node	Observe for ↓ heart rate, symptomatic ↓ BP, dizziness, and fatigue.
<b>Hydralazine-isosorbide dinitrate</b>	Dilates blood vessels ↓ BP and ↓ afterload	Observe for symptomatic ↓ BP.
<b>Digitalis</b>		
digoxin	Improves cardiac contractility ↓ Signs and symptoms of HF	Observe for ↓ heart rate and digitalis toxicity.

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BP, blood pressure; ↓, decreases; HF, heart failure; ↑, increases; I&O, input and output; K<sup>+</sup>, potassium; Na<sup>+</sup>, sodium; SA, sinoatrial.

Adapted from Burchum, J. R., & Rosenthal, L. D. (2019). *Lehne's pharmacology for nursing care* (9th ed.). St. Louis, MO: Elsevier.

## Diuretics

Diuretics are prescribed to remove excess extracellular fluid by increasing diuresis in patients with signs and symptoms of fluid overload. ACC/AHA guidelines advocate using the smallest dose of diuretic necessary to control fluid volume (Yancy et al., 2013). The type and dose of diuretic prescribed depend on clinical signs and symptoms and renal function. Careful patient monitoring and dose adjustments are necessary to balance the effectiveness of these medications with the side effects ([Chart 25-2](#)). Loop, thiazide, and aldosterone-blocking diuretics may be prescribed; these medications differ in their site of action in the kidney and their effects on renal electrolyte excretion and reabsorption.

*Loop diuretics*, such as furosemide, inhibit sodium and chloride reabsorption mainly in the ascending loop of Henle. Patients with HF and with severe volume overload are generally treated with a loop diuretic first (Burchum & Rosenthal, 2019). *Thiazide diuretics*, such as metolazone, inhibit sodium and chloride reabsorption in the early distal tubules. Both of these classes of diuretics increase potassium excretion; therefore, patients treated with these medications must have their serum potassium levels closely monitored. Diuretics can also lead to orthostatic hypotension and kidney injury. Both a loop and a thiazide diuretic may be used in patients with severe HF who are unresponsive to a single diuretic. The need for diuretics can be decreased if the patient avoids excessive fluid intake (e.g., more than 2000 mL/day) and adheres to a low sodium diet (e.g., no more than 2 g/day).

*Aldosterone antagonists*, such as spironolactone, are potassium-sparing diuretics that block the effects of aldosterone in the distal tubule and collecting duct (Yancy et al., 2016). As noted previously, they are frequently prescribed for patients with HFrEF. Serum creatinine and potassium levels are monitored frequently (e.g., within the first week and then every 4 weeks) when spironolactone is first given. These drugs are not prescribed for patients with an elevated serum creatinine.

## Chart 25-2 PHARMACOLOGY

## Administering and Monitoring Diuretic Therapy

When nursing care involves diuretic therapy for conditions such as heart failure, the nurse needs to administer the medication and monitor the patient's response carefully, as follows:

- Prior to administration of the diuretic, check laboratory results for electrolyte depletion, especially potassium, sodium, and magnesium.
- Prior to administration of the diuretic, check for signs and symptoms of volume depletion, such as orthostatic hypotension, lightheadedness, and dizziness.
- Administer the diuretic at a time conducive to the patient's lifestyle—for example, early in the day to avoid nocturia.
- Monitor urine output during the hours after administration, and analyze intake, output, and daily weights to assess response.
- Monitor blood pressure for orthostatic changes.
- Continue to monitor serum electrolytes for depletion. Replace potassium with increased oral intake of food rich in potassium or potassium supplements. Replace magnesium as needed.
- Monitor for hyperkalemia in patients receiving potassium-sparing diuretics.
- Continue to assess for signs of volume depletion.
- Monitor creatinine for increased levels indicative of diuretic-induced renal dysfunction.
- Monitor for elevated uric acid level and signs and symptoms of gout.
- Assess lungs sounds and edema to evaluate response to therapy.
- Monitor for adverse reactions such as arrhythmias.
- Assist patients to manage urinary frequency and urgency associated with diuretic therapy.

Adapted from Burchum, J. R., & Rosenthal, L. D. (2019). *Lehne's pharmacology for nursing care* (10th ed.). St. Louis, MO: Elsevier.

Loop diuretics are administered IV for exacerbations of HF when rapid diuresis is necessary, as when pulmonary edema is present (see later discussion). Diuretics improve the patient's symptoms, provided that renal function is adequate. As HF progresses, cardiorenal syndrome may develop or worsen. Cardiorenal syndrome is a type of prerenal acute kidney injury characterized by a disruption in adequate blood flow to the kidneys. Patients

with this syndrome are resistant to diuretics and may require other interventions to deal with congestive signs and symptoms.

## Angiotensin System Blockers

Angiotensin system blockers include classes of medications such as the ACE inhibitors, angiotensin receptor blockers (ARBs), and angiotensin receptor-neprilysin inhibitors (ARNIs).

### Angiotensin-Converting Enzyme Inhibitors

ACE inhibitors, such as lisinopril, have been found to relieve clinical manifestations of HF and significantly decrease mortality and morbidity in patients with HFrEF. Specifically, they slow the progression of HF, improve exercise tolerance, and decrease the number of hospitalizations in patients with HFrEF (Yancy et al., 2013; Yancy et al., 2017). ACE inhibitors are also appropriate for hypertension management in patients with HFpEF (Yancy et al., 2017). Available as oral and IV medications, ACE inhibitors promote vasodilation and diuresis, ultimately decreasing both afterload and preload. Vasodilation reduces resistance to left ventricular ejection of blood, diminishing the heart's workload and improving ventricular emptying. ACE inhibitors decrease the secretion of aldosterone, a hormone that causes the kidneys to retain sodium and water. ACE inhibitors also promote renal excretion of sodium and fluid (while retaining potassium), thereby reducing left ventricular filling pressure and decreasing pulmonary congestion. These agents are also recommended for prevention of HF in patients at risk due to vascular disease and diabetes (Yancy et al., 2013; Yancy et al., 2017).

Patients receiving ACE inhibitors are monitored for hypotension, hyperkalemia (increased potassium in the blood), and alterations in renal function, especially if they are also receiving diuretics. Because ACE inhibitors cause the kidneys to retain potassium, the patient who is also receiving a loop diuretic or a thiazide diuretic may not need to take oral potassium supplements. However, the patient receiving a potassium-sparing diuretic, such as an aldosterone antagonist, which does not cause potassium loss with diuresis, must be carefully monitored for hyperkalemia. ACE inhibitors may be discontinued if the potassium level remains greater than 5.5 mEq/L or if the serum creatinine rises.

An adverse effect of ACE inhibitors includes a dry, persistent cough that may not respond to cough suppressants due to the inhibition of the enzyme kininase, which inactivates bradykinin. The nurse should carefully assess any cough in a patient taking an ACE inhibitor, as this symptom can also indicate a worsening of ventricular function and failure. In less than 1% of patients, ACE inhibitors may cause an allergic reaction accompanied by angioedema. This reaction tends to occur more frequently in African Americans and women (Yancy et al., 2016; Yancy et al., 2017). If angioedema affects the

oropharyngeal area and impairs breathing, the ACE inhibitor must be stopped immediately and appropriate emergency care must be provided.

If the patient cannot continue taking an ACE inhibitor because of development of cough, an elevated creatinine level, or hyperkalemia, an ARB, an ARNI, or a combination of hydralazine and isosorbide dinitrate is prescribed (see [Table 25-3](#)).

### **Angiotensin Receptor Blockers**

Whereas ACE inhibitors block the conversion of angiotensin I to angiotensin II, ARBs, such as valsartan, block the vasoconstricting effects of angiotensin II at the angiotensin II receptors. ARBs are commonly prescribed as an alternative to ACE inhibitors, as they are associated with reduced morbidity and mortality in patients with HFrEF and can provide symptomatic relief in patients with HFpEF who are intolerant of ACE inhibitors (Yancy et al., 2013; Yancy et al., 2017). ARBs do not inhibit kininase; therefore, ARBs are not associated with the bothersome cough that occurs with some patients prescribed an ACE inhibitor.

### **Angiotensin Receptor-Neprilysin Inhibitors**

An ARNI combines an ARB with a neprilysin inhibitor. Neprilysin is an enzyme that breaks down natriuretic peptides. Participants with HFrEF enrolled in clinical trials who were prescribed an ARNI demonstrated a significant reduction in cardiovascular death or hospitalization as compared with participants prescribed an ACE inhibitor (Meyer, 2019b). Based on these findings, updated ACC/AHA guidelines advocate prescribing an ARNI as first-line angiotensin system blocker therapy for most patients with symptomatic HFrEF. However, an ARNI is reportedly a costlier option than most ACE inhibitors and ARBs, which may preclude its practical use. For patients unable to take an ARNI, an ACE inhibitor or ARB is a good alternative. An ARNI should not be administered concurrently or within 36 hours of an ACE inhibitor as concomitant dosing with both agents is associated with angioedema (Yancy et al., 2016; Yancy et al., 2017). Adverse effects associated with use of an ARNI are similar to those associated with ACE inhibitor or ARB use; therefore, the nurse should assess for hypotension, renal insufficiency, and angioedema in patients taking an ARNI (Yancy et al., 2016; Yancy et al., 2017). The first U.S. Food and Drug Administration (FDA) approved ARNI for use in patients with HF is sacubitril-valsartan.

### **Beta-Blockers**

Beta-blockers block the adverse effects of the sympathetic nervous system. They relax blood vessels, lower blood pressure, decrease afterload, and decrease cardiac workload. Beta-blockers, such as carvedilol, have been found to improve functional status and reduce mortality and morbidity in patients with HF (Burchum & Rosenthal, 2019). In addition, beta-blockers have been

recommended for patients with asymptomatic HFrEF to prevent progression and the onset of symptoms of HF, even if patients do not have a history of MI. The therapeutic effects of these drugs may not be seen for several weeks or even months (Yancy et al., 2013, Yancy et al., 2017).

Beta-blockers can produce a number of side effects, including dizziness, hypotension, bradycardia, fatigue, and depression. Side effects are most common in the initial few weeks of treatment. Because of the potential for side effects, beta-blockers are started at a low dose. The dose is titrated up slowly (every few weeks), with close monitoring after each dosage increase. Nurses educate patients about potential symptoms during the early phase of treatment and stress that adjustment to the drug may take several weeks. Nurses must also provide support to patients going through this symptom-provoking phase of treatment. Because beta-blockade can cause bronchial constriction, these drugs are used with caution in patients with a history of bronchospastic diseases such as asthma.

### Ivabradine

Ivabradine is a new agent that is a hyperpolarization-activated cyclic nucleotide channel blocker. It is a medication with unique electrophysiologic effects, characterized by its negative chronotropic effect on the sinoatrial node, thereby decreasing the heart rate without targeting the neurohormonal system. It is indicated as an adjunct agent to beta-blockers in patients with symptomatic HFrEF and with high resting heart rates of at least 70 bpm (Koruth, Lala, Pinney, et al., 2017). It may also be beneficial for patients with HFrEF who cannot tolerate beta-blockers (Yancy et al., 2017). Adverse effects of ivabradine include bradycardia resulting in dizziness and fatigue; it is also associated with an increased risk of atrial fibrillation (Koruth et al., 2017).

### Hydralazine and Isosorbide Dinitrate

A combination of hydralazine and isosorbide dinitrate may be an alternative medication for patients who cannot take any of the three angiotensin system blockers (i.e., ACE inhibitor, ARB, and ARNI), so long as the patient's systolic BP is at least 90 mm Hg. Nitrates (e.g., isosorbide dinitrate) cause venous dilation, which reduces the amount of blood return to the heart and lowers preload. Hydralazine lowers systemic vascular resistance and left ventricular afterload. Hydralazine-isosorbide dinitrate is associated with decreased hospitalizations and improved survival in patients with HFrEF; however, these improvements are not as robust as those associated with angiotensin system blockers (Meyer, 2019b; Yancy et al., 2017). Adverse effects may include hypotension, and rarely, a lupus-type reaction (Meyer, 2019b).

### Digitalis

For many years, digitalis (i.e., digoxin) was considered an essential agent for the treatment of HF. With the introduction of newer medications, it is not prescribed as often. Digoxin increases the force of myocardial contraction and slows conduction through the atrioventricular node. It improves contractility, increasing left ventricular output. Although the use of digoxin does not result in decreased mortality rates among patients with HFrEF, it can be effective in decreasing the symptoms of HF and may help prevent hospitalization (Yancy et al., 2013). Patients with renal dysfunction and older patients should receive smaller doses of digoxin, as it is excreted through the kidneys.

A key concern associated with digoxin therapy is digitalis toxicity. Clinical manifestations of toxicity include anorexia, nausea, visual disturbances, confusion, and bradycardia. The serum potassium level is monitored because the effect of digoxin is enhanced in the presence of hypokalemia and digoxin toxicity may occur. A serum digoxin level is obtained if the patient's renal function changes or there are symptoms of toxicity.



## Intravenous Infusions

IV inotropes (e.g., dopamine, dobutamine, milrinone) increase the force of myocardial contraction; as such, they may be indicated for hospitalized patients with pulmonary edema (i.e., acute decompensated HF). These agents are used for patients who do not respond to routine pharmacologic therapy and are reserved for patients with severe ventricular dysfunction, low blood pressure, or impaired perfusion and evidence of significantly depressed CO, with or without congestion. They are used with caution, as some studies have associated their use with increased mortality (Malotte, Saguros, & Groninger, 2018; Yancy et al., 2013). Patients usually require admission to the intensive care unit (ICU) and may also have hemodynamic monitoring with a pulmonary artery catheter or alternative technology (see Chapter 21). Hemodynamic data are used to assess cardiac function and volume status and to guide therapy with inotropes, vasodilators, and diuretics (Urden, Stacy, & Lough, 2018). Patients with end-stage HF who cannot be weaned from IV inotropes may be candidates for continuous therapy at home (Malotte et al., 2018).

### Dopamine

Dopamine is a vasopressor given to increase BP and myocardial contractility. Given at low doses, a dopamine infusion may be helpful as an adjunct therapy along with loop diuretics in improving diuresis, preserving renal function, and improving renal blood flow (Yancy et al., 2013).

### Dobutamine

Dobutamine is given to patients with significant left ventricular dysfunction and hypoperfusion. A catecholamine, dobutamine stimulates the beta-1 adrenergic receptors. Its major action is to increase cardiac contractility and

renal perfusion to enhance urine output. However, it also increases the heart rate and can precipitate ectopic beats and tachyarrhythmias (Burchum & Rosenthal, 2019).

### Milrinone

Milrinone is a phosphodiesterase inhibitor that leads to an increase in intracellular calcium within myocardial cells, increasing their contractility (Ayres & Maani, 2019). This agent also promotes vasodilation, resulting in decreased preload and afterload and reduced cardiac workload. Milrinone is administered IV to patients with severe HF, including patients who are waiting for heart transplantation (see Chapter 24). Because the drug causes vasodilation, the patient's blood pressure is monitored prior to administration; if the patient is hypovolemic, the blood pressure could drop quickly. The major side effects are hypotension and increased ventricular arrhythmias. Blood pressure and ECG are monitored closely during and following infusions of milrinone.

### Vasodilators

Intravenous vasodilators such as IV nitroglycerin, nitroprusside, or nesiritide may enhance symptom relief for acutely decompensated HF (Yancy et al., 2013). Their use is contraindicated in patients who are hypotensive. Blood pressure is continually assessed in patients receiving IV vasodilator infusions.

### Adjunct Medications for Heart Failure

The importance of ensuring that patients with hypertension take prescribed antihypertensive agents as prescribed is of paramount importance (see Chapter 27 for further discussion of antihypertensive medications). Target blood pressures should be less than 130/80 mm Hg. Maintaining BPs at these levels is associated with reduced likelihood of morbid progression to symptomatic HF in patients who are asymptomatic. It is also associated with improved morbidity in patients who are symptomatic with both HFrEF and HFpEF (Yancy et al., 2017).

Anemia is independently associated with HF disease severity, and iron deficiency appears to be uniquely associated with reduced exercise capacity. IV iron repletion in patients with HF may improve functional capacity and quality of life; however, there is mixed evidence to support the use of oral iron supplementation. Erythropoietin-stimulating agents, such as darbepoetin alfa, are not recommended in patients with both HF and anemia as a risk of thromboembolic events associated with their use has been observed during clinical trials (Yancy et al., 2017).

Anticoagulants may be prescribed, especially if the patient has a history of atrial fibrillation or a thromboembolic event. Antiarrhythmic drugs such as amiodarone may be prescribed for patients with arrhythmias, along with an evaluation for device therapy with an implantable cardioverter defibrillator

(ICD) (see Chapter 22). Medications to manage hyperlipidemia (e.g., statins) are also routinely prescribed, in tandem with guidance on nutritional therapy (see following section). It is recommended that patients with HF avoid nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen because the risk of decreased renal perfusion is higher, especially in older adults (Schwartz, Schmader, Hanlon, et al., 2018).

### Adjunct Therapies for Heart Failure

Additional therapies that may be indicated in the treatment of patients with HF include nutritional therapy, supplemental oxygen, management of sleep disorders, and procedural or surgical interventions.

#### Nutritional Therapy

Following a low sodium (no more than 2 g/day) diet and avoiding excessive fluid intake are usually recommended, although studies differ regarding the effectiveness of sodium restriction (Yancy et al., 2013). Decreasing dietary sodium reduces fluid retention and the symptoms of peripheral and pulmonary congestion. The purpose of sodium restriction is to decrease the amount of circulating blood volume, which decreases myocardial work. A balance should be achieved between the patient's ability to adhere to the diet and the recommended guidelines.

Nutritional supplements, such as vitamins and antioxidants, are not recommended for patients with HF as no benefits are associated with their use. Omega-3 polyunsaturated fatty acid (PUFA) supplementation is associated with decreased fatal cardiovascular events and is recommended for patients with either HFrEF or HFpEF, unless contraindicated (Yancy et al., 2013).

Any change in eating patterns should consider good nutrition as well as the patient's likes, dislikes, and cultural food patterns. Patient adherence is important because dietary indiscretions may result in exacerbations of HF symptoms. However, behavioral changes in eating patterns are difficult for many patients to achieve.

#### Supplemental Oxygen

Oxygen therapy may become necessary as HF progresses based on the degree of pulmonary congestion and resulting hypoxia. Some patients require supplemental oxygen only during periods of activity (see Chapter 20 for further discussion of oxygen delivery systems).

#### Management of Sleep Disorders

Sleep disorders, including sleep apnea, are common in patients with HF. It is estimated that 61% of patients with HF have either central or obstructive sleep apnea (OSA). A formal sleep study should be performed. Continuous positive

airway pressure (CPAP) might be recommended if results from the sleep study suggest OSA (see Chapter 18). CPAP has been shown to improve sleep quality, reduce apneic episodes and excessive daytime sleepiness, and improve nocturnal oxygenation in patients with OSA and HF (Yancy et al., 2017).

### Procedural and Surgical Interventions

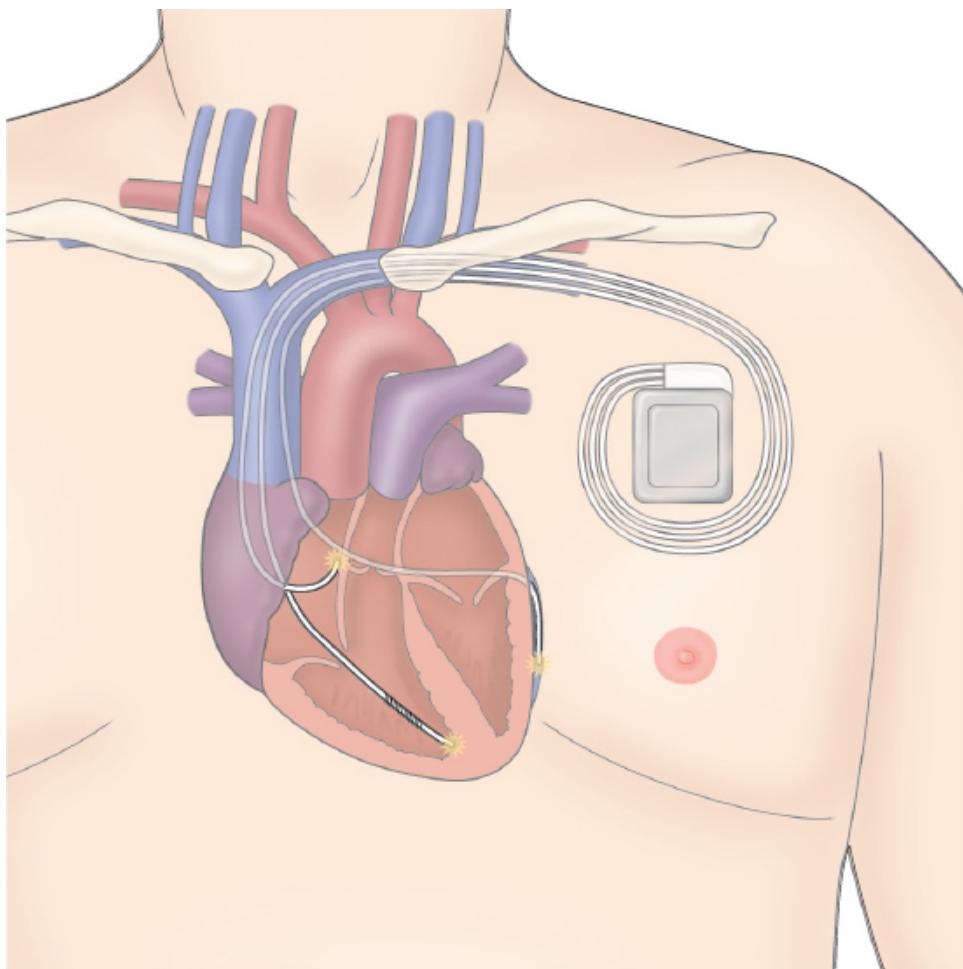
A number of procedures and surgical approaches may benefit patients with HF. If the patient has underlying CAD, coronary artery revascularization with PCI or coronary artery bypass surgery (see Chapter 23) may be considered. Ventricular function may improve in some patients when coronary flow is increased.

Patients with HF are at high risk for arrhythmias, and sudden cardiac death is common among patients with advanced HF. In patients with severe left ventricular dysfunction and the possibility of life-threatening arrhythmias, placement of an ICD can prevent sudden cardiac death and extend survival (see Chapter 22). Candidates for an ICD include those with an EF less than 35%, including those with and without a history of ventricular arrhythmias (Yancy et al., 2013).

Patients with HF who do not improve with standard therapy may benefit from **cardiac resynchronization therapy (CRT)**. CRT involves the use of a biventricular pacemaker to treat electrical conduction defects and to synchronize ventricular contractions. A prolonged QRS duration on ECG indicates left bundle branch block, which is a type of delayed conduction that is frequently seen in patients with HF. This problem results in asynchronous conduction and contraction of the right and left ventricles, which can further decrease EF (Yancy et al., 2013). The use of a pacing device with leads placed in the right atrium, right ventricle, and left ventricular cardiac vein can synchronize the contractions of the right and left ventricles (Fig. 25-3). This intervention improves CO, optimizes myocardial energy consumption, reduces mitral regurgitation, and slows the ventricular remodeling process. For patients with a CRT, improvement of left ventricular EF is associated with reduced rates of ventricular arrhythmias. There are combination devices available for patients who require CRT and an ICD (Gulati & Udelson, 2018). See Chapter 22 for further discussion of care of patients with pacemakers, CRT, and ICDs.

Ultrafiltration is an alternative intervention for patients with severe fluid overload. It is reserved for patients with advanced HF who are resistant to diuretic therapy (Yancy et al., 2013). A dual-lumen central IV catheter is placed, and the patient's blood is circulated through a small bedside filtration machine. Liters of excess fluid and plasma are removed slowly from the patient's intravascular circulating volume over a number of hours. The patient's output of filtration fluid, blood pressure, and hemoglobin (analyzed for hemoconcentration) are monitored as indicators of volume status. Research

on ultrafiltration is ongoing, and targets comparisons of its efficacy to diuretics and the optimal fluid removal target (Costanzo, 2019).



**Figure 25-3 •** Cardiac resynchronization therapy. To pace both ventricles, pacemaker leads are placed in the right atrium and right ventricle; a third lead is threaded through the coronary sinus into a lateral vein on the wall of the left ventricle.

For some patients with end-stage HF, cardiac transplantation is one of the few options for long-term survival. Patients with ACC/AHA stage D HF who may be eligible are referred for consideration of transplantation. Some of these patients require mechanical circulatory assistance with an implanted ventricular assist device as a bridge therapy to cardiac transplantation. A left ventricular assist device may also be implanted as *destination therapy* (permanent therapy) for select patients (Yancy et al., 2013).

## Gerontologic Considerations

Several normal age-related changes increase the frequency of HF: increased systolic blood pressure, increased ventricular wall thickness, and increased myocardial fibrosis. There are a number of reasons that older adults may need to be hospitalized with HF (Albert, Barnason, Deswal, et al., 2015). Older adults may not always detect or accurately interpret common symptoms of HF such as shortness of breath, or they may have atypical symptoms such as weakness and somnolence. Decreased renal function can make the older patient resistant to diuretics and more sensitive to changes in volume. The administration of diuretics to older men requires nursing surveillance for bladder distention caused by urethral obstruction from an enlarged prostate gland. The bladder may be assessed with an ultrasound scanner or the suprapubic area palpated for an oval mass and percussed for dullness, indicative of bladder fullness. Urinary frequency and urgency may be particularly stressful to older patients, as many have arthritis and limited mobility.

## NURSING PROCESS

### The Patient with Heart Failure



Despite advances in treatment of HF, morbidity and mortality remain high. Nurses have a major impact on outcomes for patients with HF, especially in the areas of patient education and monitoring.

#### Assessment

Nursing assessment for the patient with HF focuses on observing for effectiveness of therapy and for the patient's ability to understand and implement self-care management strategies. Signs and symptoms of worsening HF are analyzed and reported to the patient's provider so that therapy can be adjusted. The nurse also explores the patient's emotional response to the diagnosis of HF, because it is a chronic and often progressive condition that is commonly associated with depression and other psychosocial issues (Jiang, Shorey, Seah, et al., 2018).

#### Health History

The health history focuses on the signs and symptoms of HF, such as dyspnea, fatigue, and edema. Sleep disturbances, particularly sleep suddenly interrupted by shortness of breath, may be reported. Patients are asked about the number of pillows needed for sleep, edema, abdominal symptoms, altered mental status, activities of daily living, and the activities that cause fatigue. Nurses need to be aware of the variety of clinical manifestations that may indicate worsening HF and assess the patient accordingly. While obtaining the patient's history, the nurse assesses the patient's understanding of HF, self-care management strategies, and the patient's ability and willingness to adhere to those strategies.

#### Physical Examination

The patient is observed for restlessness and anxiety that might suggest hypoxia from pulmonary congestion. The patient's level of consciousness is also evaluated for any changes, as low CO can decrease the flow of oxygen to the brain.

The rate and depth of respirations are assessed along with the effort required for breathing. The lungs are auscultated to detect crackles and wheezes (Meyer, 2019a). Crackles are produced by the sudden opening of edematous narrowed airways and alveoli. They may be heard at the end of inspiration and are not cleared with coughing. Wheezing may also be heard in some patients who have bronchospasm along with pulmonary congestion.

The blood pressure is carefully evaluated, because patients with HF may present with hypotension or hypertension. Patients may be assessed for

orthostatic hypotension, especially if they report lightheadedness, dizziness, or syncope. The heart is auscultated for an S<sub>3</sub> heart sound, which is an early sign that increased blood volume fills the ventricle with each beat. Heart rate and rhythm are also documented, and patients are often placed on continuous ECG monitoring in the hospital setting. When the heart rate is rapid or very slow, the CO decreases and potentially worsens the HF. JVD is assessed with the patient sitting at a 45-degree angle; distention greater than 4 cm above the sternal angle is considered abnormal and indicative of right ventricular failure (Bickley, 2017). This is an estimate, not a precise measurement, of high central venous pressure.

The nurse assesses peripheral pulses and rates their volume on a scale from 0 (not palpable) to 3+ (bounding). The skin is also assessed for color and temperature. With significant decreases in SV, there is a decrease in perfusion to the periphery, decreasing the volume of pulses and causing the skin to feel cool and appear pale or cyanotic. The feet and lower legs are examined for edema; if the patient is supine in bed, the sacrum and back are also assessed for edema. The upper extremities may also become edematous in some patients. Edema is typically rated on a scale from 0 (no edema) to 4+ (severe pitting edema).

The abdomen is examined for tenderness and hepatomegaly. The presence of firmness, distention, and possible ascites is noted. The liver may be assessed for hepatojugular reflux. The patient is asked to breathe normally while manual pressure is applied over the right upper quadrant of the abdomen for 30–60 s. If neck vein distention increases more than 1 cm, the finding is positive for increased venous pressure.

If the patient is hospitalized, the nurse measures urinary output and evaluates it in terms of diuretic use. Intake and output records are rigorously maintained and analyzed. It is important to track whether the patient has excreted excessive volume (i.e., negative fluid balance is generally the goal). The intake and output is then compared with changes in weight. Although diuresis is expected, the patient with HF must also be monitored for **oliguria** (diminished urine output, less than 0.5 mL/kg/h for at least 6 h or <400 mL/24 h) or **anuria** (urine output of less than 50 mL/24 h) because of the risk of renal dysfunction.

The patient is weighed daily in the hospital or at home, at the same time of day, with the same type of clothing, and on the same scale. If there is a significant change in weight (i.e., 2–3-lb increase in a day or 5-lb increase in a wk), the primary provider is notified and medications are adjusted (e.g., the diuretic dose is increased).

## Diagnosis

### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Activity intolerance associated with decreased CO
- Hypervolaemia associated with the HF syndrome
- Anxiety associated with clinical manifestations of HF
- Powerlessness associated with chronic illness and hospitalizations
- Impaired family ability to manage regime

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Pulmonary edema
- Hypotension, poor perfusion, and cardiogenic shock (see Chapter 11)
- Arrhythmias (see Chapter 22)
- Thromboembolism (see Chapter 26)
- Pericardial effusion (see later discussion in this chapter)

#### **Planning and Goals**

Major goals for the patient may include promoting activity and reducing fatigue, relieving fluid overload symptoms, decreasing anxiety or increasing the patient's ability to manage anxiety, encouraging the patient to verbalize their ability to make decisions and influence outcomes, and educating the patient and family about health management.

#### **Nursing Interventions**

Nursing interventions revolve around promoting the patient's activity tolerance, managing the patient's fluid volume status, assisting the patient so that anxiety is relieved, helping to minimize any feelings of powerlessness that the patient might experience, and assisting the patient and family members to effectively manage the patient's health. In addition, nursing interventions must be attuned to prevent and manage acute complications that can be experienced by patients with HF.

#### **PROMOTING ACTIVITY TOLERANCE**

Reduced physical activity caused by HF symptoms leads to physical deconditioning that worsens the patient's symptoms and exercise tolerance. Prolonged inactivity, which may be self-imposed, should be avoided because of its deconditioning effects and risks, such as pressure injuries (especially in edematous patients) and venous thromboembolism. An acute illness that exacerbates HF symptoms or requires hospitalization may be an indication for temporary bed rest. Otherwise, some type of physical activity every day should be encouraged. A typical program for a patient with HF might include a daily walking regimen, with the duration increased over a 6-wk period. The primary provider, nurse, and patient collaborate to

develop a schedule that promotes pacing and prioritization of activities. The schedule should alternate activities with periods of rest and avoid having two significant energy-consuming activities occur on the same day or in immediate succession. Before undertaking physical activity, the patient should be given guidelines similar to those noted in **Chart 25-3**. Because some patients may be severely debilitated, they may need to limit physical activities to only 3–5 min at a time, one to four times per day. The patient should increase the duration of the activity, then the frequency, before increasing the intensity of the activity (Piña, 2019).

### Chart 25-3 HEALTH PROMOTION

#### An Exercise Program for Patients with Heart Failure

Before undertaking physical activity, the patient should be given the following guidelines:

- Talk with your primary provider for specific exercise program recommendations.
- Begin with low-impact activities such as walking.
- Start with warm-up activity followed by sessions that gradually build up to about 30 min.
- Follow your exercise period with cool-down activities.
- Avoid performing physical activities outside in extreme hot, cold, or humid weather.
- Wait 2 h after eating a meal before performing the physical activity.
- Ensure that you are able to talk during the physical activity; if you cannot do so, decrease the intensity of activity.
- Stop the activity if severe shortness of breath, pain, or dizziness develops.

Adapted from Piña, I. L. (2019). Cardiac rehabilitation in patients with heart failure. *UpToDate*. Retrieved on 9/11/2019 at: [www.uptodate.com/contents/cardiac-rehabilitation-in-patients-with-heart-failure](http://www.uptodate.com/contents/cardiac-rehabilitation-in-patients-with-heart-failure)

Barriers to performing activities are identified, and methods of adjusting an activity are discussed. For example, vegetables can be chopped or peeled while sitting at the kitchen table rather than standing at the kitchen counter. Small, frequent meals decrease the amount of energy needed for digestion while providing adequate nutrition. The nurse helps the patient identify peak and low periods of energy, planning energy-consuming activities for peak periods. For example, the patient may prepare the meals for the entire day in the morning. Pacing and prioritizing activities help maintain the patient's energy to promote participation in regular physical activity.

The patient's response to activities needs to be monitored. If the patient is hospitalized, vital signs and oxygen saturation levels are monitored before, during, and immediately after an activity to identify whether they are within the desired range. Heart rate should return to baseline within 3 min following the activity. If the patient is at home, the degree of fatigue felt after the activity can be used to assess the response. If the patient tolerates the activity, short- and long-term goals can be developed to gradually increase the intensity, duration, and frequency of activity.

Adherence to exercise training is essential if the patient is to benefit from it, but it may be difficult for patients with other comorbid conditions (e.g., arthritis, anemia, cardiomyopathy, obesity, chronic kidney disease, chronic obstructive pulmonary disease) and those who have had HF for a longer time (Cattadori, Segurini, Picozzi, et al., 2018). Referral to a cardiac rehabilitation program may be indicated, especially for patients newly diagnosed with HF (Piña, 2019). A supervised program may also benefit those who need a structured environment, significant educational support, regular encouragement, and interpersonal contact.

#### **MANAGING FLUID VOLUME**

Patients with severe HF may receive IV diuretic therapy; however, patients with less severe symptoms are typically prescribed oral diuretics. Oral diuretics should be given early in the morning so that diuresis does not interfere with the patient's nighttime rest. Discussing the timing of medication administration is especially important for older patients who may have urinary urgency or incontinence. A single dose of a diuretic may cause the patient to excrete a large volume of fluid shortly after its administration.

The patient's fluid status is monitored closely by auscultating the lungs, monitoring daily body weight, and assisting the patient to adhere to a low sodium diet by reading food labels and avoiding high sodium foods such as canned, processed, and convenience foods ([Chart 25-4](#)). Weight gain in a patient with HF almost always reflects fluid retention. If the diet includes fluid restriction, the nurse can assist the patient to plan fluid intake throughout the day while respecting the patient's dietary preferences. If the patient is receiving IV fluids and medications, the amount of fluid needs to be monitored closely, and the primary provider or pharmacist can be consulted about the possibility of maximizing the amount of medication in the same volume of IV fluid (e.g., double concentrating to decrease the fluid volume given).

The patient is positioned or taught how to assume a position that facilitates breathing. The number of pillows may be increased, the head of the bed may be elevated, or the patient may sit in a recliner. In these positions, the venous return to the heart (preload) is reduced, pulmonary congestion is reduced, and pressure on the diaphragm is minimized. The

lower arms can be supported with pillows to eliminate the fatigue caused by the pull of the patient's weight on the shoulder muscles. If the patient is experiencing acute decompensation, positioning them upright, preferably with the legs dangling over the side of the bed, has the immediate effect of decreasing venous return, decreasing right ventricular stroke volume, and decreasing lung congestion.

**Chart 25-4**  **HEALTH PROMOTION**

## Facts About Dietary Sodium

Although the major source of sodium in the average American diet is salt, many types of natural foods contain varying amounts of sodium. Even if no salt is added in cooking and if salty foods are avoided, the daily diet will still contain about 2000 mg of sodium. Fresh fruits and vegetables are low in sodium and should be encouraged.

## Additives in Food

In general, food prepared at home is lower in sodium than restaurant or processed foods. Added food substances (additives), such as sodium alginate, which improves food texture, sodium benzoate, which acts as a preservative, and disodium phosphate, which improves cooking quality in certain foods, increase the sodium intake when included in the daily diet. Therefore, patients on low sodium diets should be advised to check labels carefully for words such as "salt" or "sodium," especially on canned foods. For example, without looking at the sodium content per serving found on the nutrition labels, when given a choice between a serving of potato chips and a cup of canned cream of mushroom soup, most would think that soup is lower in sodium. However, when the labels are examined, the lower sodium choice is found to be the chips. Although potato chips are *not* recommended in a low sodium diet, this example illustrates that it is important to read food labels to determine both sodium content and serving size.

## Nonfood Sodium Sources

Sodium is contained in municipal water. Water softeners also increase the sodium content of drinking water. Patients on sodium-restricted diets should be cautioned against using nonprescription medications such as antacids, cough syrups, and laxatives. Salt substitutes may be allowed, but it is recognized that they are high in potassium. Over-the-counter medications should not be used without first consulting the patient's primary provider.

## Promoting Dietary Adherence

If patients find food unpalatable because of the dietary sodium restrictions and/or the taste disturbances caused by the medications, they may refuse to eat or to follow the dietary regimen. For this reason, severe sodium restrictions should be avoided, and diuretic medication should be balanced with the patient's ability to restrict dietary sodium. A variety of flavorings, such as lemon juice, vinegar, and herbs, may be used to improve the taste of the food and facilitate acceptance of the diet. It is important to consider the patient's food preferences. Diet counseling and educational handouts can be geared toward a patient–family-centered approach and with cultural practices considered.

Adapted from American Heart Association (AHA). (2016). Shaking the salt habit. Retrieved on 10/24/2019 at:  
[www.heart.org/HEARTORG/Conditions/HighBloodPressure/PreventionTreatmentofHighBloodPressure/Shaking-the-Salt-Habit\\_UCM\\_303241\\_Article.jsp#.Vzy9eNe3BK8](http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/PreventionTreatmentofHighBloodPressure/Shaking-the-Salt-Habit_UCM_303241_Article.jsp#.Vzy9eNe3BK8)

Because decreased circulation in edematous areas increases the risk of pressure injuries, the nurse assesses for skin breakdown and institutes preventive measures. Positioning to avoid pressure and frequent changes of position help prevent pressure injuries.

#### CONTROLLING ANXIETY

Patients with HF may exhibit signs and symptoms of anxiety. In addition to psychosocial sources of anxiety, the physiologic compensatory mechanisms include activation of neurohormones including catecholamines. Complex medical interventions, such as implantation of an ICD can provoke anxiety in patients and families. These sources of anxiety include living with the threat of shocks, role changes, and concerns about the patient's ability to carry out activities of daily living. The patient's anxiety may intensify at night and interfere with sleep. Emotional stress further stimulates the sympathetic nervous system, causing vasoconstriction, elevated arterial pressure, and increased heart rate. This sympathetic response increases cardiac workload.

When the patient exhibits anxiety, the nurse takes steps to promote physical comfort and provide psychological support. As mentioned previously, the patient may be more comfortable sitting in a recliner. Oxygen may be given during an acute event to diminish the work of breathing and increase the patient's comfort. In many cases, a family member's presence provides reassurance. Patients with HF rely on their families for many aspects of care; therefore, nurses should assess the needs of family caregivers and provide support to them (Hodson, Peacock, & Holtslander, 2019).

Along with reassurance, the nurse can begin educating the patient and family about techniques for controlling anxiety and avoiding anxiety-provoking situations. This includes how to identify factors that contribute to anxiety and how to use relaxation techniques to control anxious feelings. As the patient's anxiety decreases, cardiac function may improve and symptoms of HF may decrease.



#### Quality and Safety Nursing Alert

*When patients with HF are delirious, confused, or anxious, restraints should be avoided. Restraints are likely to be resisted, and resistance inevitably increases the cardiac workload.*

### **MINIMIZING POWERLESSNESS**

Patients with HF may feel overwhelmed with their diagnosis and treatment regimen, leading to feelings of powerlessness. Contributing factors may include lack of knowledge and lack of opportunity to make decisions, particularly if health care providers or family members do not encourage the patient to participate in the treatment decision-making process.

Nurses should help patients recognize their choices, and that they can positively influence the outcomes of their diagnosis and treatment. Taking time to listen actively to patients encourages them to express their concerns and ask questions. Other strategies include providing the patient with decision making opportunities, such as when activities are to occur, or encouraging food and fluid choices consistent with the dietary restrictions. Encouragement is provided, progress is identified, and the patient is assisted to differentiate between factors that can and cannot be controlled.

In addition to feelings of powerlessness, patients with HF have a high incidence of depressive symptoms, which are associated with increased morbidity and mortality (Jiang et al., 2018). Because depressive symptoms are known to increase as the disease worsens, patients with HF need to be screened for depression so that it can be treated, hopefully maintaining the patient's functional status and quality of life.

### **ASSISTING PATIENTS AND FAMILIES TO EFFECTIVELY MANAGE HEALTH**

Therapeutic regimens for HF are complex and require the patient and family to make significant lifestyle changes. An inability or unwillingness to adhere to dietary and pharmacologic recommendations can lead to episodes of acute decompensated HF and hospitalization. Nonadherence with prescribed diet and fluid restrictions and medications cause many hospital readmissions. Nursing research findings suggest that for some patients with HF who are taking more than one medication daily, the decision to not take HF medications as prescribed may actually reflect their efforts to best manage their personal health, and, therefore, may be a self-care strategy (see the Nursing Research Profile in [Chart 25-5](#)) (Meraz, 2020).

Nurses have a key role in managing episodes of acute decompensated HF and in developing a comprehensive education and discharge plan to prevent hospital readmissions and increase the patient's quality of life. Because of the high cost of hospitalization for HF, the Centers for Medicare & Medicaid Services (CMS) initiated a program that reduces reimbursement to hospitals with a high 30-d readmission rate (U.S. Department of Health and Human Services [HHS], 2019). Research continues to identify the most effective interventions that may decrease these rates. A number of evidence-based components are known to increase the effectiveness of a discharge plan for patients with HF, including providing them with comprehensive, patient-centered instructions,

scheduling follow-up visits with their primary providers within 7 d of discharge, and following up by telephone within 3 d of discharge (Yancy et al., 2013; Yancy et al., 2016; Yancy et al., 2017).

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Because HF is a complex and progressive condition, patients are at risk for many complications, including acute decompensated HF and pulmonary edema; hypotension and cardiogenic shock (see Chapter 11); arrhythmias (see Chapter 22); thromboembolism formation (see Chapter 26); and pericardial effusion (see later discussion).



**Pulmonary Edema.** As described previously, pulmonary edema is associated with acute decompensated HF that can lead to acute respiratory failure and death. If it is recognized early, pulmonary edema may be alleviated by increasing dosages of diuretics and by implementing other interventions to decrease preload. For instance, placing the patient in an upright position with the feet and legs dependent reduces left ventricular workload. The treatment regimen and the patient's understanding of and adherence to it are assessed. The long-range approach for preventing pulmonary edema must be directed at identifying and managing its precipitating factors.

**Chart 25-5**



#### **NURSING RESEARCH PROFILE**

## **Medication Nonadherence or Self-Care?**

Meraz, R. (2020). Medication nonadherence or self-care? Understanding the medication decision-making process and experiences of older adults with heart failure. *Journal of Cardiovascular Nursing*, 35(1), 26–34.

### **Purpose**

It is estimated that over half of patients with heart failure do not take their medications as prescribed. The reasons why many patients with heart failure do not adhere to their prescribed medication regimen are elusive, particularly since nonadherence is associated with hospitalization and emergency department visits. Therefore, the purpose of this study was to discover why patients with heart failure might not take their heart failure medications as prescribed.

### **Design**

This was a qualitative study that used narrative inquiry and storytelling to discover why community dwelling patients with heart failure might not adhere to their medication regimen. Eleven participants, all 65 y of age or older, volunteered to be interviewed. Each of these participants had to live independently in the community setting, had to take at least two medications daily to treat heart failure, and had to self-administer these medications in order to be eligible to participate in this study.

### **Findings**

Results from participant interviews found that patients' nonadherence with the prescribed medication regimen was deliberate, and not because of forgetfulness. In all reported instances, it was not contingent upon financial constraints, either. Some participants stopped taking a medication while others adjusted the dosages. Participants' deliberate decisions to adjust their medication regimen tended to be made based on the belief that they knew their own bodies and needs best, and felt that they were best equipped to take care of their own needs. Others noted that at times it was too difficult to get in contact with their primary providers or nurses, and that their primary providers or nurses were too busy to discuss their medication regimen with them. Most noted that they researched online their medications' effects and felt competent to manage their medication dosage without necessarily consulting their providers. Paradoxically, participants did not view their decisions to self-adjust their prescribed medication regimens as consonant with nonadherence.

### **Nursing Implications**

Patients with heart failure who are prescribed multiple medications to treat their heart failure may not view self-adjustment of their prescriptions as tantamount to nonadherence. It seems likely that these self-

care/nonadherent practices place these patients at risk for hospitalization and morbid complications. Participants in this study noted that one key reason that they self-adjusted their medications was because their primary providers and nurses were “too busy” to talk to them about their medications. Nurses are in an ideal position to talk to patients with heart failure about their prescriptions, discover their concerns about their medications, help them find ways to problem-solve and manage side effects, and help them proactively engage in self-care so that they avoid hospitalization.

Clinical management of a patient with acute pulmonary edema due to left ventricular failure is directed toward reducing volume overload, improving ventricular function, and increasing oxygenation. These goals are accomplished through a combination of oxygen and ventilatory support, IV medication, and nursing assessment and interventions.

The patient’s airway and breathing are assessed to determine the severity of respiratory distress, along with vital signs. The patient is placed on pulse oximetry, a cardiac monitor, and IV access is confirmed or established for administration of medications. Laboratory tests are obtained, including arterial blood gases, electrolytes, BUN, and creatinine; other laboratory tests that may be indicated include a complete blood cell count (CBC), BNP, or a serum troponin-I. A chest x-ray or an ultrasound of the lungs may be obtained to confirm the extent of pulmonary edema (Meyer, 2019a).

Oxygen is given in concentrations adequate to relieve hypoxemia and dyspnea; a non-rebreathing mask may be used initially. If respiratory failure is severe or persists, noninvasive positive-pressure ventilation is the preferred mode of assisted ventilation (Colucci, 2019). For some patients, endotracheal (ET) intubation and mechanical ventilation are required. The ventilator can provide positive end-expiratory pressure (PEEP), which is effective in reducing venous return, decreasing fluid movement from the pulmonary capillaries to the alveoli, and improving oxygenation (see Chapter 19). Oxygenation is monitored by pulse oximetry and by measurement of arterial blood gases.

The patient who is experiencing pulmonary edema is likely going to be highly anxious, as are the patient’s family members. As the ability to breathe decreases, the patient’s fear and anxiety rise proportionately, making the condition more severe. Reassuring the patient and family and providing skillful anticipatory nursing care are integral parts of the therapy. Because the patient is in an unstable condition, the nurse must remain with the patient. The nurse gives the patient simple, concise information in a reassuring voice about what is being done to treat the condition and the expected results.

Vasodilators such as IV nitroglycerin or nitroprusside may enhance symptom relief in pulmonary edema, as previously described (Meyer,

2019a). Blood pressure is continually assessed in patients receiving IV vasodilator infusions.

Furosemide or another loop diuretic is given by IV push or as a continuous infusion to produce a rapid diuretic effect. The blood pressure is closely monitored as the urine output increases, because it is possible for the patient to become hypotensive as intravascular volume decreases. The patient receiving diuretic therapy may excrete a large volume of urine within minutes after a potent diuretic is given. A bedside commode may be used to decrease the energy required by the patient and to reduce the resultant increase in cardiac workload induced by getting on and off a bedpan. If necessary, in order to carefully monitor urine output, an indwelling urinary catheter may be inserted.

Once the patient is stable, they may transition to oral diuretics; intake and output, daily weights, serum electrolytes, and creatinine are carefully monitored.

Many potential problems associated with HF therapy relate to the use of diuretics. These problems require ongoing nursing assessment and collaborative intervention:

Excessive and repeated diuresis can lead to hypokalemia (i.e., potassium depletion). Signs include ventricular arrhythmias, hypotension, muscle weakness, and generalized weakness. In patients receiving digoxin, hypokalemia can lead to digitalis toxicity, which increases the likelihood of dangerous arrhythmias. Patients with HF may also develop low levels of magnesium, which can add to the risk of arrhythmias. Hyperkalemia may occur, especially with the use of ACE inhibitors, ARBs, or spironolactone. Hyperkalemia can also lead to profound bradycardia and other arrhythmias. Prolonged diuretic therapy may produce hyponatremia (deficiency of sodium in the blood), which can result in disorientation, weakness, muscle cramps, and anorexia. Volume depletion from excessive fluid loss may lead to dehydration and hypotension. ACE inhibitors and beta-blockers may contribute to the hypotension. Other problems associated with diuretics include increased serum creatinine (indicative of renal dysfunction) and hyperuricemia (excessive uric acid in the blood), which leads to gout.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**

Facilitating an easy and smooth transition back into the community is of paramount importance for the patient living with HF. Community-based nursing interventions for patients with HF can improve health outcomes and health care value as evidenced by reduced hospital readmissions for up to 6 mo. Important nursing interventions that keep the patient with HF out of the hospital incorporate a multidisciplinary care planning approach (Jones, Bowles, Richard, et al., 2017).



### Educating Patients About Self-Care.

The nurse provides patient education and involves the patient and family in the therapeutic regimen to promote understanding and adherence to the plan. When the patient recognizes that the diagnosis of HF can be successfully managed with lifestyle changes and medications, recurrences of acute HF lessen, unnecessary hospitalizations decrease, and life expectancy increases. Nurses play a key role in educating patients and their families about medication management, a low sodium diet, moderate alcohol consumption, activity and exercise recommendations, smoking cessation, how to recognize the signs and symptoms of worsening HF, and when to contact the primary provider (Jones et al., 2017). Use of the teach-back technique to assess the patient's comprehension of the instructions can increase education effectiveness and prevent rehospitalization (Esquivel, White, Carroll, et al., 2018) (see Chapter 3 for further discussion of teach-back methods). In order for teach-back to be effective, the nurse must ensure adequate time is dedicated to ensuring that patient learning occurs (Esquivel et al., 2018). A basic home education plan for the patient with HF is presented in [Chart 25-6](#). The patient should receive a written copy of the instructions.

The patient's readiness to learn and potential barriers to learning are assessed. Patients with HF may have temporary or ongoing cognitive impairment due to their illness or other factors, increasing the need to rely on an identified caretaker (Hodson et al., 2019). An effective treatment plan incorporates both the patient's goals and those of the health care providers. The nurse must consider cultural factors and adapt the education plan accordingly. Patients and families need to understand that effective HF management is influenced by choices made about treatment options and their ability to follow the treatment plan. They also need to be informed that health care providers are available to assist them in reaching their health care goals.

**Chart 25-6**



### HOME CARE CHECKLIST

## The Patient with Heart Failure

**At the completion of education, the patient and/or caregiver will be able to:**

- Identify heart failure as a chronic disease that can be managed with medications and specific self-management behaviors.
- State the impact of heart failure on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Take or administer medications daily, exactly as prescribed.
- Monitor effects of medication such as changes in breathing and edema.
- Know signs and symptoms of orthostatic hypotension and how to prevent it.
- Weigh self daily at the same time, with same clothes.
- Restrict sodium intake to no more than 2 g/day:
  - Adapt diet by examining nutrition labels to check sodium content per serving.
  - Avoid canned or processed foods, eating fresh or frozen foods.
  - Consult the written diet plan and the list of permitted and restricted foods.
  - Avoid salt use.
  - Avoid excesses in eating and drinking.
- Participate in prescribed activity program:
  - Participate in a daily exercise program.
  - Increase walking and other activities gradually, provided they do not cause unusual fatigue or dyspnea.
  - Conserve energy by balancing activity with rest periods.
  - Avoid activity in extremes of heat and cold, which increase the work of the heart.
  - Recognize that air-conditioning may be essential in a hot, humid environment.
- Develop methods to manage and prevent stress:
  - Avoid tobacco.
  - Avoid alcohol.
  - Engage in social and diversional activities.
- Identify community resources for peer and caregiver/family support:
  - Identify sources of support (e.g., friends, relatives, faith community).
  - Identify the contact details for support services for patients and their caregivers/families.

- Report immediately to the primary provider or clinic any of the following:
  - Gain in weight of 2–3 lb (0.9–1.4 kg) in 1 d, or 5 lb (2.3 kg) in 1 wk
  - Unusual shortness of breath with activity or at rest
  - Increased swelling of ankles, feet, or abdomen
  - Persistent cough
  - Loss of appetite
  - Development of restless sleep; increase in number of pillows needed to sleep
  - Profound fatigue
- State how to reach primary provider with questions or complications:
  - State time and date of follow-up appointments and diagnostic tests.
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

**Continuing and Transitional Care.** Successful management of HF requires adherence to a complex medical regimen that includes multiple lifestyle changes for most patients. Assistance may be provided through a number of options that optimize evidence-based recommendations for effective management of HF. Depending on the patient's physical status and the availability of family assistance, a home care referral or another type of disease management program may be indicated for a patient who has been hospitalized. Transitional care programs (hospital to home) that include telephone contact along with home visits have been shown to decrease rehospitalizations and increase patient quality of life (Cyrille & Patel, 2017; Jones et al., 2017). Home visits by nurses who are specially trained in managing patients with HF provide assessment and management tailored to specific individualized patient needs. Older patients and those who have long-standing heart disease with compromised physical stamina often require assistance with the transition to home after hospitalization for an acute episode of HF. The home health nurse assesses the physical environment of the home and makes suggestions for adapting the home environment to meet the patient's activity limitations. If stairs are a concern, the patient can plan the day's activities so that stair-climbing is minimized; for some patients, a temporary bedroom may be set up on the main level of the home. The home health nurse works with the patient and family to maximize the benefits of these changes.

The home health nurse also reinforces and clarifies information about dietary changes and fluid restrictions, the need to monitor symptoms and

daily body weight, and the importance of obtaining follow-up care with the primary provider's office or clinic. Assistance may be given in scheduling and keeping appointments as well. The patient is encouraged to gradually increase their self-care and responsibility for carrying out the therapeutic regimen.

Evidence-based HF guidelines also recommend patient referral to HF clinics, which provide intensive nursing management along with medical care in a collaborative model. Many of these clinics are managed by advanced practice nurses. Referral to an HF clinic gives the patient ready access to continuing education, professional nursing and medical staff, and timely adjustments to treatment regimens. HF clinics can also provide outpatient treatment (e.g., IV diuretics, laboratory monitoring) as an alternative to hospitalization. Because of the additional support and coordination of care, patients managed through HF clinics have fewer exacerbations of HF, fewer hospitalizations, decreased costs of medical care, and increased quality of life (Yancy, 2013).

Other disease management programs are carried out through telehealth, using telephones or computers to maintain contact with patients and to obtain patient data. This enables nurses and others to assess and manage patients on a frequent basis, without requiring patients to make frequent visits to health care providers. A variety of techniques ranging from simple telephone monitoring to sophisticated computer and video connections that monitor symptoms, daily weight, vital signs, heart sounds, and breath sounds may be used. Patient data may also include hemodynamics and other parameters transmitted from implantable devices. Studies have shown that telehealth management can decrease costs and hospitalizations for acute exacerbations of HF (Koehler, Koehler, Deckwart, et al., 2018).

**End-of-Life Considerations.** Because HF is a chronic and often progressive condition, patients and families need to consider issues related to the end-of-life and when palliative or hospice care should be considered (Cross, Kamal, Taylor, et al., 2019). Although the prognosis in patients with HF may be uncertain, issues often arise sooner or later related to the patient's thoughts and possible concerns about the use of complex treatment options (e.g., implantation of an ICD or a ventricular assist device [VAD]). VADs are an option for some patients with HF who have failed medical therapy and who are not candidates for cardiac transplantation. Discussions concerning the use of technology, preferences for end-of-life care, and advance directives should take place while the patient is able to participate and express preferences. For example, with the expanded use of ICDs in the HF population, patients with ICDs, their families, and their primary providers should receive instructions for ICD inactivation at the end-of-life to prevent inappropriate discharges. See Chapter 13 for further discussion of end-of-life care.

## Evaluation

Expected patient outcomes may include:

1. Demonstrates tolerance for desired activity
  - a. Describes adaptive methods for usual activities
  - b. Schedules activities to conserve energy and reduce fatigue and dyspnea
  - c. Maintains heart rate, blood pressure, respiratory rate, and pulse oximetry within the targeted range
2. Maintains fluid balance
  - a. Exhibits decreased peripheral edema
  - b. Verbalizes understanding of fluid intake and diuretic use
3. Decreased anxiety
  - a. Avoids situations that produce stress
  - b. Sleeps comfortably at night
  - c. Reports decreased stress and anxiety
  - d. Denies symptoms of depression
4. Makes sound decisions regarding care and treatment
  - a. Demonstrates ability to influence outcomes
5. Patients and family members adhere to healthy regimen
  - a. Performs and records daily weights
  - b. Limits dietary sodium intake to no more than 2 g/day
  - c. Takes medications as prescribed
  - d. Reports symptoms of worsening HF
  - e. Makes and keeps appointments for follow-up care
6. Exhibits no evidence of acute decompensation and pulmonary edema
7. Denies dyspnea
8. No apparent delirium or acute anxiety
9. Maintains fluid balance as noted previously
10. No evidence of electrolyte disturbances from diuretic therapy

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## COMPLICATIONS FROM HEART DISEASE

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### Cardiogenic Shock

Cardiogenic shock occurs when decreased CO leads to inadequate tissue perfusion and initiation of the shock syndrome. Cardiogenic shock most commonly occurs following acute MI when a large area of myocardium

becomes ischemic and hypokinetic. It also can occur as a result of end-stage HF, cardiac tamponade, pulmonary embolism (PE), cardiomyopathy, and arrhythmias. Cardiogenic shock is a life-threatening condition with a high mortality rate. (See Chapter 11 for detailed information about the pathophysiology and management of cardiogenic shock.)

## Thromboembolism

Patients with cardiovascular disorders are at risk for the development of arterial thromboemboli and venous thromboemboli (VTE). Intracardiac thrombi can form in patients with atrial fibrillation because the atria do not contract forcefully, resulting in slow and turbulent flow, and increasing the likelihood of thrombus formation. Mural thrombi can also form on ventricular walls when contractility is poor. Intracardiac thrombi can break off and travel through the circulation to other structures, including the brain, where they cause a stroke. Clots within the cardiac chambers can be detected by an echocardiogram and treated with anticoagulant agents, such as heparin and warfarin (see Chapter 26 for further discussion of assessment and treatment of VTEs; [Table 26-2](#) discusses specific anticoagulant medications).

Decreased mobility and other factors in patients with cardiac disease also can lead to clot formation in the deep veins of the legs. Although signs and symptoms of deep vein thrombosis (DVT) can vary, patients may report leg pain and swelling and the leg may appear erythematous and feel warm. These clots can break off and travel through the inferior vena cava and through the right side of the heart into the pulmonary artery, where they can cause a pulmonary embolus (PE) (see Chapter 26 for further discussion of assessment and treatment of PE).

## Pericardial Effusion and Cardiac Tamponade

Pericardial effusion (accumulation of fluid in the pericardial sac) may accompany advanced HF, pericarditis, metastatic carcinoma, cardiac surgery, or trauma. Normally, the pericardial sac contains about 20 mL of fluid, which is needed to decrease friction for the beating heart. An increase in pericardial fluid raises the pressure within the pericardial sac and compresses the heart. This has the following effects:

- Elevated pressure in all cardiac chambers
- Decreased venous return due to atrial compression
- Inability of the ventricles to distend and fill adequately

Pericardial fluid may build up slowly without causing noticeable symptoms until a large amount (1 to 2 L) accumulates (Hoit, 2019). However, a rapidly

developing effusion (e.g., hemorrhage into the pericardial sac from chest trauma) can quickly stretch the pericardium to its maximum size and cause an acute problem. As pericardial fluid increases, pericardial pressure increases, reducing venous return to the heart and decreasing CO. This can result in cardiac tamponade, which causes low CO and obstructive shock.

## Clinical Manifestations

The signs and symptoms of pericardial effusion can vary according to whether the problem develops quickly or slowly. In acute cardiac tamponade, the patient suddenly develops chest pain, tachypnea, and dyspnea. JVD results from poor right atrial filling and increased venous pressure. Hypotension occurs from low CO, and heart sounds are often muted. The subacute presentation of a pericardial effusion is less dramatic. The patient may report chest discomfort or a feeling of fullness. The feeling of pressure in the chest may result from stretching of the pericardial sac. These patients also develop dyspnea, JVD, and hypotension over time (Hoit, 2019). Patients with cardiac tamponade typically have tachycardia in response to low CO. In addition to hypotension, patients with cardiac tamponade may develop **pulsus paradoxus**, a systolic blood pressure that is markedly lower during inhalation. Also known as paradoxical pulse, this finding is characterized by an abnormal difference of at least 10 mm Hg in systolic pressure between the point that it is heard during exhalation and the point that it is heard during inhalation. This difference is caused by the variation in cardiac filling that occurs with changes in intrathoracic pressure during breathing. The cardinal signs of cardiac tamponade are illustrated in [Figure 25-4](#).

## Assessment and Diagnostic Findings

An echocardiogram is performed to confirm the diagnosis and quantify the amount of pericardial fluid. A chest x-ray may show an enlarged cardiac silhouette due to pericardial effusion. The ECG shows tachycardia and may also show low voltage (Hoit, 2019). See Chapter 22 for discussion of the significance of ECG abnormalities.

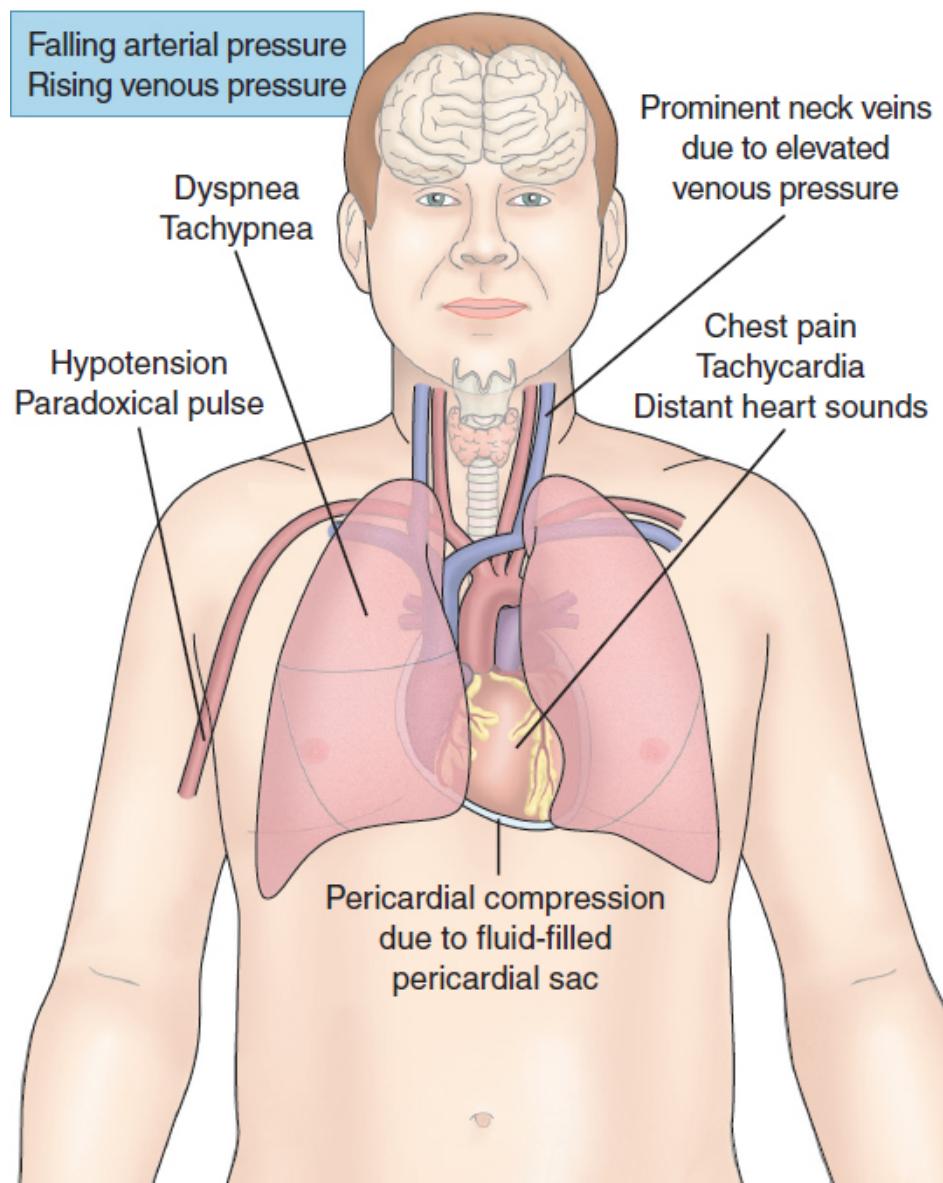
## Medical Management

Acute management of cardiac tamponade may include a pericardiocentesis; whereas, recurrent effusions may be managed with a pericardiectomy.

### Pericardiocentesis

If cardiac function becomes seriously impaired, **pericardiocentesis** (puncture of the pericardial sac to aspirate pericardial fluid) is performed. During this

procedure, the patient is monitored by continuous ECG and frequent vital signs. Catheter pericardiocentesis is performed using echocardiography to guide placement of the drainage catheter (Hoit, 2019).



**Figure 25-4 •** Assessment findings in cardiac tamponade resulting from pericardial effusion include chest pain or fullness, dyspnea, tachypnea, jugular vein distention, hypotension, paradoxical pulse, tachycardia, and distant heart sounds.

A resulting decrease in central venous pressure and an associated increase in blood pressure after withdrawal of pericardial fluid indicate that the cardiac tamponade has been relieved. The patient almost always feels immediate relief. If there is a substantial amount of pericardial fluid aspirated, a small

catheter may be left in place to drain recurrent accumulation of blood or fluid. Pericardial fluid is sent to the laboratory for examination for tumor cells, bacterial culture, chemical and serologic analysis, and differential blood cell count.

Complications of pericardiocentesis include coronary artery puncture, myocardial trauma, arrhythmias, pleural laceration, and gastric puncture. After pericardiocentesis, the patient's heart rhythm, blood pressure, venous pressure, and heart sounds are monitored frequently to detect possible recurrence of cardiac tamponade. A follow-up echocardiogram is also performed. If the effusion recurs, repeat aspiration is necessary. Cardiac tamponade may require treatment by open surgical drainage (pericardiotomy) (Hoit, 2019).

### **Pericardiotomy**

Recurrent pericardial effusions, usually associated with neoplastic disease, may be treated by a **pericardiotomy** (pericardial window). Under general anesthesia, a portion of the pericardium is excised to permit the exudative pericardial fluid to drain into the lymphatic system. The nursing care following the procedure includes routine postsurgical care (see Chapter 16) in addition to observation for recurrent tamponade.

## **Cardiac Arrest**

In cardiac arrest, the heart is unable to pump and circulate blood to the body's organs and tissues. It is often caused by an arrhythmia such as ventricular fibrillation, progressive bradycardia, or asystole (i.e., absence of cardiac electrical activity and heart muscle contraction). Cardiac arrest can also occur when electrical activity is present on the ECG but cardiac contractions are ineffective, a condition called **pulseless electrical activity (PEA)**. PEA may be caused by a variety of problems such as profound hypovolemia (e.g., hemorrhage). Diagnoses that are commonly associated with cardiac arrest include MI, massive pulmonary emboli, hyperkalemia, hypothermia, severe hypoxia, and medication overdose. Rapid identification of these problems and prompt intervention can restore circulation in some patients.

## **Clinical Manifestations**

In cardiac arrest, consciousness, pulse, and blood pressure are lost immediately. Breathing usually ceases, but ineffective respiratory gasping may occur. The pupils of the eyes begin dilating in less than a minute, and seizures may occur. Pallor and cyanosis are seen in the skin and mucous membranes. The risk of organ damage, including irreversible brain damage, and of death increases with every minute that passes. A patient's age and overall health

determine their vulnerability to irreversible damage. As soon as possible, the diagnosis of cardiac arrest must be made and action taken immediately to restore circulation.



## Emergency Assessment and Management: Cardiopulmonary Resuscitation

Cardiopulmonary resuscitation (CPR) provides blood flow to vital organs until effective circulation can be reestablished. Following the recognition of unresponsiveness, a protocol for basic life support is initiated. The AHA Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care direct the current protocols for CPR, medical emergency teams, postcardiac arrest care, and acute respiratory compromise. The primary goal of resuscitation protocols is to save lives by preventing in-hospital cardiac arrest and optimizing outcomes (AHA, 2017).

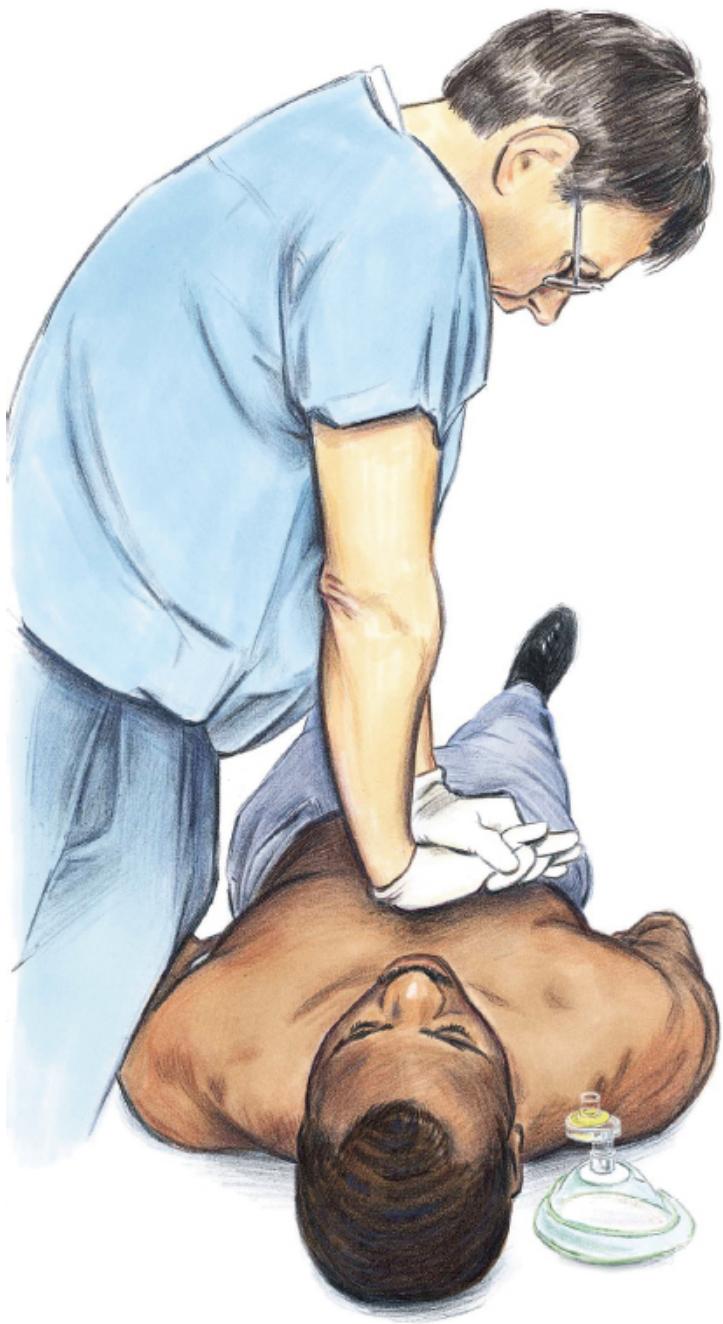
The resuscitation process begins with the immediate assessment of the patient for breathing and consciousness, then a call for assistance, as CPR can be performed most effectively with the addition of more health care providers and equipment (e.g., defibrillator). The current resuscitation protocol recommends the following practices for CPR:

1. *Quick recognition of sudden cardiac arrest.* The patient is assessed for responsiveness and breathing.
2. *Activation of the Emergency Response System.* Within a medical facility, a call is made to alert the emergency response team. Outside of a medical facility, 911 is called to activate the Emergency Medical Service (EMS).
3. *Performance of high-quality CPR.* If there is no carotid pulse detected, chest compressions are initiated at a rate of 100 bpm.
4. *Rapid cardiac rhythm analysis and defibrillation* within 2 minutes for patients in ventricular fibrillation or pulseless ventricular tachycardia, followed by continuous chest compressions.

Rescue breathing is no longer recommended unless health care providers are present; if that is the case, it is then started after chest compressions. The airway is opened using a head-tilt/chin-lift maneuver, and any obvious material in the mouth or throat is removed. An oropharyngeal airway may be inserted if available to help maintain patency of the airway. Rescue ventilations are provided using a bag-valve mask or mouth-mask device. Oxygen is given at 100% during resuscitation to correct hypoxemia and improve tissue oxygenation.

Compressions are performed with the patient on a firm surface such as the floor or a cardiac board. The provider, facing the patient's side, places one hand in the center of the chest on the lower half of the sternum and positions the other hand on top of the first hand (Fig. 25-5). The chest is compressed 2

inches (approximately 5 cm) at a rate of 100 compressions/min. Complete recoil of the chest must be allowed between compressions to allow for cardiac filling. Interruptions in CPR to switch providers or check for a pulse are minimized (Panchal, Berg, Hirsch, et al., 2019). It is recommended providers switch every 2 minutes due to the exertion of delivering effective compressions.



**Figure 25-5 •** Chest compressions in cardiopulmonary resuscitation are performed by placing the heel of one hand in the center of the chest over the sternum and the other hand on top of the first hand. Elbows are kept straight and body weight is used to apply forceful compressions to the lower sternum. The patient should be on a hard surface such as a cardiac board. Reprinted with permission from Field, J. M., Kudenchuk, P. J., O'Connor, R. E., et al. (2009). *The textbook of emergency cardiovascular care and CPR*. Philadelphia, PA: Lippincott Williams & Wilkins.

## Defibrillation

As soon as a monitor/defibrillator is available, monitor electrodes are applied to the patient's chest and the heart rhythm is analyzed. When an automated external defibrillator (AED) is used, the device is turned on, the pads are applied to the patient's chest, and the rhythm is analyzed by the defibrillator to determine whether a shock is indicated. When the ECG shows ventricular fibrillation or pulseless ventricular tachycardia, immediate defibrillation is the treatment of choice.

The AHA (2017) recommends the first defibrillation to occur within 2 minutes of the first documented, pulseless rhythm. Survival time decreases for every minute that defibrillation is delayed. Following defibrillation, high-quality chest compressions are resumed immediately. Survival after cardiac arrest has been improved by extensive education of health care providers and by the use of AEDs.

## Advanced Cardiovascular Life Support

During a resuscitation, an advanced airway (e.g., endotracheal tube, tracheal tube) may be placed by a primary provider, nurse anesthetist, or respiratory therapist to ensure a patent airway and adequate ventilation. Following confirmation of the placement of airway (auscultation of breath sounds, observation of equal chest expansion, or a carbon dioxide detector), positive-pressure ventilation should be delivered without pausing chest compressions at a rate of one breath every 6 seconds, or 10 breaths/min (Kleinman, Goldberger, Rea, et al., 2017; Panchal et al., 2019).

Specific subsequent advanced support interventions depend on the assessment of the patient's condition and response to therapy. For example, if asystole is detected on the monitor, CPR is continued while IV or intraosseous (IO) epinephrine is given. Additional medications ([Table 25-4](#)) may be indicated for the patient during and after resuscitation.

Each person on an effective Advanced Cardiac Life Support (ACLS) team, called a *CPR team* or sometimes a *code team*, has delineated roles. An efficient code team is characterized by individual members who are knowledgeable about their position and responsibilities. This ensures direct and clear lines of communication, effective team work, and a safe environment for the health care team and the patient (Panchal et al., 2019).

It is recommended practice to support family members of all patients who have had cardiac arrest and are undergoing resuscitation. In addition, there is clear support to have them present at the bedside if that is consonant with the patient's wishes. Written policies and procedures to support family presence during this time ought to be readily available for nurses and staff to easily access. Studies demonstrate family presence does not disrupt patient care, has no negative outcomes during the resuscitation event, and results in no adverse

psychological effects (American Association of Critical-Care Nurses [AACN], 2016).

CPR is stopped when vital signs are detected or the patient responds. If the patient does not respond to interventions, the resuscitation effort may be stopped by the code team leader or other provider in charge of the resuscitation after options have been exhausted. Many factors are considered in the decision, such as the initiating arrhythmia, potential etiology, length of time for initiation of life support, the patient's response to treatment, and the patient's overall clinical status.

**TABLE 25-4** Medications Used in Cardiopulmonary Resuscitation

Agent and Action	Indications	Nursing Considerations
Epinephrine—vasopressor used to optimize BP and cardiac output; improves perfusion and myocardial contractility	Given to patients in cardiac arrest caused by asystole, pulseless electrical activity, pulseless VT or VF	Administer 1 mg every 3–5 min by IV push or IO push. Follow peripheral IV administration with 20-mL saline flush and elevate extremity for 10–20 s.
Norepinephrine—vasopressor given to increase BP	Given for hypotension and shock	Administer 0.1–0.5 mcg/kg/min as IV infusion, preferably through a central line.
Dopamine—vasopressor given to increase BP and contractility	Given for hypotension and shock	Administer 5–10 mcg/kg/min as IV infusion, preferably through a central line.
Atropine—blocks parasympathetic action; increases SA node automaticity and AV conduction	Given to patients with symptomatic bradycardia (i.e., hemodynamically unstable with hypotension)	Administer 0.5-mg IV push; may repeat to dose of 3 mg, follow with saline flush.
Amiodarone—acts on sodium-potassium and calcium channels to prolong action potential and refractory period	Used to treat pulseless VT and VF unresponsive to shock delivery	Administer 300 mg IV; may give second dose of 150 mg in 3–5 min.
Sodium bicarbonate ( $\text{NaHCO}_3$ )—corrects metabolic acidosis	Given to correct metabolic acidosis that is refractory to standard advanced cardiac life support interventions (cardiopulmonary resuscitation, intubation, and respiratory management)	Administer initial dose of 1 mEq/kg IV/IO; then administer dose based on base deficit. Recognize that to prevent development of rebound metabolic alkalosis, complete correction of acidosis is not indicated.
Magnesium sulfate—promotes adequate functioning of cellular sodium-potassium pump	Given to patients with torsade de pointes, a type of VT	May administer 1–2 g diluted in 10 mL $\text{D}_5\text{W}$ over 5–20 min.

AV, atrioventricular; BP, blood pressure;  $\text{D}_5\text{W}$ , dextrose 5% in water; IO, intraosseous; IV, intravenous; SA, sinoatrial; VF, ventricular fibrillation; VT, ventricular tachycardia.

Adapted from American Heart Association. (2019b). Part 7: Adult advanced cardiovascular life support. Resuscitation science: CPR and ECC guidelines. Retrieved on 12/9/2019 at: [www.eccguidelines.heart.org/circulation/cpr-ecc-guidelines/part-7-adult-advanced-cardiovascular-life-support](http://www.eccguidelines.heart.org/circulation/cpr-ecc-guidelines/part-7-adult-advanced-cardiovascular-life-support)



## Follow-Up Monitoring and Care

The care provided to the patient following resuscitation is another determinant of survival (AHA, 2017). A 12-lead ECG is performed to detect any new ST segment elevation or myocardial ischemia (see Chapter 23). Continuous ECG monitoring and frequent blood pressure assessments are essential until hemodynamic stability is established and blood pressure is kept in a range to support adequate perfusion. Factors that precipitated the arrest such as arrhythmias or electrolyte or metabolic imbalances are identified and treated.

Following resuscitation and the return of spontaneous circulation, patients who are comatose may benefit from targeted temperature management (TTM). With TTM, core body temperature is decreased to 32° and 36°C (89.6° to 96.8°F) for at least 24 hours. This induced hypothermia decreases the cerebral metabolic rate and need for oxygen. Similarly, hyperthermic conditions, such as fever, are avoided to reduce oxygen demands (Callaway, Donnino, Fink, et al., 2015).

Advances in cardiac care, such as new techniques for effective resuscitation and postresuscitation hypothermia, have improved outcomes for patients. Research studies demonstrate better neurologic recovery and overall survival for patients when the correct algorithms and team dynamics are used after cardiac arrest; there is hope for even better outcomes in the future.

### CRITICAL THINKING EXERCISES

**1 ipo** After recently being discharged from the hospital with an episode of acute decompensated HFrEF, a 62-year-old man presents to the cardiology clinic where you work. You note that this was the third hospitalization for this patient within the past 6 months. The patient is a widower, lives alone in a two-story home without a bathroom or bedroom on the first floor, and has been receiving disability benefits for several years. Identify how you intend to further assess the patient. What questions will you ask him? Which community-based resources and health care team members could be mobilized to facilitate his transition from the hospital-based setting to the community setting so that he avoids continued rehospitalization?

**2 ebp** You are a nurse educator working in a home health agency. You are tasked with presenting an educational session on HF self-care strategies. Using knowledge of evidence-based practice guidelines, list the most important topics to cover. Consider gender differences, medications, dietary recommendations, and suggestions for exercise.

**3 pq** A 75-year-old woman with an acute MI is admitted to the unit where you work as a staff nurse. You assess the patient and find that she is developing a cough, an increasing respiratory rate (32 breaths/min), and pink-tinged sputum. The patient seems agitated and becomes disoriented; she asks you why she is in the hospital. You call the rapid response team, and the following are prescribed: chest x-ray, arterial blood gases and basic metabolic panel, furosemide 40 mg IV, oxygen per nasal cannula to maintain a saturation greater than 94%. Place your planned interventions in priority order and explain your rationale.

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

- American Association of Heart Failure Nurses (AAHFN), [www.aahfn.org](http://www.aahfn.org)
- American College of Cardiology (ACC), [www.acc.org](http://www.acc.org)
- American Heart Association (AHA), [www.heart.org](http://www.heart.org)
- Heart Failure Society of America (HFS), [www.hfsa.org](http://www.hfsa.org)
- National Heart, Lung, and Blood Institute, [www.nhlbi.nih.gov](http://www.nhlbi.nih.gov)

# **26** Assessment and

## **Management of Patients with Vascular Disorders and Problems of Peripheral Circulation**

### **LEARNING OUTCOMES**

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

- 1.** Identify anatomic and physiologic factors that affect peripheral blood flow and tissue oxygenation.
- 2.** Apply assessment parameters appropriate for determining the status of peripheral circulation.
- 3.** Use the nursing process as a framework for care of the patient with arterial and venous disorders.
- 4.** Compare the pathophysiology, clinical manifestations, management, and prevention of diseases of the arteries.
- 5.** Describe the pathophysiology, clinical manifestations, management, and prevention of venous thromboembolism, venous insufficiency, leg ulcers, and varicose veins.
- 6.** Describe the pathophysiology, clinical manifestations, and management of lymphatic disorders and cellulitis.

## **NURSING CONCEPTS**

Assessment  
Clotting  
Functional Ability  
Perfusion

## **GLOSSARY**

anastomosis: junction of two vessels

aneurysm: a localized sac or dilation of an artery formed at a weak point in the vessel wall

angioplasty: an invasive procedure that uses a balloon-tipped catheter to dilate a stenotic area of a blood vessel

ankle-brachial index (ABI): ratio of the ankle systolic pressure to the brachial systolic pressure; an objective measurement of arterial disease that provides quantification of the degree of stenosis

arteriosclerosis: diffuse process whereby the muscle fibers and the endothelial lining of the walls of small arteries and arterioles thicken

atherectomy: an invasive procedure that uses a cutting device or laser to remove or reduce plaque in an artery

atherosclerosis: inflammatory process involving the accumulation of lipids, calcium, blood components, carbohydrates, and fibrous tissue on the intimal layer of a large- or medium-sized artery

bruit: sound produced by turbulent blood flow through an irregular, tortuous, stenotic, or dilated vessel

cyanosis: a bluish tint of the skin manifested when the amount of oxygenated hemoglobin contained in the blood is reduced

deep vein thrombosis (DVT): a blood clot or thrombus located within a deep vein that causes obstruction or occlusion

dissection: separation of the weakened elastic and fibromuscular elements in the medial layer of an artery

duplex ultrasonography: combines B-mode grayscale imaging of tissue, organs, and blood vessels with capabilities of estimating velocity changes by the use of a pulsed Doppler

embolus: a blood clot, fatty deposit, or air that travels through the blood, lodges in an artery or vein, and blocks flow

endovascular: a type of procedure that uses a puncture or small incision to place catheters inside a blood vessel to repair it or insert a device

intermittent claudication: a muscular, cramplike pain or fatigue in the extremities consistently reproduced with the same degree of exercise or activity and relieved by rest

ischemia: deficient blood supply

pulmonary embolism (PE): a blood clot or thrombus within a pulmonary artery that blocks or obstructs blood flow to the lungs

rest pain: persistent pain in the foot or digits when the patient is resting, indicating a severe degree of arterial insufficiency

rubor: reddish-blue discoloration of the extremities; indicative of severe peripheral arterial damage in vessels that remain dilated and unable to constrict

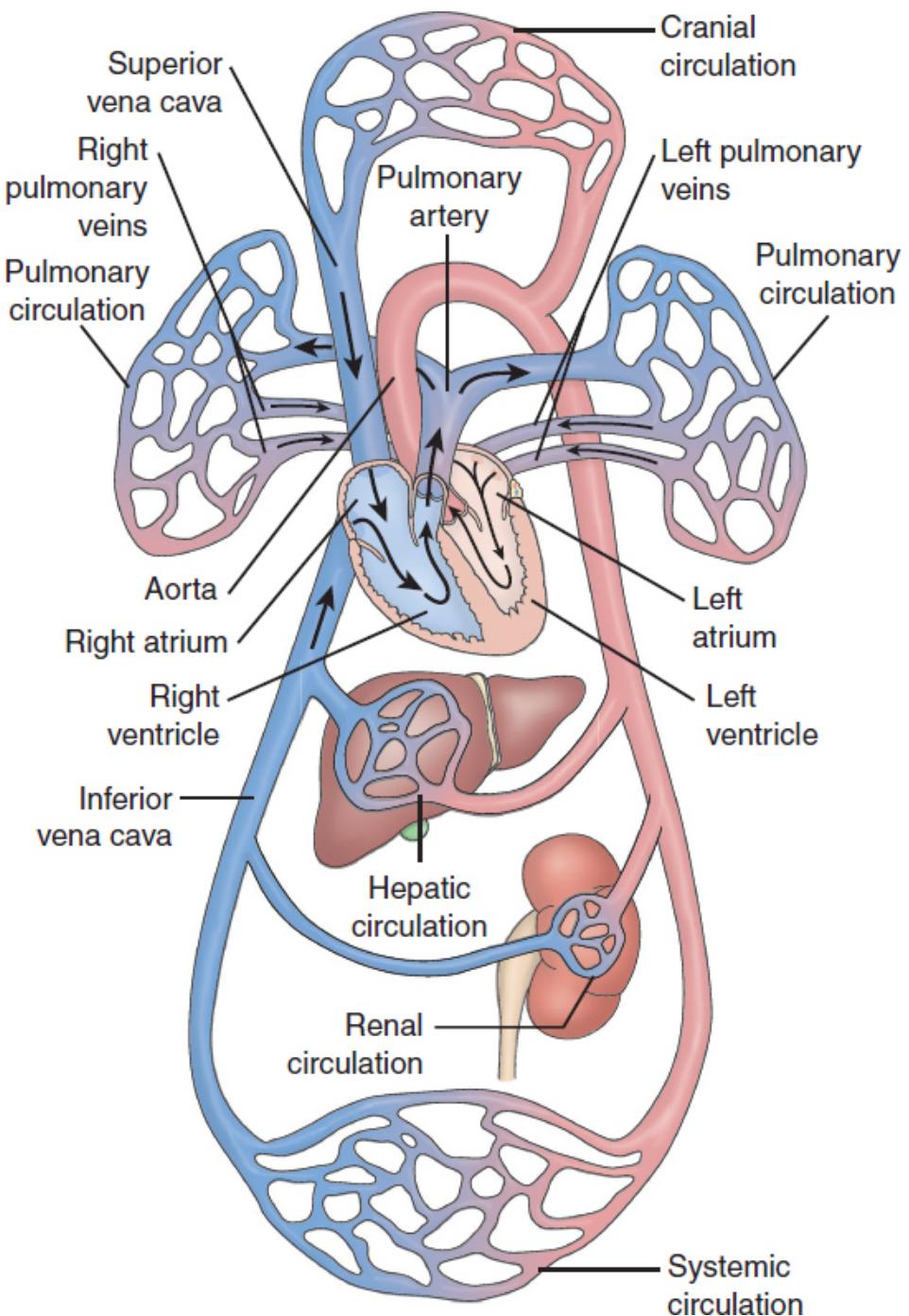
stenosis: narrowing or constriction of a blood vessel  
thromboembolus: a blood clot that may become dislodged from the vessel where it originally formed  
thrombus: a blood clot within an artery or a vein  
venous thromboembolism (VTE): a blood clot that forms in the venous vasculature that may manifest as a DVT or a PE

Conditions of the vascular system include arterial disorders, venous disorders, lymphatic disorders, and cellulitis. These disorders may be seen in patients in both the inpatient and the outpatient setting. Nursing assessment and management depends on an understanding of the vascular system.

## Anatomic and Physiologic Overview

Adequate perfusion ensures oxygenation and nourishment of body tissues, and it depends in part on a properly functioning cardiovascular system. Adequate blood flow depends on the efficiency of the heart as a pump, the patency and responsiveness of the blood vessels, and the adequacy of circulating blood volume. Nervous system activity, blood viscosity, and the metabolic needs of tissues influence the rate and adequacy of blood flow.

The vascular system consists of two interdependent systems. The right side of the heart pumps blood through the lungs to the pulmonary circulation, and the left side of the heart pumps blood to all other body tissues through the systemic circulation. The blood vessels in both systems channel the blood from the heart to the tissues and back to the heart (see [Fig. 26-1](#)). Contraction of the ventricles is the driving force that moves blood through the vascular system.



**Figure 26-1 •** Systemic and pulmonary circulation. Oxygen-rich blood from the pulmonary circulation is pumped from the left heart into the aorta and the systemic arteries to the capillaries, where the exchange of nutrients and waste products takes place. The deoxygenated blood returns to the right heart by way of the systemic veins and is pumped into the pulmonary circulation.

Arteries distribute oxygenated blood from the left side of the heart to the tissues, whereas the veins carry deoxygenated blood from the tissues to the right side of the heart. Capillary vessels located within the tissues connect the arterial and venous systems. These vessels permit the exchange of nutrients and metabolic wastes between the circulatory system and the tissues. Arterioles and venules immediately adjacent to the capillaries, together with the capillaries, make up the microcirculation.

The lymphatic system complements the function of the circulatory system. Lymphatic vessels transport lymph (a fluid similar to plasma) and tissue fluids (containing proteins, cells, and cellular debris) from the interstitial space to systemic veins. Lymphatic fluid empties into the subclavian and internal jugular veins.

## Anatomy of the Vascular System

Arteries, arterioles, capillaries, veins, venules, and lymphatic vessels are the main structures that comprise the vascular system.

### Arteries and Arterioles

Arteries are thick-walled structures that carry blood from the heart to the tissues. The aorta, which has a diameter of approximately 2.5 cm (1 inch) in the average-sized adult, gives rise to numerous branches, which continue to divide into progressively smaller arteries that are 4 mm (0.16 inches) in diameter. The vessels divide further, diminishing in size to approximately 30  $\mu\text{m}$  in diameter. These smallest arteries, called *arterioles*, are generally embedded within the tissues (Norris, 2019).

The walls of the arteries and arterioles are composed of three layers: the intima, an inner endothelial cell layer; the media, a middle layer of smooth muscle and elastic tissue; and the adventitia, an outer layer of connective tissue. The intima, a very thin layer, provides a smooth surface for contact with the flowing blood. The media makes up most of the vessel wall in the aorta and other large arteries of the body. This layer is composed chiefly of elastic and connective tissue fibers that give the vessels considerable strength and allow them to constrict and dilate to accommodate the blood ejected from the heart during each cardiac cycle (stroke volume) and maintain an even, steady flow of blood. The adventitia is a layer of connective tissue that anchors the vessel to its surroundings. There is much less elastic tissue in the smaller arteries and arterioles, and the media in these vessels is composed primarily of smooth muscle (Norris, 2019). Smooth muscle controls the diameter of the vessels by contracting and relaxing. Chemical, hormonal, and neuronal factors influence the activity of smooth muscle. Arterioles offer resistance to blood flow by altering their diameter and are often referred to as resistance vessels. Arterioles regulate the volume and pressure in the arterial system and the rate

of blood flow to the capillaries. There is a large amount of smooth muscle in the media, and the walls of the arteries are relatively thick, accounting for approximately 25% of the total diameter of the artery.

The intima and the inner third of the smooth muscle layer of the media are in such close contact with the blood that the blood vessels receive their nourishment by direct diffusion. The adventitia and the outer media layers require their own blood supply to meet metabolic needs. The *vasa vasorum* is a network of small blood vessels that supplies blood and nutrients to the larger arteries.

## Capillaries

The walls of the capillaries, which lack smooth muscle and adventitia, are composed of a single layer of endothelial cells. This thin-walled structure permits rapid and efficient transport of nutrients to the cells and removal of metabolic wastes. The diameter of capillaries ranges from 5 to 10  $\mu\text{m}$ ; this means that red blood cells must alter their shape to pass through these vessels. Changes in a capillary's diameter are passive and are influenced by contractile changes in the blood vessels that carry blood to and from a capillary. The capillary's diameter also changes in response to chemical stimuli. In some tissues, a cuff of smooth muscle, called the *precapillary sphincter*, is located at the arteriolar end of the capillary and is responsible, along with the arteriole, for controlling capillary blood flow (Norris, 2019).

Some capillary beds, such as those in the fingertips, contain arteriovenous anastomoses, through which blood passes directly from the arterial to the venous system. These vessels are believed to regulate heat exchange between the body and the external environment.

The distribution of capillaries varies with the type of tissue. For example, skeletal tissue, which has high metabolic requirements, has a denser capillary network than cartilage, which has low metabolic needs.

## Veins and Venules

Capillaries join to form larger vessels called *venules*, which join to form veins. The venous system is therefore structurally analogous to the arterial system; venules correspond to arterioles, veins to arteries, and the vena cava to the aorta. Analogous types of vessels in the arterial and venous systems have approximately the same diameters (see Fig. 26-1).

The walls of the veins, in contrast to those of the arteries, are thinner and considerably less muscular. In most veins, the wall makes up only 10% of the diameter, in contrast to 25% in most arteries. In veins, the walls are composed of three layers, like those of arteries; however, in veins, these layers are not as well defined.

The thin, less muscular structure of the vein wall allows these vessels to distend more than arteries. Greater distensibility and compliance permit large volumes of blood to remain in the veins under low pressure. For this reason, veins are referred to as capacitance vessels. Approximately 75% of total blood volume is contained in the veins. The sympathetic nervous system, which innervates the vein musculature, can stimulate venoconstriction (constriction of the veins), thereby reducing venous volume and increasing the volume of blood in the general circulation. Contraction of skeletal muscles in the extremities creates the primary pumping action to facilitate venous blood flow back to the heart (Norris, 2019).

Some veins, unlike arteries, are equipped with valves. In general, veins that transport blood against the force of gravity, as in the lower extremities, have one-way bicuspid valves that prevent blood from retrograde flow as it is propelled toward the heart. Valves are composed of endothelial leaflets, the competency of which depends on the integrity of the vein wall.

### Lymphatic Vessels

The lymphatic vessels are a complex network of thin-walled vessels similar to the capillaries. This network collects lymphatic fluid from tissues and organs, and transports the fluid to the venous circulation. The lymphatic vessels converge into two main structures: the thoracic duct and the right lymphatic duct. These ducts empty into the junction of the subclavian and internal jugular veins. The right lymphatic duct conveys lymph primarily from the right side of the head, neck, thorax, and upper arms. The thoracic duct conveys lymph from the remainder of the body. Peripheral lymphatic vessels join larger lymph vessels and pass through regional lymph nodes before entering the venous circulation. The lymph nodes play an important role in filtering foreign particles.

The lymphatic vessels are permeable to large molecules and provide the only means by which interstitial proteins can return to the venous system. With muscular contraction, lymph vessels become distorted to create spaces between the endothelial cells, allowing protein and particles to enter. Muscular contraction of the lymphatic walls and surrounding tissues aids in propelling the lymph toward the venous drainage points (Norris, 2019).

## Function of the Vascular System

Important functions of the vascular system include supplying the circulatory needs of tissue, maintaining blood flow and blood pressure, and providing capillary filtration and reabsorption, hemodynamic resistance, and other peripheral vascular regulating mechanisms.

### Circulatory Needs of Tissues

The amount of blood flow needed by the body's tissues constantly changes. The percentage of blood flow received by individual organs or tissues is determined by the rate of tissue metabolism, the availability of oxygen, and the function of the tissue. When metabolic requirements increase, blood vessels dilate to increase the flow of oxygen and nutrients to the tissues. When metabolic needs decrease, vessels constrict and blood flow to the tissues decreases. Metabolic demands of tissues increase with physical activity or exercise, local heat application, fever, and infection. Reduced metabolic requirements of tissues accompany rest or decreased physical activity, local cold application, and cooling of the body. If the blood vessels fail to dilate in response to the need for increased blood flow, tissue **ischemia** (deficient blood supply to a body part) results. The mechanism by which blood vessels dilate and constrict to adjust for metabolic changes ensures that normal arterial pressure is maintained (Norris, 2019).

As blood passes through tissue capillaries, oxygen is removed and carbon dioxide is added. The amount of oxygen extracted by each type of tissue differs. For example, the myocardium tends to extract about 50% of the oxygen from arterial blood in one pass through its capillary bed, whereas the kidneys extract only about 7% of the oxygen from the blood that passes through them. The average amount of oxygen removed collectively by all of the body tissues is about 25%. This means that the blood in the vena cava contains about 25% less oxygen than aortic blood. This is known as the systemic arteriovenous oxygen difference (Norris, 2019). This difference becomes greater when less oxygen is delivered to the tissues than they need.

## Blood Flow

Blood flow through the cardiovascular system always proceeds in the same direction: left side of the heart to the aorta, arteries, arterioles, capillaries, venules, veins, vena cava, and right side of the heart. This unidirectional flow is caused by a pressure difference that exists between the arterial and venous systems. Because arterial pressure (approximately 100 mm Hg) is greater than venous pressure (approximately 40 mm Hg) and fluid flows from an area of higher pressure to an area of lower pressure, blood flows from the arterial system to the venous system.

The pressure difference ( $\Delta P$ ) between the two ends of the vessel propels the blood. Impediments to blood flow exert an opposing force, which is known as resistance (R). The rate of blood flow is determined by dividing the pressure difference by the resistance:

$$\text{Flow rate} = \Delta P/R$$

This equation shows that when resistance increases, a greater pressure is required to maintain the same degree of flow (Norris, 2019). In the body, an

increase in pressure is accomplished by an increase in the force of contraction of the heart. If arterial resistance is chronically elevated, the myocardium hypertrophies (enlarges) to sustain a greater contractile force.

In most long, smooth blood vessels, flow is laminar or streamlined, with blood in the center of the vessel moving slightly faster than the blood near the vessel walls. Laminar flow becomes turbulent when the blood flow rate increases, when blood viscosity increases, when the diameter of the vessel becomes greater than normal, or when segments of the vessel are narrowed or constricted (Norris, 2019). Turbulent blood flow creates an abnormal sound, called a **bruit**, which can be heard with a stethoscope.

## Blood Pressure

[Chapter 27](#) provides more information on the physiology and measurement of blood pressure.

## Capillary Filtration and Reabsorption

Fluid exchange across the capillary wall is continuous. This fluid, which has the same composition as plasma without the proteins, forms the interstitial fluid. The equilibrium between hydrostatic and osmotic forces of the blood and interstitium, as well as capillary permeability, determines the amount and direction of fluid movement across the capillary. Hydrostatic force is a driving pressure that is generated by the blood pressure. Osmotic pressure is the pulling force created by plasma proteins. Normally, the hydrostatic pressure at the arterial end of the capillary is relatively high compared with that at the venous end. This high pressure at the arterial end of the capillaries tends to drive fluid out of the capillary and into the tissue space. Osmotic pressure tends to pull fluid back into the capillary from the tissue space, but this osmotic force cannot overcome the high hydrostatic pressure at the arterial end of the capillary. However, at the venous end of the capillary, the osmotic force predominates over the low hydrostatic pressure, and there is a net reabsorption of fluid from the tissue space back into the capillary (Norris, 2019).

Except for a very small amount, fluid that is filtered out at the arterial end of the capillary bed is reabsorbed at the venous end. The excess filtered fluid enters the lymphatic circulation. These processes of filtration, reabsorption, and lymph formation aid in maintaining tissue fluid volume and removing tissue waste and debris. Under normal conditions, capillary permeability remains constant.

Under certain abnormal conditions, the fluid filtered out of the capillaries may greatly exceed the amounts reabsorbed and carried away by the lymphatic vessels. This imbalance can result from damage to capillary walls and subsequent increased permeability, obstruction of lymphatic drainage, elevation of venous pressure, or a decrease in plasma protein osmotic force.

Accumulation of excess interstitial fluid that results from these processes is called *edema*.

### Hemodynamic Resistance

The most important factor that determines resistance in the vascular system is the vessel radius. Small changes in vessel radius lead to large changes in resistance. The predominant sites of change in the caliber or width of blood vessels, and therefore in resistance, are the arterioles and the precapillary sphincter. Peripheral vascular resistance is the opposition to blood flow provided by the blood vessels. This resistance is proportional to the viscosity or thickness of the blood and the length of the vessel and is influenced by the diameter of the vessels. Under normal conditions, blood viscosity and vessel length do not change significantly, and these factors do not usually play an important role in blood flow. However, a large increase in hematocrit may increase blood viscosity and reduce capillary blood flow.

### Peripheral Vascular Regulating Mechanisms

Even at rest, the metabolic needs of body tissues are continuously changing. Therefore, an integrated and coordinated regulatory system is necessary so that blood flow to individual tissues is maintained in proportion to the needs of those tissues. This regulatory mechanism is complex and consists of central nervous system influences, circulating hormones and chemicals, and independent activity of the arterial wall itself.

Sympathetic (adrenergic) nervous system activity, mediated by the hypothalamus, is the most important factor in regulating the caliber and therefore the blood flow of peripheral blood vessels. All vessels are innervated by the sympathetic nervous system except the capillary and precapillary sphincters. Stimulation of the sympathetic nervous system causes vasoconstriction. The neurotransmitter responsible for sympathetic vasoconstriction is norepinephrine (Norris, 2019). Sympathetic activation occurs in response to physiologic and psychological stressors. Diminution of sympathetic activity by medications or sympathectomy results in vasodilation.

Other hormones affect peripheral vascular resistance. Epinephrine, released from the adrenal medulla, acts like norepinephrine in constricting peripheral blood vessels in most tissue beds. However, in low concentrations, epinephrine causes vasodilation in skeletal muscles, the heart, and the brain. Angiotensin I, which is formed from the interaction of renin (synthesized by the kidney) and angiotensinogen, a circulating serum protein, is then converted to angiotensin II by an enzyme secreted by the pulmonary vasculature, called *angiotensin-converting enzyme* (ACE). Angiotensin II is a potent vasoconstrictor, particularly of the arterioles. Although the amount of angiotensin II concentrated in the blood is usually small, its profound vasoconstrictive effects

are important in certain abnormal states, such as heart failure and hypovolemia (Norris, 2019).

Alterations in local blood flow are influenced by various circulating substances that have vasoactive properties. Potent vasodilators include nitric oxide, prostacyclin, histamine, bradykinin, prostaglandin, and certain muscle metabolites. A reduction in available oxygen and nutrients and changes in local pH also affect local blood flow. Proinflammatory cytokines are substances liberated from platelets that aggregate at the site of damaged vessels, causing arteriolar vasoconstriction and continued platelet aggregation at the site of injury (Atherton, Sindone, De Pasquale, et al., 2018).

## Pathophysiology of the Vascular System

Reduced blood flow through peripheral blood vessels characterizes all peripheral vascular diseases. The physiologic effects of altered blood flow depend on the extent to which tissue demands exceed the supply of oxygen and nutrients available. If tissue needs are high, even modestly reduced blood flow may be inadequate to maintain tissue integrity. Tissues become ischemic, malnourished, and ultimately die unless adequate blood flow is restored.

### Pump Failure

Inadequate peripheral blood flow occurs when the heart's pumping action becomes inefficient. Heart failure with reduced left ventricular ejection fraction (HFrEF; also called systolic HF) causes an accumulation of blood in the lungs and a reduction in forward flow or cardiac output, which results in inadequate arterial blood flow to the tissues. Heart failure with preserved left ventricular ejection fraction (HFpEF; also called diastolic HF) causes systemic venous congestion and a reduction in forward flow (Atherton et al., 2018) (see Chapter 25).

### Alterations in Blood and Lymphatic Vessels

Intact, patent, and responsive blood vessels are necessary to deliver adequate amounts of oxygen and nutrients to tissues and to remove metabolic wastes. Arteries can become damaged or obstructed as a result of atherosclerotic plaque, a **thromboembolus** (a blood clot that may become dislodged from the vessel from where it originally formed), chemical or mechanical trauma, infections or inflammatory processes, vasospastic disorders, and congenital malformations. A sudden arterial occlusion causes profound and often irreversible tissue ischemia and tissue death. When arterial occlusions develop gradually, there is less risk of sudden tissue death because collateral circulation may develop, giving that tissue the opportunity to adapt to gradually decreased blood flow.

Venous blood flow can be reduced by a thromboembolus obstructing the vein, by incompetent venous valves, or by a reduction in the effectiveness of the pumping action of surrounding muscles. Decreased venous blood flow results in increased venous pressure, a subsequent increase in capillary hydrostatic pressure, net filtration of fluid out of the capillaries into the interstitial space, and subsequent edema. Edematous tissues cannot receive adequate nutrition from the blood and consequently are more susceptible to breakdown, injury, and infection. Obstruction of lymphatic vessels also results in edema. Lymphatic vessels can become obstructed by a tumor or by damage from mechanical trauma or inflammatory processes.

### Circulatory Insufficiency of the Extremities

Although many types of peripheral vascular diseases exist, most result in ischemia and produce some of the same symptoms: pain, skin changes, diminished pulses, and possible edema. The type and severity of symptoms depend in part on the type, stage, and extent of the disease process and on the speed with which the disorder develops. [Table 26-1](#) highlights the distinguishing features of arterial and venous insufficiency. In this chapter, peripheral vascular disease is categorized as arterial, venous, or lymphatic.



### Gerontologic Considerations

Aging produces changes in the walls of the blood vessels that affect the transport of oxygen and nutrients to the tissues. The intima thickens as a result of cellular proliferation and fibrosis. Elastin fibers of the media become calcified, thin, and fragmented, and collagen accumulates in the intima and the media. These changes cause the vessels to stiffen, which results in increased peripheral resistance, impaired blood flow, and increased left ventricular workload causing hypertrophy, ischemia and HFrEF, and thrombosis along with hemorrhage in the microvessels in the brain and kidney (Atherton et al., 2018).

**TABLE 26-1** Characteristics of Arterial and Venous Insufficiency and Resulting Ulcers

Characteristic	Arterial	Venous
<b>General Characteristics</b>		
Pain	Intermittent claudication to sharp, unrelenting, constant	Aching, throbbing, cramping
Pulses	Diminished or absent	Present, but may be difficult to palpate through edema
Skin characteristics	Dependent rubor—with elevation pallor of foot; dry, shiny skin; cool-to-cold temperature; loss of hair over toes and dorsum of foot; nails thickened and ridged	Pigmentation in gaiter area (area of medial and lateral malleolus), skin thickened and tough, may be reddish blue, frequently with associated dermatitis
<b>Ulcer Characteristics</b>		
Location	Tip of toes, web spaces, heel or other pressure points if patient is immobile	Medial malleolus, lateral malleolus, or anterior tibial area
Pain	Very painful	Minimal pain to very painful
Depth of ulcer	Deep, often involving joint space	Superficial
Shape	Circular	Irregular border
Ulcer base	Pale to black and wet to dry gangrene	Granulation tissue—beefy red to yellow fibrinous in chronic long-term ulcer
Leg edema	Minimal unless extremity kept in dependent position constantly to relieve pain	Moderate to severe

Adapted from Ermer-Selton, J. (2016). Lower extremity assessment. In R. Bryant & D. Nix (Eds.). *Acute and chronic wounds: Current management* (5th ed.). St. Louis, MO: Elsevier.

## Assessment of the Vascular System

The nurse should perform a focused health history and physical assessment to establish a patient's baseline and identify alterations in the vascular system.

### Health History

The nurse obtains an in-depth description from the patient with peripheral vascular disorders of any pain and its precipitating factors. A muscular, cramp-type pain, discomfort, or fatigue in the extremities consistently reproduced with the same degree of activity or exercise and relieved by rest is experienced by patients with peripheral arterial insufficiency. Referred to as **intermittent**

**claudication**, this pain, discomfort, or fatigue is caused by the inability of the arterial system to provide adequate blood flow to the tissues in the face of increased demands for nutrients and oxygen during exercise. As the tissues are forced to complete the energy cycle without adequate nutrients and oxygen, muscle metabolites and lactic acid are produced. Pain is experienced as the metabolites aggravate the nerve endings of the surrounding tissue. Typically, about 50% of the arterial lumen or 75% of the cross-sectional area must be obstructed before intermittent claudication is experienced. When the patient rests and thereby decreases the metabolic needs of the muscles, the pain subsides. The progression of the arterial disease can be monitored by documenting the amount of exercise or the distance the patient can walk before the onset of pain. Distance is measured in blocks, feet, or meters. Persistent pain in the anterior portion of the foot (forefoot) when the patient is resting indicates a severe degree of arterial insufficiency and a critical state of ischemia. Known as **rest pain**, this discomfort is often worse at night and may interfere with sleep. This pain frequently requires that the extremity be lowered to a dependent position to improve perfusion to the distal tissues.

The site of arterial disease can be deduced from the location of claudication, because pain occurs in muscle groups distal to the diseased vessel. Calf pain may accompany reduced blood flow through the superficial femoral or popliteal artery, whereas pain in the hip or buttock may result from reduced blood flow in the abdominal aorta or the common iliac or hypogastric (also known as internal iliac) arteries.

## Physical Assessment

A thorough assessment of the patient's skin color and temperature and the character or quality of the peripheral pulses is important in the diagnosis of arterial disorders.

### Inspection of the Skin

Adequate blood flow warms the extremities and gives individuals with lighter skin tones a rosy coloring. Inadequate blood flow results in cool and pale extremities. In people with pigmented skin, the color changes are often more difficult to discriminate because of the darker skin tone. Further reduction of blood flow to these tissues, which occurs when the extremity is elevated, for example, results in pallor (a whiter or more blanched appearance). **Rubor**, a reddish-blue discoloration of the extremities, may be observed within 20 seconds to 2 minutes after the extremity is placed in the dependent position. Rubor suggests severe peripheral arterial damage in which vessels that cannot constrict remain dilated. Even with rubor, the extremity begins to turn pale with elevation. **Cyanosis**, a bluish tint of the skin, is manifested when the amount of oxygenated hemoglobin contained in the blood is reduced.

Additional changes resulting from a chronically reduced nutrient supply include loss of hair, brittle nails, dry or scaling skin, atrophy, and ulcerations. Edema may be apparent bilaterally or unilaterally and is related to the affected extremity's chronically dependent position because of rest pain. Gangrenous changes appear after prolonged, severe ischemia and represent tissue necrosis.

### Palpation of Pulses

Determining the presence or absence, as well as the quality, of peripheral pulses is important in assessing the status of peripheral arterial circulation (see Fig. 26-2). Pulse assessment in an edematous extremity should be undertaken with caution. Palpation of pulses is subjective, and the nurse may mistake their own pulse for that of the patient. To prevent this, the nurse should use light touch and use more than just the index finger for palpation, because this finger has the strongest arterial pulsation of all the fingers. The thumb should not be used for the same reason. Absence of a pulse may indicate that the site of **stenosis** (narrowing or constriction) or occlusion is proximal to that level. Occlusive arterial disease impairs blood flow and can reduce or obliterate palpable pulsations in the extremities. Pulses should be palpated bilaterally and simultaneously, comparing both sides for symmetry in rate, rhythm, and quality.

### Diagnostic Evaluation

The nurse should educate the patient on the purpose of the diagnostic studies, what to expect, and any possible side effects related to these examinations. Trends in results are noted because they provide information about disease progression as well as the patient's response to therapy. Various tests may be performed to identify and diagnose abnormalities that can affect the vascular structures (arteries, veins, and lymphatics).

### Doppler Ultrasound Flow Studies

When pulses cannot be reliably palpated, a handheld continuous wave (CW) Doppler ultrasound device may be used to detect the blood flow. This handheld device emits a continuous signal through the patient's tissues. The signals are reflected by the moving blood cells and received by the device. The filtered-output Doppler signal is then transmitted to a loudspeaker or headphones, where it can be heard for interpretation as arterial or venous signals (Fischbach & Fischbach, 2018). The depth at which blood flow can be detected by Doppler is determined by the frequency (in megahertz [MHz]) it generates. The lower the frequency, the deeper the tissue penetration; a 5- to 10-MHz probe may be used to evaluate the peripheral arteries.

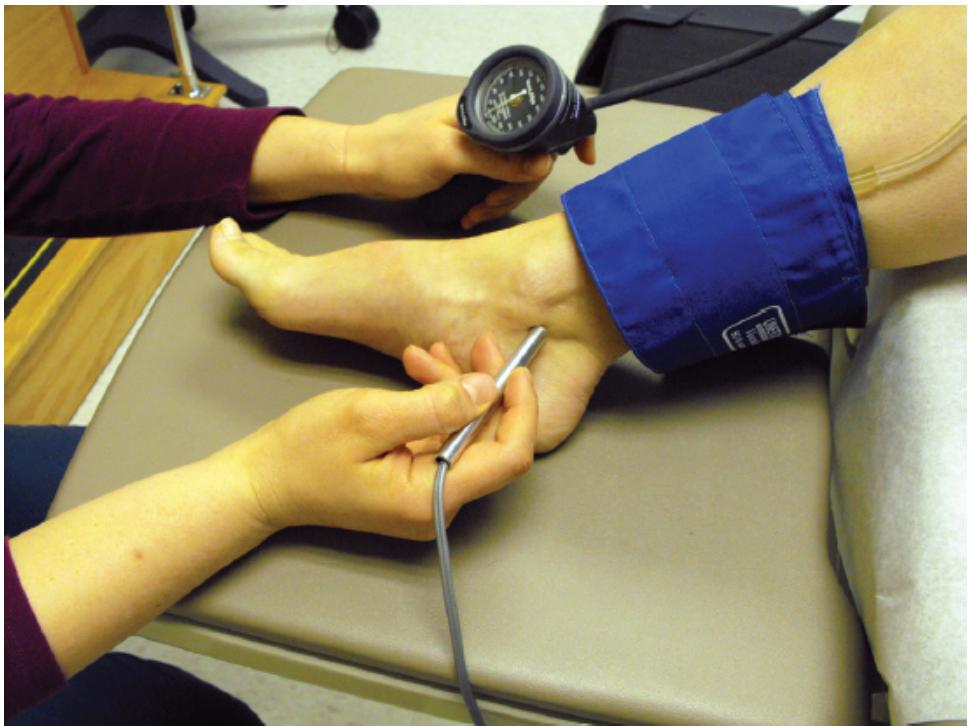


**Figure 26-2 • Assessing peripheral pulses.** **A.** Popliteal pulse. **B.** Dorsalis pedis pulse. **C.** Posterior tibial pulse. Reprinted with permission from Weber, J. R., & Kelley, J. H. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

To evaluate the lower extremities, the patient is placed in the supine position with the head of the bed elevated 20 to 30 degrees; the legs are externally rotated, if possible, to permit adequate access to the medial malleolus. Acoustic water soluble gel is applied to the patient's skin to permit uniform transmission of the ultrasound wave. The tip of the Doppler transducer is positioned at a 45- to 60-degree angle over the expected location of the artery and angled slowly to identify arterial blood flow. Excessive pressure is avoided because severely diseased arteries can collapse with even minimal pressure.

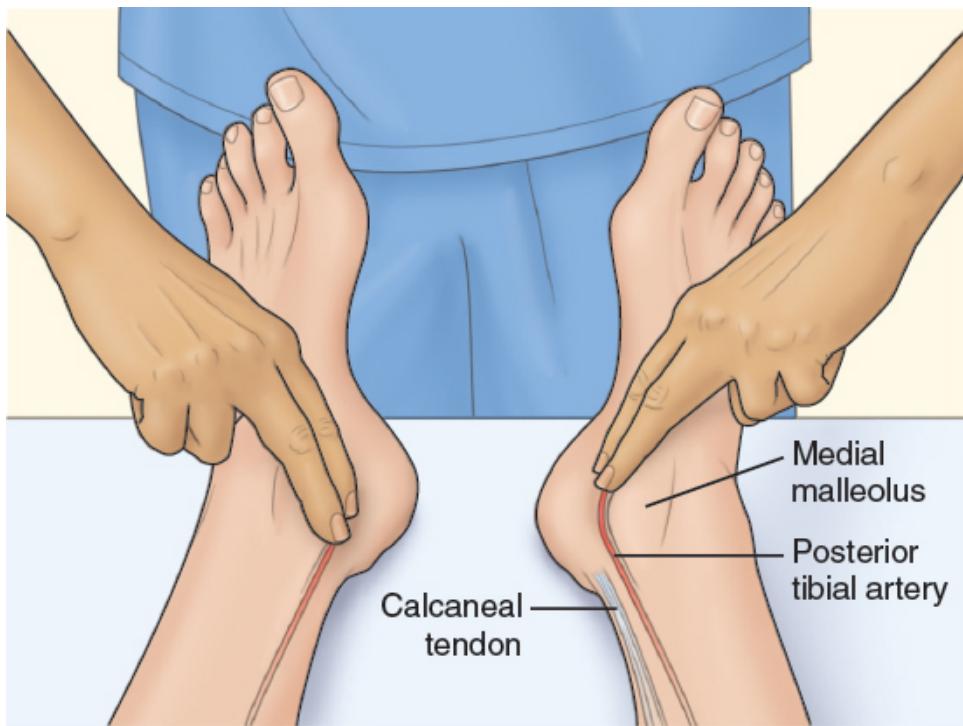
The transducer can detect blood flow in advanced arterial disease states, especially if collateral circulation has developed; thus, identifying a signal indicates only the presence of blood flow. The patient's provider must be notified of the absence of a signal if one had been detected previously.

CW Doppler is more useful as a clinical tool when combined with ankle blood pressures, which are used to determine the **ankle-brachial index (ABI)** (see Fig. 26-3). The ABI is the ratio of the systolic blood pressure in the ankle to the systolic blood pressure in the arm (Zierler & Dawson, 2016). It is an objective indicator of arterial disease that allows the examiner to quantify the degree of stenosis. With increasing degrees of arterial narrowing, there is a progressive decrease in systolic pressure distal to the involved sites.



**Figure 26-3 •** Continuous wave Doppler ultrasound detects blood flow in peripheral vessels. Combined with computation of ankle or arm pressures, this diagnostic technique helps health care providers characterize the nature of peripheral vascular disease. Photograph courtesy of Kim Cantwell-Gab, MN, ACNP, ANP.

The first step in determining the ABI is to have the patient rest in a supine position (not seated) for approximately 5 minutes. An appropriate-sized blood pressure cuff (typically, a 10-cm cuff for an average-sized adult) is applied to the patient's ankle above the malleolus. After identifying an arterial signal of the posterior tibial (see Fig. 26-4) and dorsalis pedis arteries, the systolic pressures, are obtained in both ankles, while listening to the Doppler signal of each artery. Diastolic pressures in the ankles cannot be measured with Doppler. If pressure in these arteries cannot be obtained, pressure can be measured in the peroneal artery, which can also be assessed at the ankle.



**Figure 26-4 •** Palpation of posterior tibial artery.

Doppler ultrasonography is used to measure brachial pressures in both arms. Both arms are evaluated because the patient may have an asymptomatic stenosis in the subclavian artery, causing brachial pressure on the affected side to be 15 to 20 mm Hg or more lower than systemic pressure. The abnormally low pressure should not be used for assessment.

To calculate the ABI, the highest systolic pressure for each ankle is divided by the higher of the two brachial systolic pressures (see [Chart 26-1](#)). The ABI can be computed for a patient with the following systolic pressures:

*Right brachial:* 160 mm Hg

*Left brachial:* 120 mm Hg

*Right posterior tibial:* 80 mm Hg

*Right dorsalis pedis:* 60 mm Hg

*Left posterior tibial:* 100 mm Hg

*Left dorsalis pedis:* 120 mm Hg

The highest systolic pressure for each ankle (80 mm Hg for the right, 120 mm Hg for the left) would be divided by the highest brachial pressure (160 mm Hg):

*Right:*  $80/160 \text{ mm Hg} = 0.50 \text{ ABI}$

*Left:*  $120/160 \text{ mm Hg} = 0.75 \text{ ABI}$

In general, systolic pressure in the ankle of a healthy person is the same or slightly higher than the brachial systolic pressure, resulting in an ABI of about 1.0 (no arterial insufficiency) (see [Chart 26-2](#) for ABI pressure ranges).

## Nursing Implications

Nurses should perform a baseline ABI on any patient with decreased pulses or any patient 65 years or older, especially patients with a history of diabetes or nicotine use (Gerhard-Herman, Gornik, Barrett, et al., 2016). Patients who undergo an arterial intervention or surgery should have ABIs performed per their institution's protocols. In addition, if there is a change in the clinical status of a patient, such as a sudden cold or painful limb, an ABI should be performed.

Prior to the procedure, nurses should educate patients about the indications for ABI and what to expect. Patients should be instructed to avoid use of nicotine products or drinking caffeinated beverages for at least 2 hours prior to testing (if it is done on a nonurgent basis). There may be some discomfort involved when the cuffs are inflated.

### Chart 26-1

## Avoiding Common Errors in Obtaining the Ankle-Brachial Index

Take the following precautions to ensure an accurate ankle-brachial index (ABI) calculation:

- *Use correctly sized blood pressure (BP) cuffs.* To obtain accurate BP measurements, use a cuff with a bladder width at least 40% and length at least 80% of the limb circumference.
- *On the nursing plan of care, document the cuff sizes used* (e.g., “12-cm adult cuff used for brachial pressures; 10-cm pediatric cuff used for ankle pressures”). This minimizes the risk of shift-to-shift discrepancies in ABIs.
- *Use sufficient cuff inflation.* To ensure complete closure of the artery and the most accurate measurements, inflate cuff 20 to 30 mm Hg beyond the point at which the last arterial signal is detected.
- *Do not deflate cuff too rapidly.* Try to maintain a deflation rate of 2 to 4 mm Hg/s for patients without arrhythmias and 2 mm Hg/s or slower for patients with arrhythmias. Deflating the cuff more rapidly may miss the patient’s highest pressure and result in recording an erroneous (low) BP measurement.
- *Suspect medial calcific sclerosis any time an ABI is 1.20 or greater or ankle pressure is more than 250 mm Hg.* Medial calcific sclerosis is associated with diabetes, chronic kidney disease, and hyperparathyroidism. It produces falsely elevated ankle pressures by hardening the media of the arteries, making the vessels noncompressible.
- *Be suspicious of arterial pressures recorded at less than 40 mm Hg.* This may mean the venous signal has been mistaken for the arterial signal. If the arterial pressure, which is normally 120 mm Hg, is measured at less than 40 mm Hg, ask a colleague to double-check the readings before recording this as an arterial pressure.

## Chart 26-2

### **Range of Ankle-Brachial Index (ABI) Pressure and Ischemic Manifestations**

ABI >1.40 is abnormal; indicates noncompressible arteries; requires further testing with a toe-brachial index (TBI)

ABI of 1.00 to 1.40 is normal

ABI of 0.91 to 0.99 is borderline

ABI of  $\leq 0.90$  is abnormal

ABI of 0.50 to 0.90 (i.e., moderate to mild insufficiency) is usually found in patients with claudication

ABI of  $<0.50$  is found in patients with ischemic rest pain

ABI of  $\leq 0.40$  is found in patients with severe ischemia or tissue loss

Adapted from Gerhard-Herman, M., Gornik, H., Barrett, C., et al. (2016).

AHA/ACC Guideline on the management of patients with lower extremity peripheral arterial disease: Executive summary. *Circulation*, 134(24), 1–208; Zierler, R. E., & Dawson, D. L. (Eds.). (2016). *Strandness's duplex scanning in vascular disorders* (5th ed.). Philadelphia, PA: Wolters Kluwer.

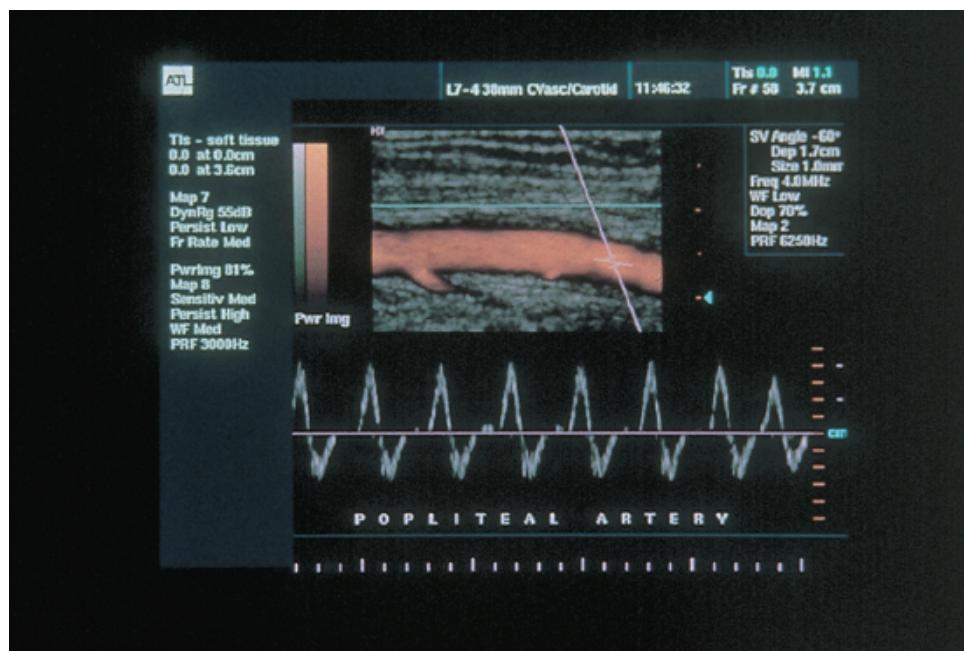
## **Exercise Testing**

Exercise testing is used to determine how long a patient can walk and to measure the ankle systolic blood pressure in response to walking. The patient's brachial systolic blood pressure is obtained on each arm prior to treadmill walking. There are several exercise testing protocols; however, in most instances the patient walks on a treadmill at 1.5 mph with a 12% incline for a maximum of 5 minutes or walks with a gradual rise in speed and incline to the point of claudication. The exercise test can be modified to walking a set distance in a hallway. Most patients can complete the test unless they have significant arterial insufficiency, severe cardiac, pulmonary, or orthopedic problems, or a physical disability. A normal response is little or no drop in ankle systolic pressure after exercise. However, in a patient with true vascular claudication, the ankle pressure drops. Combining this hemodynamic information with the walking time helps to determine whether intervention is necessary. The nurse should reassure the patient that the treadmill test will not require running; rather, the test may require walking on a slight incline. In some instances cycling may be used to evaluate the patient's ability to walk.

## **Duplex Ultrasonography**

**Duplex ultrasonography** involves B-mode grayscale imaging of the tissue, organs, and blood vessels (arterial and venous) and permits estimation of velocity changes by use of a pulsed Doppler (see Fig. 26-5). Color flow techniques, which can identify vessels, may be used to shorten the examination

time. Duplex ultrasound may be used to determine the level and extent of venous disease as well as chronicity of the disease. Using B mode and Doppler, it is possible to image and assess blood flow, evaluate flow of the distal vessels, locate the disease (stenosis versus occlusion), and determine anatomic morphology and the hemodynamic significance of plaque causing stenosis. Duplex ultrasound findings help in planning treatment and monitoring its outcomes. The test is noninvasive and usually requires no patient preparation. Patients who undergo abdominal vascular duplex ultrasound, however, should be advised to not eat or drink (i.e., NPO status) for at least 6 hours prior to the examination to decrease production of bowel gas that can interfere with the examination. The equipment is portable, making it useful anywhere for initial diagnosis, screening, or follow-up evaluations.



**Figure 26-5 •** Color flow duplex image of popliteal artery with normal triphasic Doppler flow.

## Computed Tomography Scanning

Computed tomography (CT) scanning provides cross-sectional images of soft tissue and visualizes the area of volume changes to an extremity and the compartment where changes take place. CT scans of the abdomen are useful in assessing characteristics and monitoring changes within the aorta, such as an increasing aortic diameter indicating aneurysmal formation. CT of a lymphedematous arm or leg, for example, demonstrates a characteristic honeycomb pattern in the subcutaneous tissue. In multidetector-computed tomography (MDCT), a spiral CT scanner and rapid intravenous (IV) infusion

of contrast agent are used to image very thin sections of the target area, and the results are configured in three dimensions so that the image can be rotated and viewed from multiple angles. The scanner's head moves circumferentially around the patient as the patient passes through the scanner, creating a series of overlapping images that are connected to one another in a continuous spiral. Scan times are short. However, the patient is exposed to x-rays, and a contrast agent is injected to visualize the blood vessels. The high volume of contrast agent injected into a peripheral vein may contraindicate the use of MDCT in children and patients with significantly impaired renal function (Gerhard-Herman et al., 2016).

### Nursing Implications

Patients with impaired renal function scheduled for MDCT may require preprocedural treatment to prevent contrast-induced nephropathy. This may include oral or IV hydration 6 to 12 hours preprocedure or administration of sodium bicarbonate, which alkalinizes urine and protects against free radical damage. Studies do not support the use of oral and IV *N*-acetylcysteine for protection against contrast-induced nephropathy (Fähling, Seeliger, Patzak, et al., 2017). The nurse should encourage fluids and monitor the patient's urinary output post procedure, which should be at least 0.5 mL/kg/h. Contrast-induced acute kidney injury may occur within 48 to 96 hours post procedure; therefore, the nurse should follow up with the patient's primary provider if this occurs (see [Chapter 48](#) for discussion of acute kidney injury). Patients who have known iodine or shellfish allergies may need premedication with steroids and histamine blockers.

### Angiography

An arteriogram produced by angiography may be used to confirm the diagnosis of occlusive arterial disease when surgery or other interventions are considered. It involves injecting a radiopaque contrast agent directly into the arterial system to visualize the vessels. The location of a vascular obstruction or an **aneurysm** (abnormal dilation of a blood vessel) and the collateral circulation can be demonstrated. Typically, the patient experiences a temporary sensation of warmth as the contrast agent is injected, and local irritation may occur at the injection site. Infrequently, a patient may have an immediate or delayed allergic reaction to the iodine contained in the contrast agent. Manifestations include dyspnea, nausea and vomiting, sweating, tachycardia, and numbness of the extremities. Any reaction must be reported to the interventionist at once; treatment may include the administration of epinephrine, antihistamines, or corticosteroids. Additional procedural risks include vessel injury, acute arterial occlusion, bleeding, or contrast nephropathy.

## Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) is performed with a standard magnetic resonance imaging (MRI) scanner and special software programmed to isolate the blood vessels. The resulting images can be rotated and viewed from multiple angles (Gerhard-Herman et al., 2016).

### Nursing Implications

MRA is contraindicated in patients with metal implants. Prior to the MRA, the nurse should assess for the presence of any incompatible devices, such as aneurysm clips, old tattoos, which may contain trace elements (newer materials used in tattoos such as nitinol and titanium are MRI compatible), some medication patches, or a cardiac implantable electronic device. Patients with any type of cardiac implantable electronic device need to be screened to determine if they can safely undergo MRI (Indik, Gimbel, Abe, et al., 2017).

The nurse should educate the patient regarding what to expect during and after the procedure. The patient should be prepared to lie on a cold, hard table that slides into an enclosed small tube. The nurse should inform the patient that they will hear noises, including periodic banging and popping sounds. Patients with claustrophobia may be prescribed a sedative prior to the procedure. Patients should be instructed to close their eyes before entering the tube, and to keep them closed, as this may decrease claustrophobic symptoms. Patients should be reassured that they will be provided with a panic button to press if they feel a need to stop the procedure. MRA procedures require the use of IV contrast dye; therefore, nursing implications following MRA are the same as those for MDCT (discussed in CT section).

## Contrast Phlebography (Venography)

Also known as venography, contrast phlebography involves injecting a radiopaque contrast agent into the venous system. If a **thrombus** (a blood clot within an artery or vein) exists, the x-ray image reveals an unfilled segment of vein in an otherwise completely filled vein. Injection of the contrast agent may cause brief but painful inflammation of the vein. This test is rarely performed, as duplex ultrasonography is considered the standard for diagnosing lower extremity venous thrombosis (Zierler & Dawson, 2016). The nurse should instruct the patient that they will receive contrast dye through a peripheral vein and will be monitored for 2 hours post venogram for access site oozing or hematoma. The guidelines for nursing care following venogram are the same as those for MDCT (see earlier discussion).

## Lymphoscintigraphy

Lymphoscintigraphy involves injection of a radioactively labeled colloid subcutaneously in the second interdigital space. The extremity is then exercised to facilitate the uptake of the colloid by the lymphatic system, and serial images are obtained at preset intervals.

### Nursing Implications

The nurse should educate the patient about what to expect. For instance, the blue dye typically used for this procedure may stain the injection site. If the patient has a lymphatic leak, as can occur with groin incisions, there may be blue drainage from the incision until the dye clears from the system, which may take several days.

## ARTERIAL DISORDERS

Arterial disorders cause ischemia and tissue necrosis. These disorders may occur because of chronic progressive pathologic changes to the arterial vasculature (e.g., atherosclerotic changes) or an acute loss of blood flow to the tissues (e.g., aneurysm rupture).

### Arteriosclerosis and Atherosclerosis

**Arteriosclerosis** (hardening of the arteries) is the most common disease of the arteries. It is a diffuse process whereby the muscle fibers and the endothelial lining of the walls of small arteries and arterioles become thickened. **Atherosclerosis** involves a different process, affecting the intima of large and medium-sized arteries. These changes consist of the accumulation of lipids, calcium, blood components, carbohydrates, and fibrous tissue on the intimal layer of the artery. These accumulations are referred to as atheromas or plaques.

Although the pathologic processes of arteriosclerosis and atherosclerosis differ, rarely does one occur without the other, and the terms are often used interchangeably. Atherosclerosis is a generalized disease of the arteries, and when it is present in the extremities, it is usually present elsewhere in the body.

### Pathophysiology

The most common direct results of atherosclerosis in arteries include stenosis (narrowing) of the lumen, obstruction by thrombosis, aneurysm, ulceration, and rupture. Its indirect results are malnutrition and the subsequent fibrosis of the organs that the sclerotic arteries supply with blood. All actively functioning tissue cells require an abundant supply of nutrients and oxygen and are sensitive to any reduction in the supply of these nutrients. If such reductions are severe and permanent, the cells undergo ischemic necrosis (death of cells

due to deficient blood flow) and are replaced by fibrous tissues, which require much less blood flow.

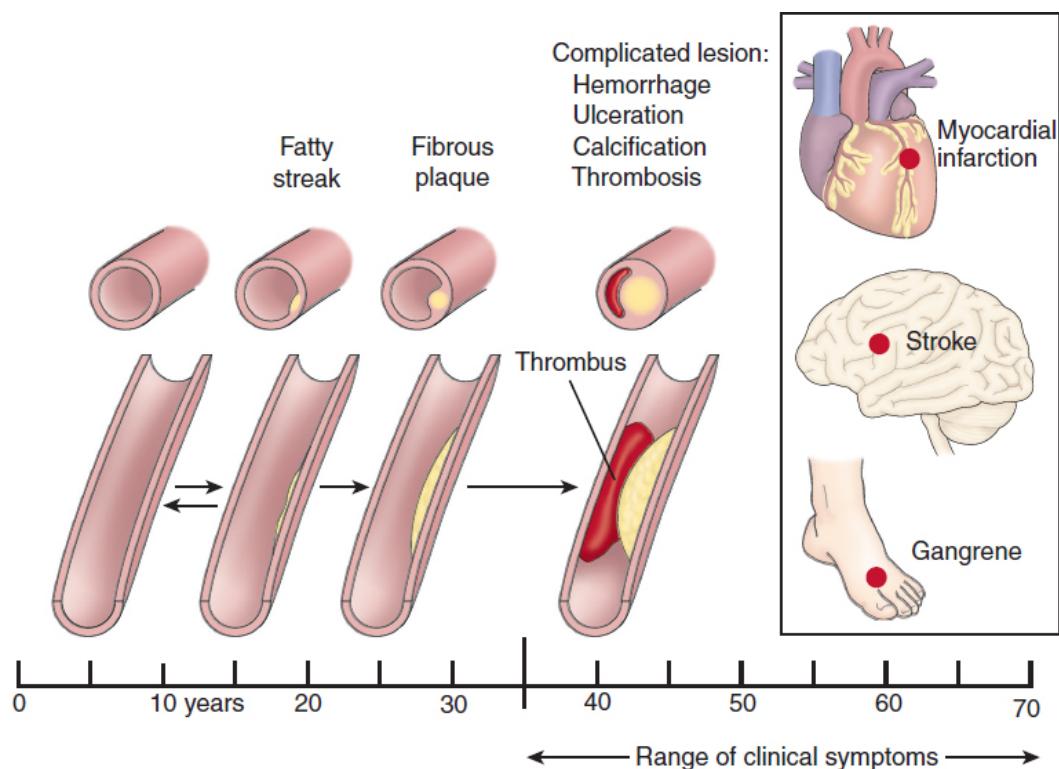
Atherosclerosis can develop in any part of the vascular system, but certain sites are more vulnerable, such as regions where arteries bifurcate or branch into smaller vessels, with males having more below-the-knee pathology than females (Jelani, Petrov, Martinez, et al., 2018). In the proximal lower extremity, these include the distal abdominal aorta, the common iliac arteries, the orifice of the superficial femoral and profunda femoris arteries, and the superficial femoral artery in the adductor canal, which is particularly narrow. Distal to the knee, atherosclerosis can occur anywhere along the course of the artery.

Although many theories exist about the development of atherosclerosis, no single theory explains the pathogenesis completely; however, tenets of several theories are incorporated into the reaction-to-injury theory. According to this theory, vascular endothelial cell injury results from prolonged hemodynamic forces, such as shearing stresses and turbulent flow, irradiation, chemical exposure, or chronic hyperlipidemia. Injury to the endothelium increases the aggregation of platelets and monocytes at the site of the injury. Smooth muscle cells migrate and proliferate, allowing a matrix of collagen and elastic fibers to form (Norris, 2019).

Atherosclerotic lesions are of two types: fatty streaks and fibrous plaque.

- Fatty streaks are yellow and smooth, protrude slightly into the lumen of the artery, and are composed of lipids and elongated smooth muscle cells. These lesions have been found in the arteries of people of all ages, including infants. It is not clear whether fatty streaks predispose a person to the formation of fibrous plaques or whether they are reversible. They do not usually cause clinical symptoms.
- Fibrous plaques are composed of smooth muscle cells, collagen fibers, plasma components, and lipids. They are white to white-yellow and protrude to various degrees into the arterial lumen, sometimes completely obstructing it. These plaques are found predominantly in the abdominal aorta and the coronary, popliteal, and internal carotid arteries, and they are believed to be progressive lesions (see [Fig. 26-6](#)).

Gradual narrowing of the arterial lumen stimulates the development of collateral circulation (see [Fig. 26-7](#)). Collateral circulation arises from preexisting vessels that enlarge to reroute blood flow around a hemodynamically significant stenosis or occlusion. Collateral flow allows continued perfusion to the tissues, but it is often inadequate to meet increased metabolic demands, and ischemia results.



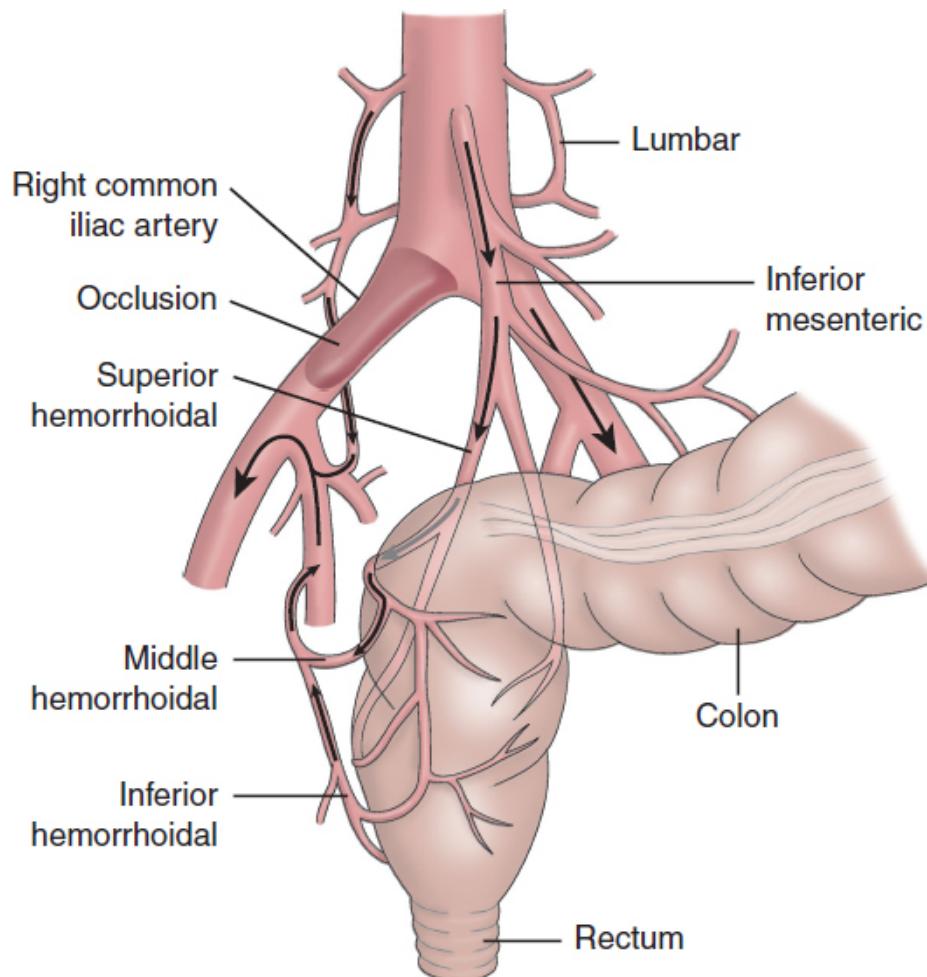
**Figure 26-6 •** Schematic concept of the progression of atherosclerosis. Fatty streaks are an early manifestation of atherosclerosis. Fatty streaks either regress or progress to fibrous plaques and eventually to atheroma. Atheromatous plaque may be complicated by hemorrhage, ulceration, calcification, or thrombosis leading to myocardial infarction, stroke, claudication, rest pain, or gangrene.

## Risk Factors

Many risk factors are associated with atherosclerosis (see [Chart 26-3](#)). Although it is not entirely clear whether modification of these risk factors prevents the development of cardiovascular disease, evidence indicates that it may slow the process.

The use of nicotine products may be one of the most important risk factors in the development of atherosclerotic lesions. Nicotine in tobacco decreases blood flow to the extremities and increases heart rate and blood pressure by stimulating the sympathetic nervous system, causing vasoconstriction (Quintella Farah, Silva Rigoni, de Almeida Correia, et al., 2019). It also increases the risk of clot formation by increasing the aggregation of platelets. Carbon monoxide, a toxin produced by burning tobacco, combines more readily with hemoglobin than oxygen, depriving the tissues of oxygen. There is evidence that smoking decreases high-density lipoprotein (HDL; good cholesterol) levels and alters the ratios between HDL and low-density

lipoprotein (LDL; bad cholesterol), HDL and triglycerides, and HDL and total cholesterol levels (see [Chapter 23](#) for discussion of HDL and LDL and their association with atherosclerosis). The amount of tobacco used—inhaled in traditional or e-cigarette form, or chewed—is directly related to the extent of the disease, and cessation of any type of nicotine product use reduces the risk (Jelani et al., 2018).



**Figure 26-7 •** Development of channels for collateral blood flow in response to occlusion of the right common iliac artery and the terminal aortic bifurcation.

**Chart 26-3**  **RISK FACTORS**

## Atherosclerosis and Peripheral Artery Disease

### Modifiable Risk Factors

- Nicotine use (i.e., tobacco product such as cigarettes, e-cigarettes, or chewing tobacco)
- Diabetes (speeds the atherosclerotic process by thickening the basement membranes of both large and small vessels)
- Hypertension
- Hyperlipidemia
- Diet (contributing to hyperlipidemia)
- Stress
- Sedentary lifestyle
- Elevated C-reactive protein
- Hyperhomocysteinemia

### Nonmodifiable Risk Factors

- Increasing age
- Familial predisposition/genetics

Adapted from Sidawy, A. N., & Perler, B. A. (2019). *Rutherford's vascular surgery and endovascular therapy* (9th ed.). Philadelphia, PA: Elsevier.

Diabetes increases the overall risk of peripheral artery disease (PAD) two- to fourfold, with amputation rates 5 to 10 times higher than in patients without diabetes. Patients with diabetes show an earlier onset and more rapid progression of PAD than patients without diabetes; furthermore, they also exhibit a different anatomic distribution of pathology, with a greater severity of disease in the profunda femoris and in all segments below the knee (Gerhard-Herman et al., 2016). How diabetes affects the onset and progression of atherosclerosis is multifactorial and includes incitation of inflammatory processes, derangement of various cell types within vessel walls, promotion of coagulation, and inhibition of fibrinolysis (Hazarika & Annex, 2017). Many other factors, such as obesity, stress, and lack of exercise, have been identified as contributing to the disease process.

C-reactive protein (CRP) is a sensitive marker of cardiovascular inflammation, both systemically and locally. Slight increases in serum CRP levels are associated with an increased risk of damage in the vasculature, especially if these increases are accompanied by other risk factors, including increasing age, hypertension, hypercholesterolemia, obesity, elevated blood glucose levels, use of nicotine products, or a positive family history of cardiovascular disease (Hazarika & Annex, 2017).

In some studies, hyperhomocysteinemia has been positively correlated with the risk of peripheral, cerebrovascular, and coronary artery disease as well as

venous thromboembolism (VTE). Homocysteine is a protein that promotes coagulation by increasing factor V and factor XI activity while depressing protein C activation and increasing the binding of lipoprotein(a) in fibrin. These processes increase thrombin formation and the propensity for thrombosis. Folic acid and vitamin B<sub>12</sub> have been reported to reduce serum homocysteine levels but there are no data demonstrating this therapy reduces adverse cardiovascular events. Thus, the use of B complex vitamins to reduce cardiovascular disease in patients with PAD is not recommended (Gerhard-Herman et al., 2016).

## Prevention

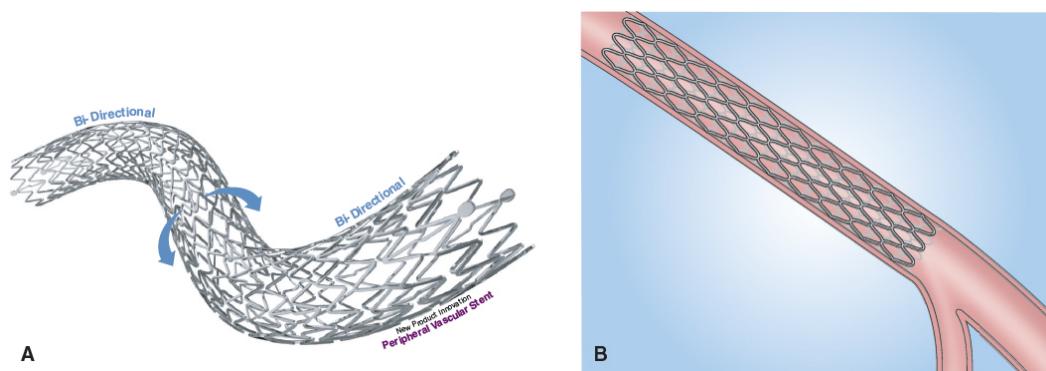
Intermittent claudication is a symptom of generalized atherosclerosis and may be a marker of atherosclerosis in other arterial territories, such as the coronary and carotid arteries. The suspicion that a high-fat diet contributes to atherosclerosis means that it is reasonable to measure serum cholesterol and to begin disease prevention efforts that include diet modification. The American Heart Association recommends reducing the amount of fat ingested, substituting unsaturated fats for saturated fats, and decreasing cholesterol intake to reduce the risk of cardiovascular disease.

Certain medications that supplement dietary modification and exercise are used to reduce blood lipid levels. Current evidence-based guidelines established by the American College of Cardiology and the American Heart Association (ACC/AHA) recommend 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins) as first-line therapy in patients with PAD for secondary prevention and cardiovascular risk reduction (Conte, Bradbury, Kohl, et al., 2019). These statins may include medications such as atorvastatin, lovastatin, pitavastatin, pravastatin, simvastatin, fluvastatin, and rosuvastatin. Several other classes of medications used to reduce lipid levels include bile acid sequestrants (cholestyramine, colestevam, colestipol), nicotinic acid (niacin), fibric acid inhibitors (gemfibrozil, fenofibrate), and cholesterol absorption inhibitors (ezetimibe). Patients receiving long-term therapy with these medications require close monitoring.

Hypertension, which may accelerate the rate at which atherosclerotic lesions form in high-pressure vessels, can lead to a stroke, ischemic renal disease, severe PAD, or coronary artery disease. Hypertension is a major risk factor for the development of PAD and may be a more significant risk factor for women than men based on research findings from the classic Framingham Heart Study and a similar study performed in Europe (Jelani et al., 2018). The majority of patients with hypertension require more than two antihypertensive agents to reach target blood pressure, and at least one third require more than three antihypertensive agents to achieve effective blood pressure control

(Sidawy & Perler, 2019). See [Chapter 27](#) for further discussion of hypertension.

Although no single risk factor has been identified as the primary contributor to the development of atherosclerotic cardiovascular disease, it is clear that the greater the number of risk factors, the greater the risk of atherosclerosis. Elimination of all controllable risk factors, particularly nicotine use, is strongly recommended.



**Figure 26-8 • A.** Flexible stent. Used with permission from QualiMed Innovative Medizinprodukte GmbH, a Q3 Medical Company. **B.** Representation of a common iliac artery with a wall stent.

## Clinical Manifestations

The clinical signs and symptoms resulting from atherosclerosis depend on the organ or tissue affected. Coronary atherosclerosis (heart disease), angina, and acute myocardial infarction are discussed in [Chapter 23](#). Cerebrovascular diseases, including transient ischemic attacks and stroke, are discussed in [Chapter 62](#). Atherosclerosis of the aorta, including aneurysm, and atherosclerotic lesions of the extremities are discussed later in this chapter. Renovascular disease (renal artery stenosis and end-stage kidney disease) is discussed in Chapter 48.

## Medical Management

The management of atherosclerosis involves modification of risk factors, a controlled exercise program to improve circulation and functional capacity, medication therapy, and interventional or surgical procedures.

## Surgical Management

Vascular surgical procedures are divided into two groups: inflow procedures, which improve blood supply from the aorta into the femoral artery, and

outflow procedures, which provide blood supply to vessels below the femoral artery. Inflow surgical procedures are described with diseases of the aorta and outflow procedures with peripheral artery disease.

## Endovascular Therapy

**Endovascular** therapies include various procedures that use a puncture or small incision to place catheters inside a blood vessel to repair it or insert a device and have replaced a large proportion of open surgical approaches to management. If an isolated lesion or lesions are identified on imaging, **angioplasty**, also called *percutaneous transluminal angioplasty* (PTA), or an atherectomy, may be performed. After the patient receives a local anesthetic agent, a balloon-tipped catheter is maneuvered across the area of stenosis. Although some clinicians theorize that PTA improves blood flow by overstretching (and thereby dilating) the elastic fibers of the nondiseased arterial segment, most believe that the procedure widens the arterial lumen by “cracking” and flattening the plaque against the vessel wall (see [Chapter 23](#)). An **atherectomy** reduces the plaque buildup within an artery using a cutting device or laser. Complications from PTA and atherectomy include hematoma formation, **embolus** (blood clot, fatty deposit, or air that travels through the blood, lodges in an artery or vein, and blocks flow), **dissection** (separation of the intima) of the vessel, acute arterial occlusion, and bleeding. To decrease the risk of restenosis, stents (small mesh tubes made of nitinol, titanium, or stainless steel) may be inserted to support the walls of the artery and prevent collapse immediately after balloon deflation (see [Fig. 26-8](#)). A variety of stents and stent grafts may be used for short-segment stenoses. Complications associated with stent or stent graft use include distal embolization, dissection, and dislodgment. The advantage of angioplasty, atherectomy, stents, and stent grafts is a decreased length of hospital stay; many of the procedures are performed on an outpatient basis.

## Nursing Management

An overview of the care of a patient with peripheral artery problems is provided in [Chart 26-4](#).

### Improving Peripheral Arterial Circulation

Arterial blood supply to a body part can be enhanced by positioning the body part below the level of the heart. For the lower extremities, this is accomplished by elevating the head of the patient’s bed or by having the patient use a reclining chair or sit with the feet resting on the floor.



### Concept Mastery Alert

For patients with PAD, blood flow to the lower extremities needs to be enhanced; therefore, the nurse encourages keeping the lower extremities in a neutral or dependent position. In contrast, for patients with venous insufficiency, blood return to the heart needs to be enhanced, so the lower extremities are elevated. Exercise promotes the development of collateral circulation (arterial) and activates the musculovenous pump (venous).

The nurse can assist the patient with walking or other moderate or graded isometric exercises that promote blood flow and encourage the development of collateral circulation. The amount of exercise a patient can tolerate before the onset of pain is determined to provide a baseline for evaluation. The nurse instructs the patient to walk to the point of pain, rest until the pain subsides, and then resume walking so that endurance can be increased as collateral circulation develops. Pain can serve as a guide in determining the appropriate amount of exercise. The onset of pain indicates that the tissues are not receiving adequate oxygen, signaling the patient to rest. A supervised exercise therapy (SET) program should be prescribed for patients with claudication. SET can result in increased walking distance before the onset of claudication (Gerhard-Herman et al., 2016).

Before recommending any exercise program or SET, the patient's primary provider should be consulted. Conditions that worsen with exercise include leg ulcers, cellulitis, gangrene, or acute thrombotic occlusions.

Chart 26-4



## PLAN OF NURSING CARE

## **The Patient with Peripheral Vascular Problems**

**Nursing Diagnosis:** Impaired peripheral tissue perfusion associated with compromised circulation

**Goal:** Increased arterial blood supply to extremities

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Lower the extremities below the level of the heart (if condition is arterial).</li> <li>2. Encourage moderate amount of walking or enrollment in supervised exercise therapy program if no contraindications exist.</li> </ol>	<ol style="list-style-type: none"> <li>1. Dependency of lower extremities enhances arterial blood supply.</li> <li>2. Exercise promotes blood flow and the development of collateral circulation.</li> </ol>	<ul style="list-style-type: none"> <li>• Has extremities warm to touch</li> <li>• Has extremities with improved color</li> <li>• Experiences decreased muscle pain with exercise</li> <li>• Able to walk further distances</li> </ul>

**Goal:** Decrease in venous congestion

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Elevate the extremities above the level of the heart (if condition is venous).</li> <li>2. Discourage standing still or sitting for prolonged periods.</li> <li>3. Encourage walking.</li> </ol>	<ol style="list-style-type: none"> <li>1. Elevation of extremities counteracts gravity, promotes venous return, and prevents venous stasis.</li> <li>2. Prolonged standing still or sitting promotes venous stasis.</li> <li>3. Walking promotes venous return by activating the "calf muscle pump."</li> </ol>	<ul style="list-style-type: none"> <li>• Elevates the extremities as prescribed</li> <li>• Has decreased edema of extremities</li> <li>• Avoids prolonged standing still or sitting</li> <li>• Gradually increases walking time daily</li> </ul>

**Goal:** Promotion of vasodilation and prevention of vascular compression

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>1. Maintain warm temperature and avoid chilling.</li> <li>2. Discourage use of nicotine products.</li> <li>3. Counsel to avoid emotional upsets;</li> </ol>	<ol style="list-style-type: none"> <li>1. Warmth promotes arterial flow by preventing vasoconstriction from chilling.</li> <li>2. Nicotine in all tobacco products causes vasospasm, which</li> </ol>	<ul style="list-style-type: none"> <li>• Protects extremities from cold</li> <li>• Avoids all nicotine products</li> <li>• Uses stress management to</li> </ul>

encourage stress management.	impedes peripheral circulation.	minimize emotional upset
4. Encourage avoidance of constrictive clothing and accessories.	3. Emotional stress causes peripheral vasoconstriction by stimulating the sympathetic nervous system.	<ul style="list-style-type: none"> <li>• Avoids constrictive clothing and accessories</li> </ul>
5. Encourage avoidance of crossing the legs.	4. Constrictive clothing and accessories impede circulation and promote venous stasis.	<ul style="list-style-type: none"> <li>• Avoids crossing legs</li> </ul>
6. Administer vasodilator medications and adrenergic-blocking agents as prescribed, with appropriate nursing considerations.	5. Crossing the legs causes compression of vessels with subsequent impairment of circulation, resulting in venous stasis.	<ul style="list-style-type: none"> <li>• Takes medication as prescribed</li> </ul>
	6. Vasodilators relax smooth muscle; adrenergic-blocking agents block the response to sympathetic nerve impulses or circulating catecholamines.	

**Nursing Diagnosis:** Chronic pain associated with impaired ability of peripheral vessels to supply tissues with oxygen

**Goal:** Relief of pain

Nursing Interventions	Rationale	Expected Outcomes
1. Promote increased circulation through exercise (e.g., walking, upper extremity exercises, water aerobics, stationary cycling, supervised exercise therapy).	1. Enhancement of peripheral circulation increases the oxygen supplied to the muscle and decreases the accumulation of metabolites that cause muscle spasms.	<ul style="list-style-type: none"> <li>• Uses measures to increase arterial blood supply to extremities</li> </ul>
2. Administer analgesic agents as prescribed, with appropriate nursing considerations.	2. Analgesic agents help reduce pain and allow the patient to participate in activities and exercises that promote circulation.	<ul style="list-style-type: none"> <li>• Uses analgesic agents as prescribed</li> </ul>

**Nursing Diagnosis:** Risk for impaired skin integrity associated with compromised circulation

**Goal:** Attainment/maintenance of tissue integrity

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>Instruct in ways to avoid trauma to extremities.</li> <li>Encourage wearing protective shoes and padding for pressure points; wear new shoes for short period of time and then inspect feet for signs of injury.</li> <li>Encourage meticulous hygiene: bathing with neutral soaps, applying lotions (avoiding application between toes), and carefully trimming nails; see podiatrist for nail care.</li> <li>Use caution to avoid scratching or vigorous rubbing.</li> <li>Promote good nutrition; adequate intake of vitamins A and C, protein, and zinc; and weight reduction, if the patient is overweight or has obesity.</li> </ol>	<ol style="list-style-type: none"> <li>Poorly nourished tissues are susceptible to trauma and microbial invasion; wound healing is delayed or inhibited due to poor tissue perfusion.</li> <li>Protective shoes and padding prevent foot injuries.</li> <li>Neutral soaps and lotions prevent drying and cracking of skin; lotion application between toes increases moisture, which can lead to maceration of tissue.</li> <li>Scratching and rubbing can cause skin abrasions and microbial invasion.</li> <li>Good nutrition promotes healing and prevents tissue breakdown.</li> </ol>	<ul style="list-style-type: none"> <li>• Inspects skin daily for evidence of injury or ulceration</li> <li>• Avoids trauma and irritation to skin</li> <li>• Wears protective shoes</li> <li>• Adheres to meticulous hygiene regimen</li> <li>• Eats a healthy diet that contains adequate protein, zinc, and vitamins A and C</li> </ul>

**Nursing Diagnosis:** Lack of knowledge regarding self-care activities

**Goal:** Adherence to the self-care program

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> <li>Include family/significant others in education.</li> <li>Provide written instructions about foot and leg care and exercise therapy program.</li> <li>Assist to obtain properly fitting clothing, shoes, and stockings.</li> <li>Refer to self-help groups as indicated, such as smoking cessation clinics or stress management,</li> </ol>	<ol style="list-style-type: none"> <li>Adherence to the self-care program is enhanced when the patient receives support from family and from appropriate self-help groups and agencies.</li> <li>Written instructions serve as a reminder and reinforcement of information.</li> <li>Constrictive clothing and accessories impede circulation and promote venous stasis.</li> </ol>	<ul style="list-style-type: none"> <li>• Practices frequent position changes as prescribed</li> <li>• Practices postural exercises as prescribed</li> <li>• Takes medications as prescribed</li> <li>• Avoids vasoconstrictors</li> </ul>

weight management, and supervised exercise therapy programs.	4. Reducing risk factors may reduce symptoms or slow disease progression.	<ul style="list-style-type: none"> <li>• Uses measures to prevent trauma</li> <li>• Uses stress management</li> <li>• Accepts condition as chronic but amenable to therapies that will decrease symptoms</li> </ul>
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## Promoting Vasodilation and Preventing Vascular Compression

Arterial dilation increases blood flow to the extremities and is therefore a goal for patients with PAD. However, if the arteries are severely sclerosed, inelastic, or damaged, dilation is not possible. For this reason, measures to promote vasodilation, such as medications, endovascular interventions or surgery, may be only minimally effective.

Nursing interventions may involve applications of warmth to promote arterial flow and instructions to the patient to avoid exposure to cold temperatures, which causes vasoconstriction. Adequate clothing and warm temperatures protect the patient from chilling.



### Quality and Safety Nursing Alert

*Patients are instructed to test the temperature of bath water and to avoid using hot-water bottles and heating pads on the extremities. It is safer to apply a hot-water bottle or a heating pad to the abdomen; this can cause reflex vasodilation in the extremities.*

In patients with vasospastic disorders (e.g., Raynaud's disease), heat may be applied directly to ischemic extremities using a warmed or electric blanket; however, the temperature of the heat source must not exceed body temperature. Even at low temperatures, trauma to the tissues can occur in ischemic extremities.



### Quality and Safety Nursing Alert

*Excess heat may increase the metabolic rate of the extremities and the need for oxygen beyond that provided by the reduced arterial flow through the diseased artery. Heat must be used with great caution!*

Nicotine from any tobacco product causes vasospasm and can thereby dramatically reduce circulation to the extremities. Tobacco smoke also impairs transport and cellular use of oxygen and increases blood viscosity. Patients with arterial insufficiency who smoke, chew tobacco, or use electronic nicotine delivery systems (ENDS), including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars, must be fully informed of the effects of nicotine on circulation and encouraged to stop.

Emotional stress can stimulate the sympathetic nervous system and cause peripheral vasoconstriction. Emotional stress can be minimized to some degree by avoiding stressful situations when possible or by consistently following a stress management program. Counseling services or use of alternative or complementary therapies (e.g., relaxation, yoga, aromatherapy, mindfulness) may be indicated for patients who cannot cope effectively with situational stressors.

Constrictive clothing and accessories such as tight socks or shoelaces may impede arterial circulation to the extremities and promote venous stasis and therefore should be avoided. Crossing the legs for more than 15 minutes at a time should be discouraged because it compresses vessels in the legs.

## **Relieving Pain**

Frequently, the pain associated with peripheral arterial insufficiency is chronic, continuous, and disabling. It limits activities, affects work and life responsibilities, disturbs sleep, and alters the patient's sense of well-being. Patients may be depressed, irritable, and unable to exert the energy necessary to execute prescribed therapies, making pain relief even more difficult. Analgesic agents such as hydrocodone plus acetaminophen, oxycodone, oxycodone plus acetylsalicylic acid, or oxycodone plus acetaminophen may be helpful in reducing pain so that the patient can participate in therapies that can increase circulation and ultimately relieve pain more effectively. Particularly in older patients, these medications can be dangerous, contributing to delirium and falls. In all patients, issues of dependence need to be considered.

## **Maintaining Tissue Integrity**

Poorly perfused tissues are susceptible to damage and infection. Patients with peripheral vascular disease and diabetes are at increased risk. When lesions develop, healing may be delayed or inhibited because of the poor blood supply to the area. Infected, nonhealing ulcerations of the extremities can be

debilitating and may require prolonged and often expensive treatments. Amputation of an ischemic toe, forefoot or limb may eventually be necessary. Measures to prevent these complications must be a high priority and vigorously implemented. Centers of excellence for the prevention of amputation which involve a multidisciplinary, specialty team approach to management have become increasingly important for early intervention and surveillance.

Trauma to the extremities must be avoided. Advising the patient to wear sturdy, well-fitting shoes or slippers to prevent injury to the skin may be helpful, and recommending neutral soaps and body lotions may prevent drying and cracking of skin. However, the nurse should instruct the patient not to apply lotion between the toes, because the increased moisture can lead to maceration of the interdigital skin. Scratching and vigorous rubbing can abrade skin and create sites for microbial invasion; therefore, feet should be patted dry. Stockings should be clean and dry. Fingernails and toenails should be carefully trimmed straight across and sharp corners filed to follow the contour of the nail. If the nails cannot be trimmed safely, it is necessary to consult a podiatrist, who can also remove corns and calluses. Special shoe inserts may be needed to prevent calluses from recurring. Blisters, ingrown toenails, infection, or other problems should be reported to health care professionals for treatment and follow-up. Patients with diminished vision and those with disability that limits mobility of the arms or legs may require assistance in periodically examining the lower extremities for trauma or evidence of inflammation or infection.

Good nutrition promotes healing and prevents tissue breakdown and is therefore included in the plan for patients with peripheral vascular disease. Eating a diet that contains adequate protein and vitamins is necessary for patients with arterial insufficiency. Key nutrients, such as vitamin C and zinc, play specific roles in wound healing. However, a meta-analysis of randomized controlled trials found no evidence to support that supplementation with vitamins and antioxidants prevents vascular diseases (Sultan, Murarka, Jahangir, et al., 2017). Obesity strains the heart, increases venous congestion, and reduces circulation; therefore, a weight reduction plan may be necessary for patients who are overweight or have obesity. A diet low in fats and lipids is indicated for patients with atherosclerosis.



## Gerontologic Considerations

In older adults, the symptoms of PAD may be more pronounced than in younger people. In older patients who are inactive, limb ischemia or gangrene may be the first sign of disease (Schorr, Treat-Jacobson, Lindquist, et al., 2017). See the Nursing Research Profile in [Chart 26-5](#). These patients may have adjusted their lifestyle to accommodate the limitations imposed by the

disease and may not walk far enough to develop symptoms of claudication due to other comorbid conditions such as chronic obstructive pulmonary disease (COPD) or heart failure. Circulation is decreased, although this is not apparent to the patient until trauma occurs. At this point, gangrene develops when minimal arterial flow is impaired further by edema formation resulting from the traumatic event.

Intermittent claudication may occur after walking only one half to one block or after walking up a slight incline. Any prolonged pressure on the foot can cause pressure injuries that may become ulcerated, infected, or gangrenous. The outcomes of arterial insufficiency can include reduced mobility and activity as well as a loss of independence. Older adults with reduced mobility are less likely to remain in the community setting, have higher rates of hospitalizations, and experience a poorer quality of life. Those with cognitive impairment may also be unable to verbalize symptoms such as pain.

**Chart 26-5**



## **NURSING RESEARCH PROFILE**

## **Understanding the Relationship Between Peripheral Artery Disease (PAD) Symptoms and Ischemia**

Schorr, E. N., Treat-Jacobson, D., Lindquist, R. (2017). The relationship between peripheral artery disease symptomatology and ischemia. *Nursing Research*, 66(5), 378–387.

### **Purpose**

Peripheral artery disease (PAD) impacts over eight million Americans and is associated with an increased risk for cardiovascular disease and mortality. It is typically identified when patients report claudication (i.e., aching, cramping pain, fatigue in calves with activity). However, less than 33% of individuals who have a diagnosis of PAD report classic claudication, potentially resulting in underdiagnosis of PAD. A better understanding of symptom presentation and changes in tissue oxygenation may enhance earlier detection and treatment of PAD. The aim of this study was to explore the range of symptoms (typical versus atypical, location, and description) that patients with PAD experience to better understand the relationship between symptom variation and calf muscle ischemia.

### **Design**

This descriptive study explored symptoms during exercise testing and recovery in patients with a diagnosis of PAD. Individuals who were English speaking, 21 years of age or older, and met specific diagnostic criteria were recruited from a larger study. Participants with uncontrolled hypertension, angina or dyspnea on exercise testing, or vascular procedures performed within the last 3 months were excluded. Demographic and clinical data were collected. Ankle-brachial index (ABI) was used as a measure of disease severity. Participants exercised on a treadmill with a near-infrared spectroscopy device to measure calf muscle tissue saturation index (TSI). In addition, participants rated symptom intensity using a numerical rating scale (NRS) and provided self-report of symptom location and descriptors. Data were collected at three points (e.g., rest, exercise, recovery) during three successive treadmill tests. Descriptive statistics were generated and symptom variables and relevant demographic and clinical data (e.g., age, gender, race, ABI, diabetes, neuropathy) were examined with the TSI measurements during exercise and recovery using multilevel modeling procedures.

### **Findings**

Three successive episodes of treadmill testing with 40 participants resulted in 120 exercise tests. The majority of participants were Caucasian males (80%) with an average age of 68 years (SD 0.92). More than half (69.2%) of tests were stopped due to discomfort in the calf, with only 55% of participants reporting typical descriptors of claudication. TSI declined rapidly between the start of exercise and symptom onset. The lowest TSI was often reached

before reported maximum discomfort. Changes in TSI were related to exercise time ( $p < 0.001$ ), baseline TSI ( $p < 0.001$ ), exercise rating ( $p < 0.001$ ), and ABI ( $p < 0.5$ ). In the recovery phase, TSI steadily increased as pain reduced; TSI was associated with recovery rating ( $p < 0.001$ ) and ABI ( $p < 0.03$ ).

### Nursing Implications

Recognizing variations in symptom presentation (e.g., intensity, location, description) and associated ischemic changes in PAD is important in understanding individual patient experiences and tailoring education and treatment. Nurses should be aware that some patients with PAD may use terms such as *burn*, *pressure*, or *tight* to describe related discomfort and that patients may report discomfort in atypical locations, such as the foot. A better understanding of the full range of ischemic symptoms in PAD may prompt earlier diagnosis and facilitate risk factor modification and exercise therapy. Unlike other medical conditions, where the onset of pain indicates the potential of increasing tissue damage with ongoing activity, pain in PAD can be used as an indicator to adjust the exercise program to extend the person's walking distance. Exercise prescription can greatly reduce the progression of atherosclerosis, limiting not only distal disease but also coronary and cerebral disease. Although additional research is needed, nurses can consider these findings when planning care for patients with PAD to ensure they are providing appropriate education and encouraging safe exercise regimens.

### Promoting Home, Community-Based, and Transitional Care

The self-care program is planned with the patient so that activities that promote arterial and venous circulation, relieve pain, and promote tissue integrity are acceptable. The patient and family are helped to understand the reasons for each aspect of the plan, the possible consequences of nonadherence, and the importance of keeping follow-up appointments. Long-term care of the feet and legs is of prime importance in the prevention of trauma, ulceration, and gangrene. [Chart 26-6](#) provides detailed patient instructions for foot and leg care.

### Peripheral Artery Disease



Arterial insufficiency of the extremities occurs most often in men and is a common cause of disability. The legs are most frequently affected; however, the upper extremities may be involved. The age of onset and the severity are influenced by the type and number of atherosclerotic risk factors (see [Chart 26-3](#)). In PAD, obstructive lesions are predominantly confined to segments of

the arterial system extending from the aorta below the renal arteries to the popliteal artery (see Fig. 26-9). Distal occlusive disease is frequently seen in patients with diabetes and in older patients (Gerhard-Herman et al., 2016).

Chart 26-6



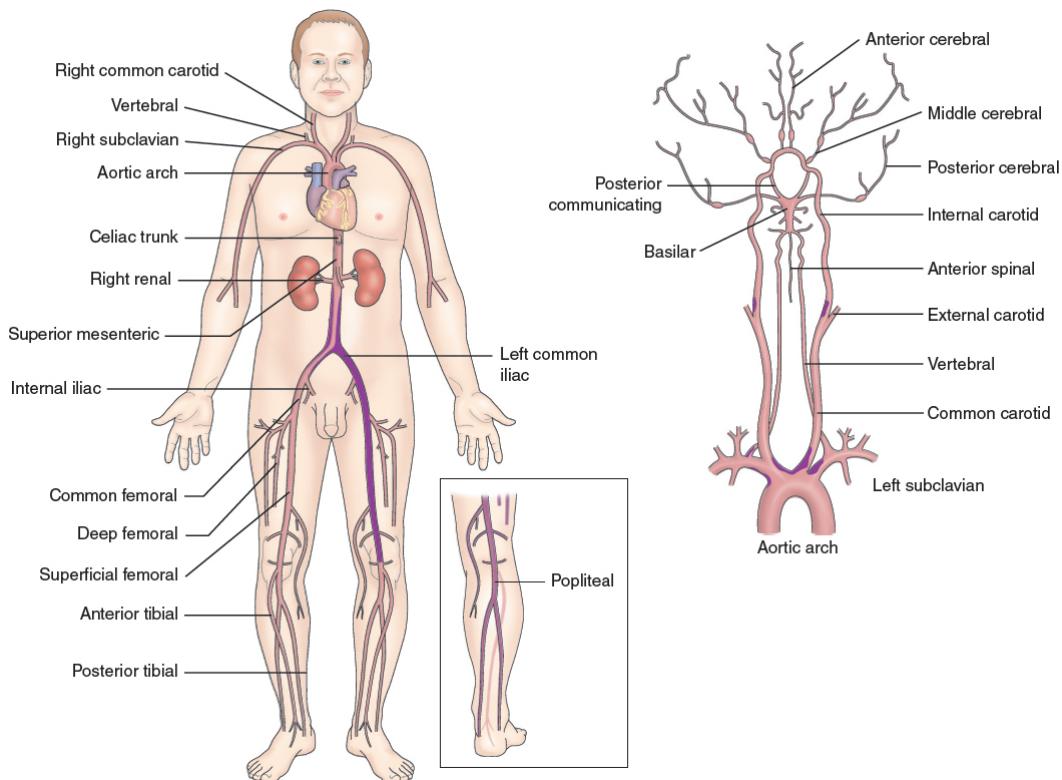
## HOME CARE CHECKLIST

## **Foot and Leg Care in Peripheral Vascular Disease**

**At the completion of education, the patient and/or caregiver will be able to:**

- Describe the rationale for proper foot and leg care in managing peripheral vascular disease.
- Demonstrate daily foot hygiene: Wash between toes with mild soap and lukewarm water, then rinse thoroughly and pat rather than rub dry.
- Recognize the dangers of thermal injury.
  - Wear clean, loose, soft cotton socks (they are comfortable, allow air to circulate, and absorb moisture).
  - In cold weather, wear extra socks in extra-large shoes.
  - Avoid heating pads, whirlpools, and hot tubs.
  - Avoid sunburn.
- Identify safety concerns.
  - Inspect feet daily with a mirror for redness, dryness, cuts, blisters, and so forth.
  - Always wear soft shoes or slippers when out of bed.
  - Trim nails straight across after showering.
  - Consult podiatrist to trim nails if vision is decreased and for care of corns, blisters, and ingrown nails.
  - Clear pathways in house to prevent injury.
  - Avoid wearing thong sandals.
  - Use lamb's wool or foam between toes if they overlap or rub each other.
- Demonstrate the use of comfort measures.
  - Wear leather shoes with an extra-depth toe box. Synthetic shoes do not allow air to circulate.
  - If feet become dry and scaly, use cream or lotion with emollient. Never put cream or lotion between toes unless it has been prescribed.
  - Avoid scratching or vigorous rubbing, which could cause abrasions.
  - If feet perspire, especially between toes, use lamb's wool between toes to promote drying.
- Demonstrate strategies to decrease risk of constricting blood vessels.
  - Avoid circumferential compression around feet or legs—for example, by knee-high stockings or tight socks or constricting bandages.
  - Do not cross legs at knees.
  - Stop using all nicotine products (i.e., smoking or chewing) because nicotine causes vasoconstriction and vasospasm.

- Participate in regular walking or supervised exercise therapy to stimulate circulation.
- Recognize when to seek medical attention.
  - Contact health care provider at the onset of skin breakdown such as abrasions, blisters, fungal infection (athlete's foot), or pain.
  - Do not use any medication on feet or legs unless prescribed.
  - Avoid using iodine, alcohol, corn/wart-removing compound, or adhesive products before checking with primary provider.
- State understanding of community resources and referrals (if any).



**Figure 26-9 •** Common sites of atherosclerotic obstruction in major arteries.

## Clinical Manifestations

The hallmark symptom is intermittent claudication described as aching, cramping, or inducing fatigue or weakness that occurs with some degree of activity or exercise, which is relieved with rest. The pain commonly occurs in muscle groups distal to the area of stenosis or occlusion. As the disease progresses, the patient may have a decreased ability to walk the same distance as before or may notice increased pain with ambulation. When the arterial insufficiency becomes severe, the patient has rest pain. This pain is associated

with critical ischemia of the distal extremity and is described as persistent, aching, or boring; it may be so excruciating that it is unrelieved by opioids and can be disabling. Ischemic rest pain is usually worse at night and often wakes the patient. Elevating the extremity or placing it in a horizontal position increases the pain, whereas placing the extremity in a dependent position reduces the pain. In an attempt to prevent or relieve the pain, some patients sleep with the affected leg hanging over the side of the bed or sleep in a reclining chair.

## Assessment and Diagnostic Findings

A sensation of coldness or numbness in the extremities may accompany intermittent claudication and is a result of reduced arterial flow. The extremity is cool and pale when elevated or ruddy and cyanotic when placed in a dependent position. Skin and nail changes, ulceration, gangrene, and muscle atrophy may be evident. Bruits may be auscultated with a stethoscope. Peripheral pulses may be diminished or absent.

Examination of the peripheral pulses is an important part of assessing PAD. Unequal pulses between extremities or the absence of a normally palpable pulse is a sign of PAD.

The presence, location, and extent of PAD are determined by a careful history of the symptoms and by physical examination. The color and temperature of the extremity are noted and the pulses palpated. The nails may be thickened and opaque, and the skin may be shiny, atrophic, and dry, with sparse or absent hair. The assessment includes comparison of the right and left extremities.

The diagnosis of PAD may be made using CW Doppler and ABIs, treadmill testing for claudication, duplex ultrasonography, or other imaging studies described earlier in this chapter.

## Medical Management

Generally, patients feel better and have fewer symptoms of claudication after they participate in a SET program. SET programs are covered by insurance for a specific number of sessions. Reimbursement requires that SET is administered under direct provider supervision. A provider (e.g., physician, nurse practitioner, clinical nurse specialist, physician assistant) must be immediately and physically available, although not necessarily physically present, while SET is provided. The person providing the program supervision must be trained in the optimal delivery of SET and in both basic life support and advanced cardiac life support techniques. Unsupervised walking exercise programs are attractive for many patients with PAD with limited access to a SET program. Two recent trials in patients with PAD had similar findings

between the supervised and unsupervised groups, suggesting no greater therapeutic benefit for those who engage in supervised walking programs (McDermott, 2018). These findings suggest that home-based programs may be a viable and efficacious option for patients unable to participate in a structured, on-site, supervised exercise program. If a walking program is combined with weight reduction and cessation of nicotine use, patients often can further improve their activity tolerance. Patients should not be promised that their symptoms will be relieved if they stop nicotine use, however, because claudication may persist, and they may lose their motivation to stop using nicotine. In addition to these interventions, arm-ergometer exercise training effectively improves physical fitness, central cardiorespiratory function, and walking capacity in patients with claudication (Treat-Jacobson, McDermott, Beckman, et al., 2019).

### Pharmacologic Therapy

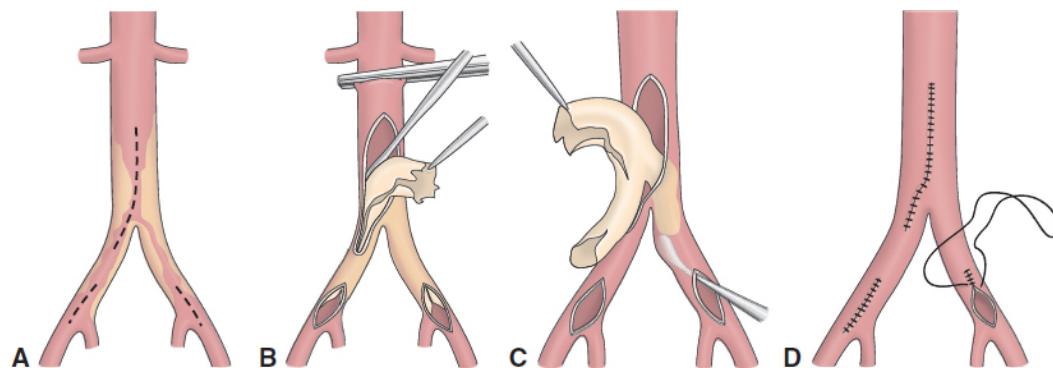
Cilostazol is approved by the U.S. Food and Drug Administration (FDA) for the treatment of claudication. Cilostazol, a phosphodiesterase III inhibitor, is a direct vasodilator that inhibits platelet aggregation. Studies have shown it plays a role in decreasing intimal hyperplasia after angioplasty and stenting. Furthermore, patients prescribed cilostazol report improvement in maximal walking distance and pain-free walking distance within 4 to 6 weeks (Farkas, Járai, & Kolossváry, 2017). This agent is contraindicated in patients with a history of heart failure.

Antiplatelet agents, such as aspirin or clopidogrel, prevent the formation of thromboemboli which can lead to myocardial infarction and stroke and are recommended to treat patients with symptomatic PAD (Gerhard-Herman et al., 2016). Aspirin has been shown to reduce the risk of cardiovascular events (e.g., myocardial infarction, stroke, cardiovascular death) in patients with vascular disease; however, adverse events associated with aspirin use include gastrointestinal upset or bleeding (Gerhard-Herman et al., 2016).

Use of dual antiplatelet agents, such as aspirin and clopidogrel, in patients with symptomatic PAD has not been well established. However, dual antiplatelet agents may be effective and may be reasonable to reduce limb-threatening events after revascularization. Statins improve endothelial function in patients with PAD. Studies suggest that statins reduce severity of intermittent claudication and increase walking distance to the onset of claudication (Gerhard-Herman et al., 2016). These medications have beneficial effects on vascular inflammation, plaque stabilization, endothelial dysfunction, and thrombosis, and have been linked to decreased rates of repeat peripheral interventions, amputations, and major adverse cardiovascular events up to 3 years post procedure (Saxon, Safley, & Mena-Hurtado, 2020).

### Endovascular Management

Endovascular interventions can include a balloon angioplasty, stent, stent graft, or an atherectomy. These revascularization procedures are less invasive than conventional surgery; their objective is to establish adequate inflow to the distal vessels. A meta-analysis reported that the efficacy and safety of all of these endovascular procedures are comparable to surgical interventions. Some stents that may be selected are drug eluting. Although costly, these are particularly efficacious in patients who have recurrent disease. By releasing antiproliferative drugs, drug-eluting balloons and stents have been shown to reduce the risk of restenosis. Eligible candidates for drug-eluting stents must be able to take antiplatelet medications for at least 6 months post procedure (Sidawy & Perler, 2019).



**Figure 26-10 •** In an aortoiliac endarterectomy, the vascular surgeon identifies the diseased area (A), clamps off the blood supply to the vessel (B), removes the plaque (C), and sutures the vessel shut (D), after which blood flow is restored. Adapted with permission from Sidawy, A. N., & Perler, B. A. (2019). *Rutherford's vascular surgery and endovascular therapy* (9th ed.). Philadelphia, PA: Elsevier.

## Surgical Management

Surgery is reserved for the treatment of rest pain, severe and disabling claudication, or when the limb is at risk for amputation because of tissue necrosis. The choice of the surgical procedure depends on the degree, length, and location of the stenosis or occlusion and whether there are single or multiple lesions. Other important considerations are the overall health of the patient, the length of the procedure, and anesthesia required. If endarterectomy is performed, an incision is made into the artery and the atheromatous obstruction is removed (see Fig. 26-10).

Bypass grafts are performed to reroute the blood flow around the stenosis or occlusion. Before bypass grafting, the surgeon determines where the distal **anastomosis** (site where the vessels are surgically joined) will be placed. The distal outflow vessel must be at least 50% patent for the graft to remain open.

If the atherosclerotic occlusion is below the inguinal ligament in the superficial femoral artery, the surgical procedure of choice is the femoral-to-popliteal graft. This procedure is further classified as above- and below-knee grafts, referring to the location of the distal anastomosis. Bypass grafts may be synthetic materials or autologous vein. Several synthetic materials are available for use as a peripheral bypass graft: woven or knitted Dacron or expanded polytetrafluoroethylene (PTFE). Cryopreserved saphenous veins and umbilical veins are also available. When using an autologous conduit (i.e., the patient's own veins), the vein is either grafted to the artery in situ (the vein remains in place with the valves stripped and the vein is anastomosed to the proximal and distal target arteries), or the vein is harvested, reversed, and anastomosed to the proximal and distal target arteries.

Lower leg or ankle vessels with occlusions may also require grafts. Occasionally, the popliteal artery is completely occluded and only collateral vessels maintain perfusion. The distal anastomosis may be made onto any of the tibial arteries (posterior tibial, anterior tibial, or peroneal arteries) or the dorsalis pedis or plantar artery. The distal anastomosis site is determined by the ease of exposure of the vessel in surgery and by which vessel provides the best flow to the distal limb. These grafts require the use of an autologous vein to ensure patency. The greater or lesser saphenous vein or a combination of one of the saphenous veins and an upper extremity vein such as the cephalic vein is used to provide the required length.

How long the graft remains patent is determined by several factors, including the size of the graft, graft location, and development of intimal hyperplasia at anastomosis sites (Sidawy & Perler, 2019). Infection of synthetic grafts may result in sepsis and almost always requires removal.

If a vein graft is the surgical choice, care must be taken in the operating room not to damage the vein after harvesting (removing the vein from the patient's body). The vein is occluded at one end and inflated with a heparinized solution to check for leakage and competency. The graft is then placed in a heparinized solution to keep it from becoming dry and brittle until use during the operative procedure.

For patients who cannot tolerate an extensive vascular surgical procedure, a palliative approach involving primary amputation rather than an endarterectomy or bypass may be considered (Conte et al., 2019).

## Nursing Management

Nursing care for patients with PAD is reviewed in [Chart 26-4](#). Nursing care for the patient who has had endovascular revascularization procedures mostly mirrors the care of patients who have had endovascular repair to aortic aneurysms (see later section). The patient who has had an endovascular

procedure may be discharged home the day of the procedure, or by the following day.

## Nursing Care of the Postoperative Patient

During the postoperative period, the nurse's care focuses on maintaining circulation, identifying and managing potential complications, and discharge planning.

### Maintaining Circulation

The primary objective in the postoperative period is to maintain adequate circulation through the arterial repair. Pulses, Doppler assessment, color and temperature, capillary refill, and sensory and motor function of the affected extremity are checked and compared with those of the other extremity; these observations are recorded initially every 15 minutes and then at progressively longer intervals if the patient's status remains stable. Doppler evaluation of the vessels distal to the bypass graft should be performed, because it is more sensitive than palpation for pulses. The ABI is monitored at least once every 8 hours for the first 24 hours (not usually assessed with pedal artery bypasses due to the risk of compression of the anastomosis by the cuff) and then once each day until discharge; the typical hospital length of stay is 3 to 5 days. An adequate circulating blood volume should be established and maintained. Disappearance of a pulse that was present may indicate thrombotic occlusion of the graft; the surgeon is immediately notified.

### Monitoring and Managing Potential Complications

Continuous monitoring of urine output, central venous pressure, mental status, and pulse rate and volume permits early recognition and treatment of fluid imbalances. Bleeding can result from the heparin given during surgery or from an anastomotic leak. A hematoma may form as well. The nurse should review the operative report to determine if the heparin was reversed (usually with protamine sulfate) in the operating room.

Leg crossing and prolonged extremity dependency are avoided to prevent thrombosis. Edema is a normal postoperative finding due to increased arterial flow; however, elevating the extremities and encouraging the patient to exercise the extremities while in bed reduces edema. Graduated compression or anti-embolism stockings may be prescribed for some patients, but care must be taken to avoid compressing distal vessel bypass grafts, inducing pressure injuries, and obscuring visualization of the extremity. Severe edema of the extremity, pain, and decreased sensation of toes or fingers can be an indication of compartment syndrome (see [Chapter 37](#)).

### Promoting Home, Community-Based, and Transitional Care

Discharge planning includes assessing the patient's ability to manage activities of daily living (ADLs) independently. The nurse determines whether the patient has a network of family and friends to assist with ADLs. The patient is encouraged to make the lifestyle changes necessitated by the onset of disease, including pain management and modifications in diet, activity, and hygiene (skin care). The nurse ensures that the patient has the knowledge and ability to assess for any postoperative complications such as infection, occlusion of the artery or graft, and decreased blood flow. The nurse assists the patient in developing and implementing a plan to stop using tobacco products.

## Upper Extremity Arterial Disease

Arterial stenosis and occlusions occur less frequently in the upper extremities (arms) than in the legs, and cause less severe symptoms because the collateral circulation is significantly better in the arms. The arms also have less muscle mass and are not subjected to the workload of the legs.

## Clinical Manifestations

Stenosis and occlusions in the upper extremity result from atherosclerosis or trauma. The stenosis usually occurs at the origin of the vessel proximal to the vertebral artery, which results in the vertebral artery becoming the dominant vessel for blood flow. The patient typically complains of arm fatigue and pain with exercise (forearm claudication), inability to hold or grasp objects (e.g., combing hair, placing objects on shelves above the head), and occasionally difficulty driving.

The patient may develop a “subclavian steal” syndrome characterized by reverse flow in the vertebral and basilar arteries to provide blood flow to the arm. This syndrome may cause vertebrobasilar (cerebral) symptoms, including vertigo, ataxia, syncope, or bilateral visual changes.

## Assessment and Diagnostic Findings

Findings on assessment include coolness and pallor of the affected extremity, decreased capillary refill, and a difference in arm blood pressures of more than 15 to 20 mm Hg (Zierler & Dawson, 2016). Noninvasive studies performed to evaluate upper extremity arterial occlusions include upper and forearm blood pressure determinations and duplex ultrasonography to identify the anatomic location of the lesion and to evaluate the hemodynamics of blood flow. Transcranial Doppler evaluation is performed to evaluate the intracranial circulation and to detect any siphoning of blood flow from the posterior

circulation to provide blood flow to the affected arm. If an endovascular or surgical procedure is planned, a diagnostic arteriogram may be necessary.

## Medical Management

If a short focal lesion is identified in an upper extremity artery, a PTA with possible stent or stent graft placement may be performed. If the lesion involves the subclavian artery with documented siphoning of blood flow from the intracranial circulation and an endovascular procedure is not possible, a surgical bypass may be performed.

## Nursing Management

Nursing assessment involves bilateral comparison of upper arm blood pressures (obtained by stethoscope and Doppler), radial, ulnar, and brachial pulses, motor and sensory function, temperature, color changes, and capillary refill every 2 hours. Disappearance of a pulse or Doppler flow that had been present may indicate an acute occlusion of the vessel, and the primary provider is notified immediately.

After surgery or an endovascular procedure, the arm is kept at heart level or elevated, with the fingers at the highest level. Pulses are monitored with Doppler assessment of the arterial flow every hour for 2 hours and then every shift. Blood pressure (obtained by stethoscope and Doppler) is also assessed every hour for 4 hours and then every shift. Motor and sensory function, warmth, color, and capillary refill are monitored with each arterial flow (pulse) assessment.



### Quality and Safety Nursing Alert

*Before surgery and for 24 hours after surgery, the patient's arm is kept at heart level and protected from cold, venous and arterial punctures, tape, pressure, and constrictive dressings.*

Discharge planning is similar to that for the patient with PAD. [Chart 26-4](#) describes nursing care for patients with peripheral vascular disease.

## Aortoiliac Disease

If collateral circulation has developed, patients with a stenosis or occlusion of the aortoiliac segment may be asymptomatic, or they may complain of buttock or low back discomfort associated with walking. Men may have erectile

dysfunction or experience impotence. These patients may have decreased or absent femoral pulses.

## Medical Management

The treatment of aortoiliac disease is essentially the same as that for atherosclerotic PAD. An endovascular procedure, such as bilateral common iliac stents, may be attempted if the aorta has a less than 50% diameter reduction (Gerhard-Herman et al., 2016). If there is significant aortic disease, the surgical procedure of choice is the aortoiliac graft. If possible, the distal graft is anastomosed to the iliac artery, and the entire surgical procedure is performed within the abdomen. If the iliac vessels are occluded, the distal anastomosis is made to the femoral arteries (aortobifemoral graft). A femoralfemoral crossover graft may also be needed to maintain circulation. Bifurcated woven or knitted Dacron grafts are preferred for this surgical procedure.

## Nursing Management

Preprocedural or preoperative assessment, in addition to the standard parameters (see [Chapter 14](#)), includes evaluating the brachial, radial, ulnar, femoral, popliteal, posterior tibial, and dorsalis pedis pulses to establish a baseline for follow-up after arterial lines are placed and postoperatively. Patient education includes an overview of the procedure to be performed, the preparation for an endovascular procedure or surgery, and the anticipated postprocedural or postoperative plan of care. Sights, sounds, and sensations that the patient may experience are discussed.

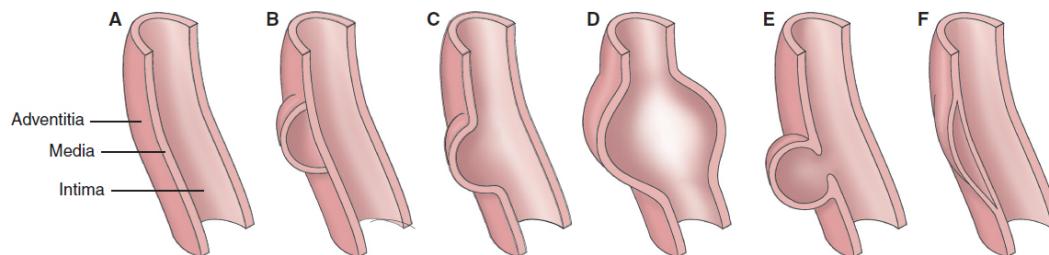
Postprocedural endovascular care mirrors the care described for the patient who has had endovascular repair of an aortic aneurysm (see later discussion). Postoperative care includes monitoring for signs of thrombosis in arteries distal to the surgical site. The nurse assesses color and temperature of the extremity, capillary refill time, sensory and motor function, and pulses by palpation and Doppler initially every 15 minutes and then at progressively longer intervals if the patient's status remains stable. Any dusky or bluish discoloration, coldness, decrease in sensory or motor function, or decrease in pulse quality is reported immediately to the primary provider.

Postoperative care also includes monitoring urine output and ensuring that output is at least 0.5 mL/kg/h. Renal function may be impaired as a result of hypoperfusion from hypotension, ischemia to the renal arteries during the surgical procedure, hypovolemia, or embolization of the renal artery or renal parenchyma. Vital signs, pain, and intake and output are monitored with the pulse and extremity assessments. Results of laboratory tests are monitored and reported to the primary provider. Abdominal assessment for bowel sounds and paralytic ileus is performed at least every 8 hours. Bowel sounds may not

return before the third postoperative day. The absence of bowel sounds, absence of flatus, and abdominal distention are indications of paralytic ileus. Manual manipulation of the bowel during surgery may have caused bruising, resulting in decreased peristalsis. Nasogastric suction may be necessary to decompress the bowel until peristalsis returns. A liquid bowel movement before the third postoperative day may indicate bowel ischemia, which may occur when the mesenteric blood supply (celiac, superior mesenteric, or inferior mesenteric arteries) is occluded. Ischemic bowel usually causes increased pain and a markedly elevated white blood cell count (20,000 to 30,000 cells/mm<sup>3</sup>).

## Aneurysms

An aneurysm is a localized sac or dilation formed at a weak point in the wall of the artery (see Fig. 26-11). It may be classified by its shape or form. The most common forms of aneurysms are saccular and fusiform. A saccular aneurysm projects from only one side of the vessel. If an entire arterial segment becomes dilated, a fusiform aneurysm develops. Very small aneurysms due to localized infection are called *mycotic aneurysms*.



**Figure 26-11 • Characteristics of arterial aneurysm.** **A.** Normal artery. **B.** False aneurysm—actually a pulsating hematoma. The clot and connective tissue are outside the arterial wall. **C.** True aneurysm. One, two, or all three layers of the artery may be involved. **D.** Fusiform aneurysm—symmetric, spindle-shaped expansion of entire circumference of involved vessel. **E.** Saccular aneurysm—a bulbous protrusion of one side of the arterial wall. **F.** Dissecting aneurysm—this usually is a hematoma that splits the layers of the arterial wall. Adapted with permission from Sidawy, A. N., & Perler, B. A. (2019). *Rutherford's vascular surgery and endovascular therapy* (9th ed.). Philadelphia, PA: Elsevier.

Historically, the cause of abdominal aortic aneurysm, the most common type of degenerative aneurysm, has been attributed to atherosclerotic changes in the aorta. Other causes of aneurysm formation are listed in Chart 26-7.

Aneurysms are potentially serious; if they are located in large vessels that rupture, this can lead to hemorrhage and death.

### Chart 26-7

#### Etiologic Classification of Arterial Aneurysms

**Anastomotic (postarteriotomy) and graft aneurysms:** Infection, arterial wall failure, suture failure, graft failure

**Congenital:** Primary connective tissue disorders (Marfan syndrome, Ehlers–Danlos syndrome) and other diseases (focal medial agenesis, tuberous sclerosis, Turner syndrome, Menkes syndrome)

**Infectious (mycotic):** Bacterial, fungal, spirochetal infections

**Inflammatory (noninfectious):** Associated with arteritis (Takayasu disease, giant cell arteritis, systemic lupus erythematosus, Behçet syndrome, Kawasaki disease) and periarterial inflammation (i.e., pancreatitis)

**Mechanical (hemodynamic):** Poststenotic and arteriovenous fistula and amputation related

**Pregnancy-related degenerative:** Nonspecific, inflammatory variant

**Traumatic (pseudoaneurysms):** Penetrating arterial injuries, blunt arterial injuries, pseudoaneurysms

Adapted from Sidawy, A. N., & Perler, B. A. (2019). *Rutherford's vascular surgery and endovascular therapy* (9th ed.). Philadelphia, PA: Elsevier.

### Thoracic Aortic Aneurysm

Approximately 70% of all cases of thoracic aortic aneurysm are caused by atherosclerosis. They occur most frequently in men between the ages of 50 and 70 years, and are estimated to affect 10 of every 100,000 older adults. The thoracic area is the most common site for a dissecting aneurysm. Thoracic aortic emergencies are associated with high morbidity and mortality rates, but with the emergence of endovascular aortic repair there is an improvement in the mortality rate; in particular, the mortality rate for patients treated at high-volume aortic centers can be as low as 4.8% (Harris, Olson, Panthofer, et al., 2019).

### Clinical Manifestations

Symptoms vary and depend on how rapidly the aneurysm dilates and how the pulsating mass affects surrounding intrathoracic structures. Some patients are asymptomatic. In most cases, pain is the most prominent symptom. The pain is usually constant and boring but may occur only when the person is supine. Other conspicuous symptoms are dyspnea, the result of pressure of the aneurysm sac against the trachea, a main bronchus, or the lung itself; cough,

frequently paroxysmal and with a brassy quality; hoarseness, stridor, or vocal weakness or aphonia (complete loss of the voice), resulting from pressure against the laryngeal nerve; and dysphagia (difficulty in swallowing) due to impingement on the esophagus by the aneurysm.

## Assessment and Diagnostic Findings

When large veins in the chest are compressed by the aneurysm, the superficial veins of the chest, neck, or arms become dilated, and edematous areas on the chest wall and cyanosis are often evident. Pressure against the cervical sympathetic chain can result in unequal pupils. Diagnosis of a thoracic aortic aneurysm is principally made by chest x-ray, computed tomography angiography (CTA), MRA, or transesophageal echocardiography (TEE). CTAs are typically performed because they are widely available, can be completed rapidly, and can remove cardiac motion artifacts, enhancing their accuracy (Sidawy & Perler, 2019).

## Medical Management

Treatment is based on whether the patient is symptomatic and whether the aneurysm is expanding in size, caused by an iatrogenic injury, contains a dissection, or involves branch vessels. General measures such as controlling blood pressure and correcting risk factors are helpful. For decades, beta-blockers (e.g., atenolol, metoprolol, carvedilol) have been the mainstay of medical treatment for aortic aneurysms; however, angiotensin receptor blockers (ARBs) (e.g., losartan, valsartan, irbesartan) may also retard aortic dilation (Rurali, Perrucci, Pilato, et al., 2018). It is also important to control blood pressure in patients with dissecting aneurysms. Preoperatively, the systolic pressure is maintained at approximately 90 to 120 mm Hg in order to maintain a mean arterial pressure at 65 to 75 mm Hg with a beta-blocker such as esmolol or metoprolol. Occasionally, antihypertensive agents such as hydralazine are used for this purpose. Sodium nitroprusside is the most established drug used for this purpose; it is given by continuous IV drip to emergently lower the blood pressure, as it has a rapid onset and short action of duration and is easily titrated (Sidawy & Perler, 2019). The goal of surgery is to repair the aneurysm and restore vascular continuity with a vascular graft. Intensive monitoring is required after this type of surgery, and the patient is cared for in the critical care unit.

Repair of thoracic aneurysms using endovascular grafts placed percutaneously in an interventional suite (e.g., interventional radiology, cardiac catheterization laboratory) or combined interventional suite and operating room (hybrid suite) may decrease postoperative recovery time and decrease complications compared with traditional surgical techniques. Thoracic

endografts are made of PTFE material reinforced with nitinol or titanium stents. These endovascular grafts are inserted into the thoracic aorta via various vascular access routes, usually the brachial or femoral artery. Because a large surgical incision is not necessary to gain vascular access, the overall patient recovery time tends to be shorter than with open surgical repair. Despite the absence of aortic cross-clamping, there is still a 2% to 15% chance of spinal cord ischemia as a potential complication (Miranda, Sousa, & Mansilha, 2018). To decrease the chances of spinal cord ischemia and paraplegia, lumbar spinal drains are usually placed in patients undergoing an endovascular repair of thoracic aortic aneurysms. Cerebrospinal fluid drainage is performed to decrease the arterial to cerebral spinal fluid gradient, thereby improving spinal perfusion. What appears to be most important in preventing neurologic deficit is to maintain the cerebrospinal fluid pressure less than or equal to 10 mm Hg (14 cm H<sub>2</sub>O) and to keep the mean arterial pressure greater than 90 mm Hg for the first 36 to 48 hours postoperatively (Scali, Kim, Kubilis, et al., 2018).

### **Abdominal Aortic Aneurysm**

The most common cause of abdominal aortic aneurysm is atherosclerosis. This condition affects men two to six times more often than women, is two to three times more common in White versus Black men, and is most prevalent in patients older than 65 years of age (Sidawy & Perler, 2019). Most of these aneurysms occur below the renal arteries (infrarenal aneurysms). Untreated, the eventual outcome may be rupture and death.

### **Pathophysiology**

All aneurysms involve a damaged media layer of the vessel. This may be caused by congenital weakness, trauma, or disease. After an aneurysm develops, it tends to enlarge. Risk factors include genetic predisposition, nicotine use, and hypertension; more than half of patients with aneurysms have hypertension.

### **Clinical Manifestations**

Only about 40% of patients with abdominal aortic aneurysms have symptoms. Some patients complain that they can feel their heart beating in their abdomen when lying down, or they may say that they feel an abdominal mass or abdominal throbbing. If the abdominal aortic aneurysm is associated with thrombus, a major vessel may be occluded or smaller distal occlusions may result from emboli. Small cholesterol, platelet, or fibrin emboli may lodge in

the interosseous or digital arteries, causing cyanosis and mottling of the toes (also referred to as trashing or trash toes).

Signs of impending rupture include severe back or abdominal pain, which may be persistent or intermittent. Abdominal pain is often localized in the middle or lower abdomen to the left of the midline. Low back pain may be present because of pressure of the aneurysm on the lumbar nerves. Indications that the abdominal aortic aneurysm may be leaking or rupturing include constant, intense back pain; falling blood pressure; and decreasing hematocrit. Rupture into the peritoneal cavity is rapidly fatal. A retroperitoneal rupture (contained rupture) of an aneurysm may result in hematomas in the scrotum, perineum, flank, or penis. Signs of heart failure or a loud bruit may suggest a rupture into the vena cava. If the aneurysm adheres to the adjacent vena cava, the vena cava may become damaged when rupture or leak of the aneurysm occurs. Rupture into the vena cava results in higher-pressure arterial blood entering the lower-pressure venous system and causing turbulence, which is heard as a bruit. The high blood pressure and increased blood volume returning to the right side of the heart from the vena cava may cause heart failure.

## Assessment and Diagnostic Findings

The most important diagnostic indication of an abdominal aortic aneurysm is a pulsatile mass in the middle and upper abdomen. Most clinically significant aortic aneurysms are palpable during routine physical examination; however, the sensitivity depends on the size of the aneurysm, abdominal girth of the patient (i.e., more difficult to find in the patient with obesity), and the skill of the examiner (Sidawy & Perler, 2019). A systolic bruit may be heard over the mass. Duplex ultrasonography or CTA is used to determine the size, length, and location of the aneurysm. When the aneurysm is small, ultrasonography is conducted at 6-month intervals until the aneurysm reaches a size so that surgery to prevent rupture is of more benefit than the possible complications of a surgical procedure. Some aneurysms remain stable over many years of monitoring.



## Gerontologic Considerations

Most abdominal aortic aneurysms occur in patients between 60 and 90 years of age. Rupture is likely with coexisting hypertension and with aneurysms more than 6 cm wide. In most cases at this point, the chances of rupture are greater than the chances of death during surgical repair. If the older patient is considered at risk for complications related to surgery or anesthesia, the aneurysm is not repaired until it is at least 5.5 cm (2 inches) wide (Chaikof, Dalman, Eskandari, et al., 2018).

## **Medical Management**

Medical management consists of pharmacologic, endovascular, and surgical interventions.

### **Pharmacologic Therapy**

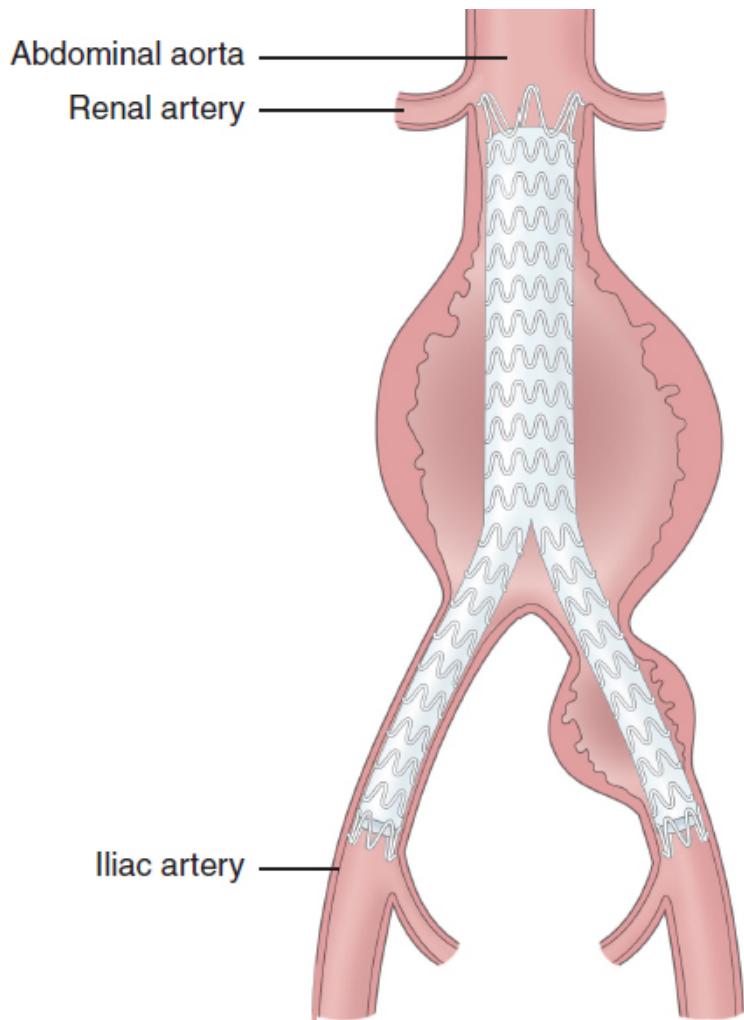
If the aneurysm is stable in size based on serial duplex ultrasound scans, the blood pressure is closely monitored over time, because there is an association between increased blood pressure and aneurysm rupture (Sidawy & Perler, 2019). Antihypertensive agents, including diuretics, beta-blockers, ACE inhibitors, ARBs, and calcium channel blockers, are frequently prescribed to maintain the patient's blood pressure within acceptable limits (see [Chapter 27](#)).

### **Endovascular and Surgical Management**

An expanding or enlarging abdominal aortic aneurysm is likely to rupture. When an abdominal aortic aneurysm measured at least 5.5 cm (2 inches) wide or was enlarging, the standard treatment had been open surgical repair of the aneurysm by resecting the vessel and sewing a bypass graft in place. However, endovascular aortic repair has become a mainstay of therapy for treating an infrarenal abdominal aortic aneurysm and involves the transluminal placement and attachment of a sutureless aortic graft across the aneurysm (see [Fig. 26-12](#)). This procedure can be performed under local or regional anesthesia. Endovascular grafting of abdominal aortic aneurysms may be performed if the patient's abdominal aorta and iliac arteries are not extremely tortuous, small, calcified, or filled with thrombi. Results from multiple, prospective studies suggest comparable mortality rates among patients with aneurysms treated by endovascular grafting and those treated with surgical repair, with similar 5-year survival rates (Chaikof et al., 2018). Potential complications include bleeding, hematoma, or wound infection at the arterial insertion site; distal ischemia or embolization; dissection or perforation of the aorta; graft thrombosis or infection; break of the attachment system; graft migration; proximal or distal graft leaks; delayed rupture; and bowel ischemia.

## **Nursing Management**

Before endovascular repair or surgery, nursing assessment is guided by anticipating rupture and recognizing that the patient may have cardiovascular, cerebral, pulmonary, and renal impairment from atherosclerosis. The functional capacity of all organ systems should be assessed. Medical therapies designed to stabilize physiologic function should be promptly implemented. Hemorrhage that leads to shock is a serious adverse consequence that must be treated decisively (see [Chapter 11](#)).



**Figure 26-12 •** Endograft repair of an abdominal aortic aneurysm.

The patient who has had an endovascular repair must lie supine for 6 hours; the head of the bed may be elevated up to 45 degrees after 2 hours. The patient needs to use a bedpan or urinal while on bed rest. Vital signs and Doppler assessment of peripheral pulses are performed initially every 15 minutes and then at progressively longer intervals if the patient's status remains stable. The access site (usually the femoral artery) is assessed when vital signs and pulses are monitored. The nurse assesses for bleeding and hematoma formation. Skin changes of the lower extremity, lumbar area, or buttocks that might indicate signs of embolization, such as extremely tender, irregularly shaped, cyanotic areas, as well as any changes in vital signs, pulse quality, bleeding, pulsation, swelling, pain, or hematoma, are immediately reported to the primary provider.

The patient's temperature should be monitored every 4 hours, and any signs of postimplantation syndrome should be reported. Postimplantation syndrome typically begins within 24 hours of stent graft placement and consists of a spontaneously occurring fever, leukocytosis, and occasionally, transient

thrombocytopenia. This condition has been attributed to complex immunologic changes that occur because of manipulations with sheaths and catheters with the aortic lumen, although the exact etiology is unknown. The symptoms are thought to be related to the activation of cytokines (Martinelli, Di Girolamo, Belli, et al., 2019). They can be managed with a mild analgesic (e.g., acetaminophen) or an anti-inflammatory agent (e.g., ibuprofen) and usually subside within a week.

Because of the increased risk of hemorrhage, the primary provider is also notified of persistent coughing, sneezing, vomiting, or systolic blood pressure greater than 180 mm Hg. Most patients can resume their preprocedure diet and are encouraged to drink fluids. An IV infusion may be continued until the patient can drink normally. Fluids are important to maintain blood flow through the arterial repair site and to assist the kidneys with excreting IV contrast agents and other medications used during the procedure. Six hours after the procedure, the patient may be able to roll from side to side and may be able to ambulate with assistance to the bathroom. Once the patient can take adequate fluids orally, the IV infusion may be discontinued.

Postoperative care requires frequent monitoring of pulmonary, cardiovascular, renal, and neurologic status. Possible complications of surgery include arterial occlusion, hemorrhage, infection, ischemic bowel, kidney injury, and erectile dysfunction.

## Other Aneurysms

Aneurysms may also arise in the peripheral vessels, most often as a result of atherosclerosis. These may involve such vessels as the subclavian artery, renal artery, femoral artery, or (most frequently) popliteal artery. Between 50% and 60% of popliteal aneurysms are bilateral; the true incidence is unknown but has been reported to be between 0.1% and 3% of the adult population and is associated with abdominal aortic aneurysms (Sidawy & Perler, 2019).

The aneurysm produces a pulsating mass and disturbs peripheral circulation distal to it. Pain and swelling develop because of pressure on adjacent nerves and veins. Diagnosis is made by duplex ultrasonography or CTA to determine the size, length, and extent of the aneurysm. Arteriography may be performed to evaluate the level of proximal and distal involvement. The major complication associated with popliteal artery aneurysms is not rupture but distal embolization (Sidawy & Perler, 2019). Surgical repair is performed with replacement grafts. As an alternative, endovascular repair using a stent graft or wall graft, which is a Dacron or PTFE graft with external structures made from a variety of materials (e.g., nitinol, titanium, stainless steel) may be selected for additional support.

## Aortic Dissection

Occasionally, in an aorta diseased by arteriosclerosis, a tear develops in the intima or the media degenerates, resulting in a dissection (see Fig. 26-11). Aortic dissections are three times more common in men than in women, occur most commonly in the 50- to 70-year age group and are associated with hypertension (Sidawy & Perler, 2019).

## Pathophysiology

Aortic dissections (separations) are commonly associated with poorly controlled hypertension, blunt chest trauma, and cocaine use. The profound increase in sympathetic response caused by cocaine use creates an increase in the force of left ventricular contraction that causes heightened shear forces upon the aortic wall leading to disruption of the intima (Sidawy & Perler, 2019). Dissection is caused by rupture in the intimal layer. A rupture may occur through adventitia or into the lumen through the intima, allowing blood to reenter the main channel and resulting in chronic dissection (e.g., pseudoaneurysm) or occlusion of branches of the aorta.

As the separation progresses, the arteries branching from the involved area of the aorta shear and occlude. The tear occurs most commonly in the region of the aortic arch, with the highest mortality rate associated with ascending aortic dissection (Sidawy & Perler, 2019). The dissection of the aorta may progress backward in the direction of the heart, obstructing the openings to the coronary arteries or producing hemopericardium (effusion of blood into the pericardial sac) or aortic insufficiency, or it may extend in the opposite direction, causing occlusion of the arteries supplying the gastrointestinal tract, kidneys, spinal cord, and legs.

## Clinical Manifestations

Onset of symptoms is usually sudden. Severe and persistent pain, described as tearing or ripping, may be reported. The pain is in the anterior chest or back and extends to the shoulders, epigastric area, or abdomen. Aortic dissection may be mistaken for an acute myocardial infarction, which confuses the clinical picture and initial treatment. Cardiovascular, neurologic, and gastrointestinal symptoms are responsible for other clinical manifestations, depending on the location and extent of the dissection. The patient may appear pale. Sweating and tachycardia may be detected. Blood pressure may be elevated or markedly different from one arm to the other if dissection involves the orifice of the subclavian artery on one side.

## Assessment and Diagnostic Findings

Arteriography, multidetector-computed tomography angiography (MDCTA), TEE, duplex ultrasonography, and MRA, while limited in terms of expediency during an emergency situation, may aid in the diagnosis.

## Medical Management

The medical or surgical treatment of an aortic dissection depends on the type of dissection present and follows the general principles outlined for the treatment of thoracic aortic aneurysms.

## Nursing Management

A patient with an aortic dissection requires the same nursing care as a patient with an aortic aneurysm requiring intervention, as described earlier in this chapter. Nursing care as described in [Chart 26-4](#) is also appropriate.

## Arterial Embolism and Arterial Thrombosis

Acute vascular occlusion may be caused by an embolus or acute thrombosis. Acute arterial occlusions may result from iatrogenic injury, which can occur during insertion of invasive catheters such as those used for arteriography, PTA or stent placement, or an intra-aortic balloon pump, or it may occur as a result of illicit IV drug use. Other causes include trauma from a fracture or dislocation, crush injury, compartment syndrome, and penetrating wounds that disrupt the arterial intima. The accurate diagnosis of an arterial occlusion as embolic or thrombotic in origin is necessary to initiate appropriate treatment.

## Pathophysiology

Arterial emboli arise most commonly from thrombi that develop in the chambers of the heart as a result of atrial fibrillation, myocardial infarction, infective endocarditis, or chronic heart failure. These thrombi become detached and are carried from the left side of the heart into the arterial system, where they lodge in and obstruct an artery that is smaller than the embolus. Emboli may also develop in advanced aortic atherosclerosis because the atheromatous plaques ulcerate or become rough. Acute thrombosis frequently occurs in patients with preexisting ischemic symptoms.

## Clinical Manifestations

Symptoms of arterial emboli depend primarily on the size of the embolus, organ involvement, and the state of collateral vessels. The immediate effect is

cessation of distal blood flow. The blockage can progress distal and proximal to the site of the obstruction. Secondary vasospasm can contribute to the ischemia. The embolus can fragment or break apart, resulting in occlusion of distal vessels. Emboli tend to lodge at arterial bifurcations and areas narrowed by atherosclerosis. Cerebral, mesenteric, renal, and coronary arteries are often involved in addition to the large arteries of the extremities.

The symptoms of acute arterial embolism in extremities with poor collateral flow are acute, severe pain, and a gradual loss of sensory and motor function. The six Ps associated with acute arterial embolism are *pain*, *pallor*, *pulselessness*, *paresthesia*, *poikilothermia* (coldness), and *paralysis*. Eventually, superficial veins may collapse because of decreased blood flow to the extremity. Because of ischemia, the part of the extremity distal to the occlusion is markedly colder and paler than the part proximal to the occlusion.

Arterial thrombosis can also acutely occlude an artery. A thrombosis is a slowly developing clot that usually occurs where the arterial wall has become damaged, generally as a result of atherosclerosis. Thrombi may also develop in an arterial aneurysm. The manifestations of an acute thrombotic arterial occlusion are similar to those described for an embolic occlusion. However, treatment is more difficult with a thrombus because the arterial occlusion has occurred in a degenerated vessel and requires more extensive reconstructive surgery to restore flow than is required with an embolic event (Sidawy & Perler, 2019).

## Assessment and Diagnostic Findings

An arterial embolus is usually diagnosed on the basis of the sudden onset of symptoms and an apparent source for the embolus. Two-dimensional transthoracic echocardiography or transthoracic echocardiogram (TTE), chest x-ray, and electrocardiography (ECG) may reveal underlying cardiac disease. Noninvasive duplex and Doppler ultrasonography can determine the presence and extent of underlying atherosclerosis, and arteriography may be performed.

## Medical Management

Management of arterial thrombosis depends on its cause. Management of acute embolic occlusion usually requires surgery because there is only a 4- to 6-hour window to restore blood flow before irreversible death of tissue. The event is acute with no collateral circulation developed, and the patient quickly moves through the list of six Ps to paralysis, the most advanced stage. Heparin therapy is initiated immediately to prevent further development of emboli and to prevent the extension of existing thrombi. Typically, an initial IV bolus of 60 to 80 U/kg body weight is given, followed by a continuous infusion of 12 to

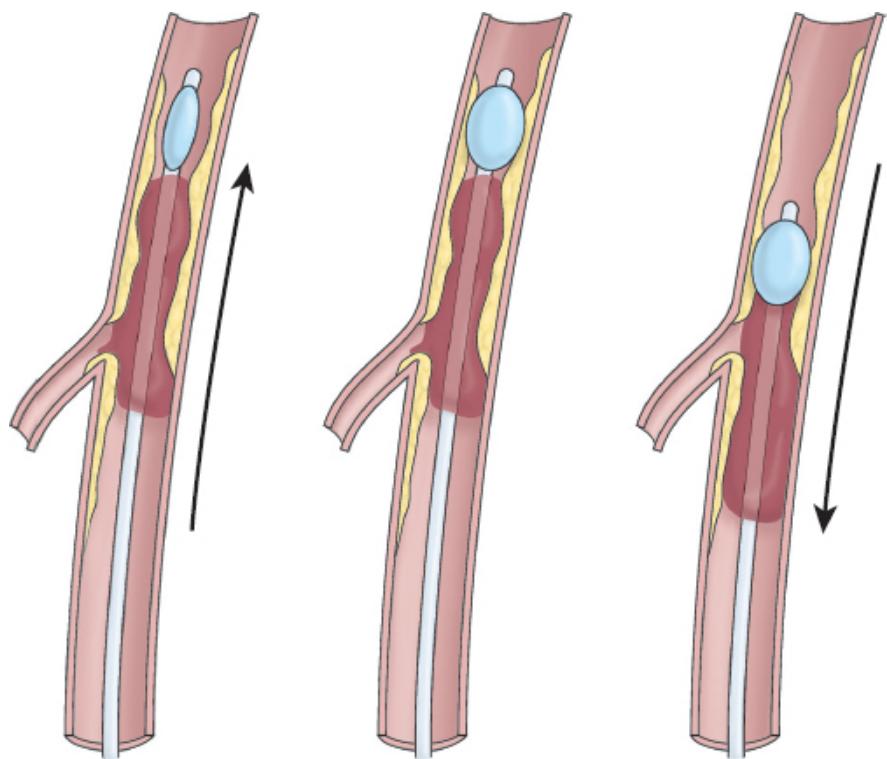
18 U/kg/h until the patient undergoes an endovascular intervention or surgery (Comerford & Durkin, 2020; IBM Watson Health, 2020).

## Endovascular Management

Emergency embolectomy is the procedure of choice if the involved extremity is viable (see Fig. 26-13). Arterial emboli are usually treated by insertion of an embolectomy catheter. The catheter is passed through an incision into the affected artery and extended through the embolus that is causing the arterial occlusion. The embolectomy catheter balloon is inflated with sterile saline solution, and the thrombus is extracted as the catheter is withdrawn. This procedure involves incising the vessel and removing the clot.

Percutaneous mechanical thrombectomy devices may also be used for the treatment of an acute thrombosis. All endovascular devices necessitate obtaining access to the patient's arterial system and inserting a catheter into the patient's artery to obtain access to the thrombus. The approach is similar to that used for angiograms, in that it is typically made through the groin to the femoral artery. In select cases, the radial or brachial artery can be accessed, allowing patients to be ambulatory post procedure. To qualify for this approach, the height of the patient is considered to ensure that the length of the catheter can reach the thrombus. Patients who are 5 feet 6 inches or shorter are potential candidates.

Some endovascular devices require that a small incision (cut-down) be made into the patient's artery. These devices may use a jet of fluid to disrupt the thrombus and then aspirate the particles; a rotating, sinusoidal-shaped wire that mixes a thrombolytic agent that simultaneously dissolves the clot; or high-frequency, low-energy ultrasound to dissolve an occlusive thrombus. Complications arising from the use of any endovascular device may include arterial dissection or distal artery embolization.



**Figure 26-13 •** Extraction of an embolus by a balloon-tipped embolectomy catheter. The deflated balloon-tipped catheter is advanced past the embolus, inflated, and then gently withdrawn, carrying the embolic material with it. Adapted with permission from Sidawy, A. N., & Perler, B. A. (2019). *Rutherford's vascular surgery and endovascular therapy* (9th ed.). Philadelphia, PA: Elsevier.

### Pharmacologic Therapy

When the patient has adequate collateral circulation, treatment may include IV anticoagulation with heparin, which can prevent the thrombus from extending and reduce muscle necrosis. Intra-arterial thrombolytic medications are used to dissolve the embolus. Thrombolytic medications (e.g., tissue plasminogen activator [t-PA] and single-chain urokinase-type plasminogen activator) interact with plasminogen to generate plasmin which then breaks down fibrin clots. If t-PA is used for the treatment, heparin is usually given to prevent another thrombus from forming at the site of the lesion. The t-PA activates plasminogen on the thrombus, but it does not decrease the clotting factors as much as other thrombolytic therapies; therefore, patients receiving t-PA can make new thrombi more readily than if they receive other thrombolytics. Other thrombolytic medications are reteplase and tenecteplase (Millar & Laffan, 2017). Although these agents differ in their pharmacokinetics, they are given in a similar manner: A catheter is advanced under x-ray visualization to the clot, and the thrombolytic agent is infused.

Thrombolytic therapy should not be used when there are known contraindications to therapy or when the extremity cannot tolerate the several additional hours of ischemia that it takes for the agent to lyse (disintegrate) the clot. Contraindications to peripheral thrombolytic therapy include active internal bleeding, cerebrovascular hemorrhage, recent major surgery, uncontrolled hypertension, and pregnancy.

## Nursing Management

Before an intervention or surgery, the patient remains on bed rest with the affected extremity level or slightly dependent (15 degrees). The affected extremity is kept at room temperature and protected from trauma. Heating and cooling pads are contraindicated because ischemic extremities are easily traumatized by alterations in temperature. If possible, tape and ECG electrodes should not be used on the extremity to protect from trauma. Pressure injury prevention through offloading the heel with a heel device and lifting the bedsheets using a bed cradle are important to protect the affected leg (European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance [EPUAP/NPIAP/PPPIA], 2019).

If the patient is treated with thrombolytic therapy, the dose is based on the patient's weight. The patient is admitted to a critical care unit for continuous monitoring. Vital signs are taken initially every 15 minutes and then at progressively longer intervals if the patient's status remains stable. The patient is closely monitored for bleeding. The nurse minimizes the number of punctures for inserting IV lines and obtaining blood samples, avoids intramuscular injections, prevents any possible tissue trauma, and applies pressure at least twice as long as usual after any puncture is performed.

During the recovery phase, the nurse collaborates with the primary provider about the patient's appropriate activity level based on the patient's condition. Generally, every effort is made to encourage the patient to move the extremity to stimulate circulation and prevent stasis. Anticoagulant therapy may be continued postendovascular intervention to prevent thrombosis of the affected artery and to diminish the development of subsequent thrombi at the initiating site. The nurse assesses for evidence of local and systemic hemorrhage, including mental status changes, which can occur when anticoagulants are given. Pulses, Doppler signals, ABI, and motor and sensory function are assessed every hour for the first 24 hours, because significant changes may indicate reocclusion. Metabolic abnormalities, acute kidney injury, and compartment syndrome may be complications after an acute arterial occlusion.

## Raynaud's Phenomenon and Other Acrosyndromes

Raynaud's phenomenon is a form of intermittent arteriolar vasoconstriction that results in coldness, pain, and pallor of the fingertips or toes. There are two forms of this disorder. Primary or idiopathic Raynaud's (Raynaud's disease) occurs in the absence of an underlying disease. Secondary Raynaud (Raynaud syndrome) occurs in association with an underlying disease, usually a connective tissue disorder, such as systemic lupus erythematosus, rheumatoid arthritis, or scleroderma; trauma; or obstructive arterial lesions. Symptoms may result from a defect in basal heat production that eventually decreases the ability of cutaneous vessels to dilate. Episodes may be triggered by emotional factors, stress, or by unusual sensitivity to cold. Raynaud's phenomenon is five times more common in women with the typical onset before age 30 (Dean, 2018). Acrocyanosis has been thought to be a variant of Raynaud's phenomenon because both are aggravated by cold and emotional stress and both present with blue discoloration of the fingers and hyperhidrosis (excessive sweating).

The prognosis for patients with Raynaud's phenomenon varies; some slowly improve, some become progressively worse, and others show no change. Raynaud symptoms may be mild so that treatment is not required. However, secondary Raynaud is characterized by vasospasm and fixed blood vessel obstructions that may lead to ischemia, ulceration, and gangrene. Acrocyanosis is a poorly understood phenomenon that may be benign and require little or no treatment, or the patient may have chronic pain and ulcerations.

## Clinical Manifestations

The classic clinical picture of Raynaud reveals pallor brought on by sudden vasoconstriction. The skin then becomes cyanotic because of pooling of deoxygenated blood during vasospasm. As a result of hyperemia (exaggerated reflow) due to vasodilation, rubor is produced when oxygenated blood returns to the digits after the vasospasm stops. The characteristic sequence of color change of Raynaud's phenomenon is described as white, blue, and red. Numbness, tingling, and burning pain occur as the color changes. The manifestations tend to be bilateral and symmetric and may involve toes and fingers.

Acrocyanosis is differentiated from Raynaud by a relative persistence of skin color changes, symmetry, and an absence of the paroxysmal pallor that is found with Raynaud. Almost all patients with acrocyanosis have marked clamminess and hyperhidrosis of their hands and feet, which tend to worsen in warmer temperatures while the color changes improve. Finger color normalizes when the hands are transferred from the dependent to horizontal position (Dean, 2018).

## **Medical Management**

Avoiding the particular stimulus (e.g., cold, nicotine) that provokes vasoconstriction is a primary factor in controlling Raynaud's phenomenon. Decongestants and other over-the-counter preparations containing sympathomimetics should be avoided. Calcium channel blockers (nifedipine, amlodipine) may be effective in relieving symptoms. Sympathectomy (interrupting the sympathetic nerves by removing the sympathetic ganglia, or blocking or dividing their branches) may help some patients.

Avoidance of exposure to cold and trauma and implementing measures to improve local circulation are the primary focus of treatment for acrocyanosis. Calcium channel blockers have not been effective in treating acrocyanosis (Belch, Carlizza, Carpentier, et al., 2017).

## **Nursing Management**

The nurse instructs the patient with Raynaud or acrocyanosis to avoid situations that may be stressful or unsafe. Stress management strategies may be helpful. Exposure to cold must be minimized, and in areas where the fall and winter months are cold, the patient should wear layers of clothing when outdoors. Hats and gloves or mittens should be worn at all times when outside. Fabrics specially designed for cold climates (e.g., Thinsulate™) are recommended. Patients should wear gloves when accessing freezers. Patients should warm up their vehicles before getting in so that they can avoid touching a cold steering wheel or door handle, which could elicit an attack. During summer, a sweater should be available when entering air-conditioned rooms.

Patients are often concerned about serious complications, such as gangrene and amputation; however, these complications are uncommon unless the patient has another underlying disease causing arterial occlusions. Patients should avoid all forms of nicotine, which may induce attacks; this includes nicotine gum or patches used to aid smoking cessation.

Patients should be cautioned to handle sharp objects carefully to avoid injuring their fingers. In addition, patients should be informed about the orthostatic hypotension that may result from medications, such as calcium channel blockers, used to treat Raynaud's phenomenon.

## **VENOUS DISORDERS**

Venous disorders cause a reduction in venous blood flow, which results in stasis of blood. This may then cause a host of pathologic changes, including coagulation defects, edema, tissue breakdown, and an increased susceptibility to infections.

## Venous Thromboembolism

Deep vein thrombosis (DVT) and pulmonary embolism (PE) collectively make up the condition called **venous thromboembolism (VTE)**. The annual incidence of VTE is estimated at 1 to 2 per 1000 population (Peñaloza-Martínez, Demelo-Rodríguez, Proietti, et al., 2018; Serhal & Barnes, 2019). The incidence of VTE is 10% to 20% in general medical patients and up to 80% in critically ill patients. VTE is frequently not diagnosed because DVT and PE are often clinically silent or asymptomatic. It is estimated that as many as 30% of patients hospitalized with VTE develop long-term postthrombotic complications. In surgical patients, most symptomatic thromboembolic complications occur after hospital discharge due to shorter lengths of stay (Stubbs, Assareh, Curnow, et al., 2018).



## COVID-19 Considerations

An underlying cause of death in patients with severe coronavirus disease 2019 (COVID-19) has been linked to VTE or primary pulmonary thrombi. The majority of patients who are severely ill with COVID-19 have a markedly elevated D-dimer assay (blood test for evidence of blood clots) and a presumed prothrombotic state. Because of this, routine thromboprophylaxis of all hospitalized patients with COVID-19 is recommended regardless of their risk score (see later discussion) (Obi, Barnes, Wakefield, et al., 2020). The systemic inflammatory changes in the vascular system in patients with COVID-19 likely contribute to thrombosis, hemodynamic instability, and autonomic dysregulation. Early reports suggest long-term cardiovascular effects of COVID-19 may include accelerated atherosclerosis, VTE, arterial thromboembolic disease, and aortic aneurysm formation (Becker, 2020).

## Pathophysiology

Superficial veins, such as the greater saphenous, short saphenous (also known as lesser saphenous), cephalic, basilic, and external jugular veins, are thick-walled muscular structures that lie just under the skin. In contrast, deep veins are thin walled and have less muscle in the media. Deep veins run parallel to arteries and bear the same names as the arteries. Deep and superficial veins have valves that permit unidirectional flow back to the heart. The valves lie at the base of a segment of vein that is expanded into a sinus. This arrangement enables the valves to open without coming into contact with the wall of the vein, thus permitting rapid closure when the blood starts to flow backward. Other types of veins are known as perforating veins. These veins have valves that allow one-way blood flow from the superficial venous system to the deep venous system.

Although the exact cause of VTE remains unclear, three factors, known as Virchow triad, are believed to play a significant role in its development: endothelial damage, venous stasis, and altered coagulation (see [Chart 26-8](#)). Damage to the intimal lining of blood vessels creates a site for clot formation. Direct trauma to the veins may occur with fractures or dislocation, diseases of the veins, and chemical irritation of the vein from IV medications or solutions. Venous stasis occurs when blood flow is reduced, as in heart failure or shock; when veins are dilated, as an effect of some medication therapies; and when skeletal muscle contraction is reduced, as in immobility, paralysis of the extremities, or anesthesia. Altered coagulation occurs most commonly in patients for whom anticoagulant medications have been abruptly withdrawn. Oral contraceptive use, elevated CRP levels (Cauci, Francescato, Colannino, et al., 2020), and several blood dyscrasias (abnormalities) also can lead to hypercoagulability, with prevalence depending on the ethnicity of the patient. For example, factor V Leiden and prothrombin G20210A mutation is more prevalent in White persons, whereas antithrombin III deficiency, protein C deficiency, and protein S deficiency are found more commonly in patients of Southeast Asian descent (Sidawy & Perler, 2019). An increase in factor VIII concentrations is more common among African Americans (Folsom, Basu, Hong, et al., 2019). Pregnancy is also considered a hypercoagulable state, as it is accompanied by an increase in circulating clotting factors that may not return to baseline until longer than 6 weeks postpartum, increasing the risk of thrombosis. In addition, during pregnancy there is a 50% decrease in venous outflow due to hormonally decreased venous capacitance and reduced venous outflow due to compression from the uterus (Zheng, Chen, Fu, et al., 2019).

**Chart 26-8**  **RISK FACTORS**

## Deep Vein Thrombosis and Pulmonary Embolism

### Endothelial Damage

- Central venous catheters
- Dialysis access catheters
- Local vein damage
- Pacing wires
- Repetitive motion injury
- Surgery
- Trauma

### Venous Stasis

- Age (>65 years)
- Bed rest or immobilization
- Heart failure
- History of varicosities
- Obesity
- Spinal cord injury

### Altered Coagulation

- Antiphospholipid antibody syndrome
- Antithrombin III deficiency
- Cancer
- Elevated factors II, VIII, IX, XI
- Factor V Leiden defect
- Hyperhomocysteinemia
- Oral contraceptive use
- Polycythemia
- Pregnancy
- Protein C deficiency
- Protein S deficiency
- Prothrombin G20210A defect
- Septicemia

Adapted from Patel, K., Fasanya, A., Yadam, S., et al. (2017). Pathogenesis and epidemiology of venous thromboembolic disease. *Critical Care Nursing Quarterly*, 40(3), 191–200.

### Deep Vein Thrombosis

**Deep vein thrombosis (DVT)** refers to thrombus formation in the deep veins, usually in the thigh or calf, but sometimes in the arm (e.g., patients with peripherally inserted central catheters).

## Pathophysiology

Formation of a thrombus frequently accompanies phlebitis, which is an inflammation of the vein walls. When a thrombus develops initially in the veins as a result of stasis or hypercoagulability but without inflammation, the process is referred to as phlebothrombosis. Venous thrombosis can occur in any vein, but it occurs more often in the veins of the lower extremities. The superficial and deep veins of the extremities may be affected.

Upper extremity venous thrombosis accounts for 5% to 10% of all cases of DVT, but its incidence may be as high as 93% in the presence of central venous cannulation or upper extremity compression (Mintz & Levy, 2017). It typically involves more than one venous segment, with the subclavian vein the most frequently affected. In addition, upper extremity venous thrombosis is more common in patients with IV catheters or in patients with an underlying disease that causes hypercoagulability, such as cancer. Internal trauma to the vessels may result from pacemaker leads, chemotherapy ports, dialysis catheters, or parenteral nutrition lines. The lumen of the vein may be decreased as a result of the catheter or from external compression, such as by neoplasms or an extra cervical rib. Effort thrombosis, also known as Paget–Schroetter syndrome, of the upper extremity is caused by repetitive motion (e.g., as seen in competitive swimmers, tennis players, baseball players, weight lifters, or construction workers) that irritates the vessel wall, causing inflammation and subsequent thrombosis and is a manifestation of venous thoracic outlet syndrome where the veins becomes distorted and narrowed (Sidawy & Perler, 2019).

Venous thrombi are aggregates of platelets attached to the vein wall that have a tail-like appendage containing fibrin, white blood cells, and many red blood cells. The “tail” can grow or can propagate in the direction of blood flow as successive layers of the thrombus form. A propagating venous thrombosis is dangerous because parts of the thrombus can break off and occlude the pulmonary blood vessels. Fragmentation of the thrombus can occur spontaneously as it dissolves naturally, or it can occur with an elevated venous pressure, as may occur after standing suddenly or engaging in muscular activity after prolonged inactivity. After an episode of acute DVT, reestablishment of the lumen of the vessel, or recanalization, typically occurs. Lack of recanalization within the first 6 months after DVT appears to be an important predictor of postthrombotic syndrome, which is one complication of venous thrombosis (Sidawy & Perler, 2019) (see later discussion). Other complications of venous thrombosis are listed in [Chart 26-9](#).

## Clinical Manifestations

A major challenge in recognizing DVT is that the signs and symptoms are nonspecific. The exception is phlegmasia cerulea dolens (massive iliofemoral venous thrombosis), in which the entire extremity becomes massively swollen, tense, painful, and cool to the touch. The large DVT creates severe and sudden venous hypertension that leads to tissue ischemia with resultant translocation of fluid into the interstitial space. Venous gangrene occurs in 20% to 50% of cases and is associated with poor survival (Sidawy & Perler, 2019).

### Chart 26-9

#### Complications of Venous Thrombosis

Chronic venous occlusion

Pulmonary emboli from dislodged thrombi

Valvular destruction:

- Chronic venous insufficiency
- Increased venous pressure
- Varicosities
- Venous ulcers

Venous obstruction:

- Edema
- Fluid stasis
- Increased distal pressure
- Venous gangrene

Adapted from Kahn, S. R., Galanaud, J. P., Vedantham, S., et al. (2016). Guidance for the prevention and treatment of the post-thrombotic syndrome. *Journal of Thrombosis and Thrombolysis*, 41(1), 144–153.

## Deep Veins

Clinical manifestations of obstruction of the deep veins include edema and swelling of the extremity because the outflow of venous blood is inhibited. The affected extremity may feel warmer than the unaffected extremity, and the superficial veins may appear more prominent. Tenderness, which usually occurs later, is produced by inflammation of the vein wall and can be detected by gently palpating the affected extremity. In some cases, signs and symptoms of a PE are the first indication of DVT.

## Superficial Veins

Thrombosis of superficial veins, or superficial thrombophlebitis, produces pain or tenderness, redness, and warmth in the involved area. The risk of the superficial venous thrombi becoming dislodged or fragmenting into emboli is

very low because most of them dissolve spontaneously. This condition can be treated at home with bed rest, elevation of the leg, analgesic agents, and possibly, anti-inflammatory medication.

## **Assessment and Diagnostic Findings**

Careful assessment is invaluable in detecting early signs of venous disorders of the lower extremities (see [Chart 26-8](#)). Diagnostic tests for VTE include laboratory and venous duplex studies. Laboratory tests are conducted to determine baseline levels and include complete blood count (CBC) and coagulation studies that include prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR). If an underlying hypercoagulable state is suspected, additional laboratory testing may include CRP, factor V Leiden, prothrombin G20210A mutation, antithrombin III, protein C, and protein S (Fischbach & Fischbach, 2018). Duplex ultrasound findings may show veins that appear larger than normal with thrombus formation or veins that are incompressible and dilated. The thrombus may appear to be adherent to the vein wall or mobile with a thrombus tail.

## **Prevention**

Patients with a prior history of VTE are at increased risk of a new episode; the rate of recurrence can be as high as 13.7% in men and 14.1% in women (Serhal & Barnes, 2019). VTE can be prevented, especially if patients who are considered at high risk are identified and preventive measures are instituted without delay. Preventive measures include encouragement of early ambulation and leg exercises, the application of graduated compression stockings, and the use of intermittent pneumatic compression devices (see later discussion). An additional method to prevent venous thrombosis in surgical patients is administration of subcutaneous unfractionated heparin or low-molecular-weight heparin (LMWH). Patients should be advised to make lifestyle changes as appropriate, which may include weight loss, smoking cessation, and regular exercise.

## **Medical Management**

The objectives of treatment for DVT are to prevent the thrombus from extending and fragmenting (thus risking PE), recurrent thromboemboli, and postthrombotic syndrome (discussed later in the chapter) (Peñaloza-Martínez et al., 2018). Anticoagulant therapy involves the administration of a medication to delay the clotting time of blood, prevent the formation

of a thrombus in postoperative patients, and forestall the extension of a thrombus after it has formed; it can be used to meet the treatment goals for DVT (Witt, Nieuwlaat, Clark, et al., 2018). However, anticoagulant agents cannot dissolve a thrombus that has already formed. Combining anticoagulation with mechanical thrombectomy and ultrasound-guided thrombolytic therapy may eliminate venous obstruction, maintain venous patency, and prevent postthrombotic syndrome by early removal of the thrombus (Kruger, Eikelboom, Douketis, et al., 2019).

## Pharmacologic Therapy

Medications for preventing or reducing blood clotting within the vascular system are indicated in patients diagnosed with DVT. These medications are also indicated for patients with thrombophlebitis, recurrent embolus formation, persistent leg edema from heart failure, and in select patients who must be immobilized for a protracted time (e.g., older adults with hip fractures). The primary pharmacologic treatment for the prevention and management of DVT is anticoagulant therapy which consists of vitamin K antagonists (e.g., warfarin), indirect and direct thrombin inhibitors, and factor Xa inhibitors (see [Table 26-2](#)).

## Endovascular Management

Endovascular management is necessary for DVT when anticoagulant or thrombolytic therapy is contraindicated, the danger of PE is extreme, or venous drainage is so severely compromised that permanent damage to the extremity is likely. A thrombectomy may be necessary. This mechanical method of clot removal may involve using intraluminal catheters with a balloon or other devices. Some of these spin to break the clot, and others use oscillation to break up the clot to facilitate removal. Ultrasound-assisted thrombolysis may be another option. This intervention uses bursts or continuous high-frequency ultrasound waves emanating from the catheters to cause cavitation of the thrombus, making it more permeable to the thrombolytic agent (Kruger et al., 2019). A vena cava filter may be placed at the time of the thrombectomy or thrombolysis; this filter traps large emboli and prevents PE (see later discussion). Retrievable caval filters can be left in place and retrieved up to 6 months after placement. In patients with chronic iliac vein compression (e.g., as is seen in May–Thurner syndrome), balloon angioplasty with stent placement may successfully treat the patient's chronic leg symptoms (Ignatyev, Pokrovsky, & Gradusov, 2019).

## Nursing Management

When performing the nursing assessment, key concerns include limb pain, a feeling of heaviness, functional impairment, ankle engorgement, and edema; increase in the surface temperature of the leg, particularly the calf or ankle; and areas of tenderness or superficial thrombosis (i.e., cordlike venous segment). The amount of swelling in the extremity can be determined by measuring the circumference of the affected extremity at various levels (i.e., thigh to ankle) with a tape measure and comparing one extremity with the other at the same level to determine size differences. If both extremities are swollen, a size difference may be difficult to detect. Homan sign (pain in the calf after the foot is sharply dorsiflexed) is *not* a reliable sign of DVT because it can be elicited in any painful condition of the calf and has no clinical value in assessment for DVT.

### Assessing and Monitoring Anticoagulant Therapy

If the patient is receiving anticoagulant therapy, the nurse monitors laboratory values as indicated. The aPTT, PT, INR, ACT, hemoglobin and hematocrit values, platelet count, and fibrinogen levels can be affected, depending on the anticoagulant prescribed; baseline assessments before the initiation of therapy should be performed. Close observation is required to detect bleeding; if bleeding occurs, it must be reported immediately and anticoagulant therapy reversed or discontinued if appropriate (see [Table 26-2](#)).

### Monitoring and Managing Potential Complications

Potential complications of anticoagulant therapy consist of bleeding, thrombocytopenia, and drug–drug interactions. The nurse should monitor for these potential complications, be familiar with medications approved to reverse effects of various anticoagulants, and educate patients and caregivers on ways to reduce these potential risks (see [Table 26-2](#) and [Chart 26-10](#)).

### Reducing Discomfort

Elevation of the affected extremity, graduated compression stockings (see later discussion), and analgesic agents for pain relief are adjuncts to therapy. They help improve circulation and increase comfort. Warm

packs applied to the affected extremity reduce the discomfort associated with DVT.

### **Positioning the Body and Encouraging Exercise**

When the patient is on bed rest, the feet and lower legs should be elevated periodically above the level of the heart. This position allows the superficial and tibial veins to empty rapidly and to remain collapsed. Active and passive leg exercises, particularly those involving calf muscles, should be performed to increase venous flow. Early ambulation is most effective in preventing venous stasis. The patient is encouraged to walk once anticoagulation therapy has been initiated and is advised that walking is better than standing or sitting for long periods. Once ambulatory, the patient is instructed to avoid sitting for more than an hour at a time. The goal is to walk at least 10 minutes every 1 to 2 hours. The patient is also instructed to perform active and passive leg exercises as frequently as necessary when they cannot ambulate, such as during long car, bus, train, and plane trips. In addition, deep breathing exercises are beneficial because they produce increased negative pressure in the thorax, which assists in emptying the large veins.

**TABLE 26-2** Select Anticoagulant and Thrombolytic Agents Prescribed to Treat Venous Thromboemboli (VTE)

Medication	Mechanism of Action	Actions and Effects	Nursing Considerations
<b>Unfractionated Heparin</b>			
Heparin	Binds to antithrombin; inactivates clotting factors XII, Xa; half-life of 60–90 min	Prevent extension of thrombus Prevent development of new thrombus	Can be administered subcutaneously or intermittently or continuous intravenously (IV); IV dosing is weight based Administer continuous IV infusions using an electronic infusion device Requires monitoring of aPTT and platelet count; monitor platelet counts for HIT Assess patient for bleeding Administer protamine sulfate for overdose or to reverse effects, as prescribed; monitor patient for hypotension and bradycardia if administered
<b>Low-Molecular-Weight Heparin (LMWH)</b>			
Dalteparin Enoxaparin	Inhibit factor Xa	Prevent extension of thrombus Prevent development of new thrombus	Can be administered subcutaneously once daily or twice a day Associated with fewer bleeding complications than unfractionated heparin Has lower risk of HIT Administer protamine sulfate for overdose or to reverse effects, as prescribed; monitor patient for hypotension and bradycardia if administered; protamine sulfate is less effective in reversing LMWH compared to unfractionated heparin Can be used in pregnancy if clearly indicated; however, patients should be monitored closely for bleeding
<b>Oral Anticoagulant</b>			
Warfarin	Vitamin K	Has narrow	Administer once a day at the

	antagonist inhibits synthesis of vitamin K– dependent clotting factors: II, VII, IX, X  Inhibits synthesis of vitamin K– dependent proteins: protein C and protein S	therapeutic window Has slow onset of action Anticoagulant effect occurs 12–24 h after first dose  Antithrombotic effect occurs 2–7 days after first dose	same time each day  Requires routine monitoring of PT with goal of 1.5–2 times normal; and INR with goal of 2.0–3.0  Requires administration with heparin during drug initiation until desired anticoagulation is achieved  Monitor for food and drug– drug interactions (see <a href="#">Chart 26-10</a> )  Assess patient for bleeding Administer vitamin K, fresh- frozen plasma, or prothrombin complex concentrate as prescribed for overdose or to reverse effects  Requires periprocedural bridging (i.e., giving a short- acting anticoagulant, usually heparin or LMWH, if warfarin needs to be temporarily discontinued) Contraindicated in pregnancy
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### Factor Xa Inhibitor

Fondaparinux	Selective inhibitor of factor Xa	Prevent DVT or PE in patients undergoing orthopedic surgery (e.g., hip or knee arthroplasty)  Treatment of DVT or PE  Excreted unchanged via kidneys  Does not affect aPTT or ACT	Administer subcutaneously once daily  Use with caution in patients with renal insufficiency Monitor creatinine Routine coagulation tests are not necessary
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### Oral Direct Factor Xa Inhibitor

Rivaroxaban	Direct inhibitor of factor Xa	Fixed-dose regimen for	Administer once or twice daily as prescribed based on indication
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	treatment of DVT and PE Fixed-dose regimen to prevent recurrent DVT and PE Not indicated for patients with creatinine clearance less than 30 mL/min	Assess renal function Alter dose in individuals with obesity May require periprocedural bridging Administer andexanet alfa or activated charcoal as prescribed for overdose or to reverse effects
Apixaban	Fixed-dose regimen for treatment of DVT and PE Fixed-dose regimen to prevent recurrent DVT and PE Not indicated for patients with creatinine clearance less than 25 mL/min.	Administer twice a day Assess renal function Alter dose in patients 80 yrs of age or older and weight $\leq$ 60 kg Administer andexanet alfa or activated charcoal as prescribed for overdose or to reverse effects
Edoxaban	Fixed-dose regimen for treatment of DVT and PE	Administer once daily Assess renal function Alter dose for patients who weigh $\leq$ 60 kg
Betrixaban	Prevent DVT and PE in adults hospitalized for acute medical illness	Administer once daily Assess renal function No antidote available to reverse effects

#### **Direct Thrombin Inhibitor**

Argatroban Lepirudin	Reversibly binds to thrombin active site, inhibiting thrombin-	Prevention and treatment of thrombosis in patients with HIT or at risk	Administer as an IV bolus followed by a continuous infusion Assess hepatic function Monitor aPTT and ACT
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	mediated stimulation of coagulation factors	for HIT during PCI	
<b>Oral Direct Thrombin Inhibitor</b>			
Dabigatran	Reversibly binds to thrombin active site, inhibiting thrombin-mediated stimulation of coagulation factors	Fixed-dose regimen for treatment of DVT Fixed-dose regimen to prevent recurrent DVT Not indicated for patients with creatinine clearance <30 mL/min	Administer twice a day Assess renal function Routine coagulation tests are not necessary; however if measured, aPTT levels may be prolonged 1.5–2 times normal Administer idarucizumab as prescribed to reverse effects
<b>Thrombolytic</b>			
Alteplase Reteplase Tenecteplase Urokinase	Binds to fibrin within the thrombus converting plasminogen to plasmin.	Lyse and dissolve existing thrombus	Monitor lab values prior to initiation: CBC, platelets, aPTT; repeat during and post therapy Monitor for bleeding and hemorrhage Contraindicated with active bleeding
Plasmin	degrades fibrin, fibrinogen, and factors V, VIII, and XII.		

ACT, activated clotting time; aPTT, activated partial thromboplastin time; CBC, complete blood count; DVT, deep vein thrombosis; HIT, heparin-induced thrombocytopenia; INR, international normalized ratio; PCI, percutaneous coronary intervention; PE, pulmonary emboli; PT, prothrombin time; PTT, partial thromboplastin time.

Adapted from Cohen, A. T., Lip, G. Y., De Caterina, R., et al. (2018). State of play and future direction with NOACs: An expert consensus. *Vascular Pharmacology*, 106, 9–21; Comerford, K. C., & Durkin, M. T. (Eds.). (2020). *Nursing2020 Drug Handbook*. Philadelphia, PA: Wolters Kluwer; Kruger, P. C., Eikelboom, J. W., Douketis, J. D., et al. (2019). Deep vein thrombosis:

Update on diagnosis and management. *Medical Journal of Australia*, 210(11), 516–524; Peñaloza-Martínez, E., Demelo-Rodríguez, P., Proietti, M., et al. (2018). Update on extended treatment for venous thromboembolism. *Annals of Medicine*, 50(8), 666–674; Serhal, M., & Barnes, G. D. (2019). Venous thromboembolism: A clinician update. *Vascular Medicine*, 24(2), 122–131.

**Chart 26-10**



**PATIENT EDUCATION**

## Taking Anticoagulant Medications

The nurse instructs the patient as follows:

- Take anticoagulation medication as prescribed.
- Do not stop taking your anticoagulation medication unless directed.
- If prescribed warfarin, take at the same time each day.
- Wear or carry identification indicating anticoagulant medication being taken.
- Keep all scheduled appointments for blood tests.
- Be aware that other medications may affect the action of the anticoagulant medication; if taking warfarin, consult with your health care provider before taking any of the following medications or supplements: vitamins, cold medicines, antihistamines, antibiotics, aspirin, laxatives, and anti-inflammatory agents, such as ibuprofen, and similar medications or herbal or nutritional supplements. Your primary provider should be contacted before taking any over-the-counter drugs.
- Avoid alcohol if taking warfarin because it may change the body's response to the warfarin. There are no interactions with the oral factor Xa inhibitors (e.g., rivaroxaban, apixaban, edoxaban) and alcohol.
- Avoid marked changes in eating habits, especially involving foods high in vitamin K, such as green leafy vegetables, which can reduce anticoagulation effectiveness if taking warfarin; dietary habits have no interactions with the oral factor Xa inhibitors.
- When seeking treatment from any health care provider, be sure to inform them that you are taking an anticoagulant medication.
- Contact your provider who manages your anticoagulation therapy before having dental work or surgery.
- Describe potential side effects of coagulation, such as bruising and bleeding, and identify ways to prevent bleeding.
  - Avoid the use of sharps (razors, knives, etc.) to prevent cuts; shave with an electric shaver.
  - Use a toothbrush with soft bristles to prevent gum injury.
  - Avoid contact sports or activities that may result in an injury that causes bleeding.
- If taking warfarin, report the following immediately to your primary provider:
  - Any bleeding—for example, cuts that do not stop bleeding

- Bruises that enlarge, nosebleeds, or unusual bleeding from
  - any part of the body
  - Reddish or brownish urine
  - Red or black bowel movements
- For women:* Notify your primary provider and obstetrical provider if you suspect pregnancy.

## Promoting Home, Community-Based, and Transitional Care

In addition to instructing the patient on how to apply graduated compression stockings as indicated (see later discussion) and explaining the importance of elevating the legs and exercising adequately, the nurse provides education about the prescribed anticoagulant, its purpose, and the need to take the correct amount at the specific times, especially if warfarin is prescribed (see [Chart 26-10](#)). The patient should also be aware that if warfarin is prescribed, periodic blood tests are necessary to determine if a change in medication or dosage is required. If the patient fails to adhere to the therapeutic regimen, continuation of the medication therapy should be questioned. In patients with liver disease, the potential for bleeding may be exacerbated by anticoagulant therapy.

## Pulmonary Embolism

**Pulmonary embolism (PE)** refers to the obstruction of the pulmonary artery or one of its branches by a thrombus (or thrombi) that originate(s) somewhere in the venous system or in the right side of the heart.

## Pathophysiology

Most commonly, PE is due to a dislodged or fragmented DVT (see previous pathophysiology discussion in DVT). However, there are other types of emboli that may be implicated: air, fat, amniotic fluid, and septic (from bacterial invasion of the thrombus) (Norris, 2019).

A PE is described as an occlusion of the outflow tract of the main pulmonary artery or of the bifurcation of the pulmonary arteries. Multiple small emboli can lodge in the terminal pulmonary arterioles, producing multiple small infarctions of the lungs. A pulmonary infarction causes ischemic necrosis of part of the lung (Thompson & Kabrhel, 2020).

When a thrombus completely or partially obstructs a pulmonary artery or its branches, the alveolar dead space is increased. The area, although continuing to be ventilated, receives little or no blood flow. Therefore, gas exchange is impaired or absent in this area. In addition, various substances are released from the clot and surrounding area that cause regional blood vessels and bronchioles to constrict. This results in an increase in pulmonary vascular resistance—a reaction that compounds the ventilation–perfusion (V./Q.) imbalance that ensues.

The hemodynamic consequences are increased pulmonary vascular resistance due to the regional vasoconstriction and reduced size of the pulmonary vascular bed. In severe instances, this may result in an increase in pulmonary arterial pressure and, in turn, an increase in right ventricular work to maintain pulmonary blood flow. When the work requirements of the right ventricle exceed its capacity, right ventricular failure occurs, leading to a decrease in cardiac output followed by a decrease in systemic blood pressure and the development of shock (Norris, 2019).

## Clinical Manifestations

Symptoms of PE depend on the size of the thrombus and the area of the pulmonary artery occluded by the thrombus; they may be nonspecific. Dyspnea is the most frequent symptom; the duration and intensity of the dyspnea depend on the extent of embolization. Chest pain is common and is usually sudden and pleuritic in origin; however, it may be substernal and may mimic angina (Thompson & Kabrhel, 2020). Other symptoms include anxiety, fever, tachycardia, apprehension, cough, diaphoresis, hemoptysis, and syncope. The most frequent sign is tachypnea (rapid respiratory rate) (De Palo, 2020).

In many instances, PE causes few signs and symptoms, whereas in other instances, it mimics various other cardiopulmonary disorders (e.g., pneumonia, heart failure). Obstruction of the pulmonary artery can result in pronounced dyspnea, sudden substernal pain, rapid and weak pulse, shock, syncope, and sudden death (Thompson & Kabrhel, 2020).

## Assessment and Diagnostic Findings

Because the symptoms of PE can vary from few to severe, a diagnostic workup is performed to rule out other diseases. The initial diagnostic workup may include chest x-ray, ECG, pulse oximetry, arterial blood gas

analysis, D-dimer assay and MDCTA or pulmonary arteriogram or V/Q scan. The chest x-ray is usually normal but may show infiltrates, atelectasis, elevation of the diaphragm on the affected side, or a pleural effusion. The chest x-ray is most helpful in excluding other possible causes. In addition to sinus tachycardia, the most frequent ECG abnormality is nonspecific ST-T wave abnormalities. If an arterial blood gas analysis is performed, it may show hypoxemia and hypocapnia (from tachypnea); however, arterial blood gas measurements may be normal even in the presence of PE (De Palo, 2020).

MDCTA is the criterion standard for diagnosing PE. The MDCTA can be performed quickly and provides the advantage of high-quality visualization of the lung parenchyma (Weinberger, Cockrill, & Mandel, 2019). If MDCTA is not available, pulmonary angiography is considered a reasonable alternative diagnostic method (Ouellette, 2019). The pulmonary angiogram allows for direct visualization under fluoroscopy of the arterial obstruction and accurate assessment of the perfusion deficit. A specially trained team must be available to perform the procedure, in which a catheter is threaded through the vena cava to the right side of the heart to inject dye, similar to a cardiac catheterization.

The V/Q scan continues to be used to diagnose PE, especially in facilities that do not use pulmonary angiography or do not have access to MDCTA. The V/Q scan is minimally invasive and requires IV administration of a contrast agent. This scan evaluates different regions of the lung (upper, middle, lower) and allows comparisons of the percentage of V/Q in each area. This test has a high sensitivity but is not as accurate as an MDCTA or pulmonary angiogram (De Palo, 2020).

## Medical Management

Medical management of the patient with PE revolves around whether the patient is diagnosed with a hemodynamically unstable PE (also called a *massive PE*) or a stable PE. The patient with a hemodynamically unstable PE, which comprises a life-threatening emergency, may evidence hypotension, tachycardia, confusion, and cardiovascular collapse.



### Medical Management of Unstable Pulmonary Embolism

The immediate objective is to stabilize the cardiopulmonary system in the patient with a hemodynamically unstable PE. A sudden increase in

pulmonary resistance increases the work of the right ventricle, which can cause acute right-sided heart failure with cardiogenic shock. Emergent measures are initiated to improve respiratory and cardiovascular status (see [Chapter 11](#) for discussion of management of the patient in shock).

After emergency measures have been initiated, the treatment goal is to lyse (dissolve) the existing embolus and prevent new ones from forming. Thrombolytic therapy with t-PA or other agents such as reteplase (see [Table 26-2](#)) is used in treating unstable PE, particularly in patients who are severely compromised (e.g., those who are hypotensive and have significant hypoxemia despite oxygen supplementation) (Ouellette, 2019). Thrombolytic therapy lyses the thrombi or emboli quickly and restores hemodynamic functioning of the pulmonary circulation, thereby reducing pulmonary hypertension and improving perfusion, oxygenation, and cardiac output. However, the risk of bleeding is significant. Contraindications to thrombolytic therapy include having had a stroke within the past 2 months, other active intracranial processes, active bleeding, surgery within 10 days of the thrombotic event, recent labor and delivery, trauma, or severe hypertension. Consequently, thrombolytic agents are advocated only for PE affecting a significant area of blood flow to the lung and causing hemodynamic instability (Tapson & Weinberg, 2020).

Before thrombolytic therapy is started, INR, aPTT, hematocrit, and platelet counts are obtained. Any anticoagulant is stopped prior to administration of a thrombolytic agent. During therapy, all but essential invasive procedures are avoided because of potential bleeding. After the thrombolytic infusion is completed (which varies in duration according to the agent used), maintenance anticoagulation therapy is initiated.

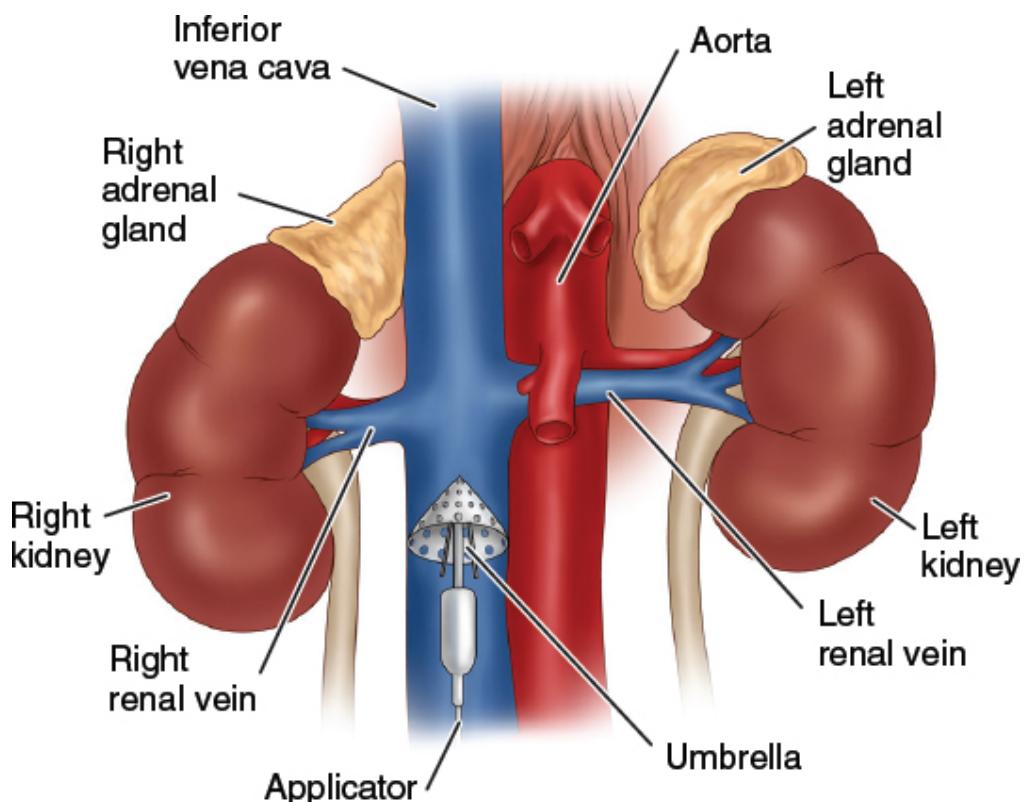
A surgical embolectomy is rarely performed but may be indicated if there are contraindications to thrombolytic therapy. Embolectomy can be performed using catheters or surgically. Surgical removal must be performed by a cardiovascular surgical team with the patient on cardiopulmonary bypass (Ouellette, 2019).

For patients who have recurrent PE despite therapeutic anticoagulation, an inferior vena cava (IVC) filter may be inserted (Tapson, 2019). IVC filters are not recommended for the initial treatment of patients with PE and should not be used in patients receiving anticoagulants. The IVC filter provides a screen in the IVC, allowing blood to flow unobstructed while large emboli from the pelvis or lower extremities are blocked or fragmented before reaching the lung.

Numerous devices have been developed since the introduction of the original Greenfield filter (see Fig. 26-14).

### Medical Management of Stable Pulmonary Embolism

In patients with PE who do not demonstrate any cardiopulmonary instability (e.g., normotensive, no evidence of hypoxemia) immediate anticoagulation is indicated to prevent recurrence or extension of the thrombus and may continue for 10 days (Tapson, 2019). Long-term anticoagulation is also indicated up to 6 months following the PE and is critical in preventing recurrence of VTE. This duration may be extended indefinitely in patients who are at high risk for recurrence (Weinberger et al., 2019).



**Figure 26-14 •** An umbrella filter is in place in the inferior vena cava to prevent PE. The filter (compressed within an applicator catheter) is inserted through an incision in the right internal jugular vein. The applicator is withdrawn when the filter fixes itself to the wall of the inferior vena cava after ejection from the applicator.

In patients with stable PE, the initial anticoagulant selected may include an LMWH (e.g., enoxaparin), unfractionated heparin, or a direct oral anticoagulant (DOAC), such as a direct thrombin inhibitor (e.g., dabigatran), or a factor Xa inhibitor (e.g., fondaparinux, rivaroxaban, apixaban, edoxaban) (Tapson & Weinberg, 2020) (see [Table 26-2](#)).

In select patients with PE who are hemodynamically stable, outpatient therapy can be started by administering the first dose in the emergency department or urgent care center and the remaining doses given at home. Although there are not specific selection criteria for outpatient treatment, the patient is usually at low risk of death, has no respiratory or hemodynamic compromise, does not require opioids for pain control, has no risk factors for bleeding, has no serious comorbid conditions, and has stable baseline mental status with a good understanding of the benefits and risks of treatment (Tapson, 2019). The ideal agent for outpatient administration is not empirically confirmed, although the DOACs are often prescribed.

Long-term treatment options include warfarin and the DOACs. An LMWH may also be selected but is usually not prescribed for long-term therapy since it is given via a subcutaneous injection. Warfarin dosing requires regular blood draws for INR monitoring and has a higher bleeding risk, but it has long been the standard of care prior to the development of DOACs. An antidote (vitamin K) is available if the INR is high and there is a risk of bleeding. Warfarin does have interactions with several medications (see [Chart 26-10](#)) and has dietary restrictions. DOACs do not require regular blood test monitoring; however, they are more costly than warfarin. The choice of warfarin versus a DOAC is dependent upon risk of bleeding, cost, presence of comorbidities, and provider preference (The Joint Commission [TJC], 2019).

## Nursing Management

### Monitoring Thrombolytic Therapy

The nurse is responsible for monitoring the patient's response to thrombolytic and anticoagulant therapy. During the thrombolytic infusion, while the patient remains on bed rest, vital signs are frequently assessed and invasive procedures are avoided. Tests to determine INR or aPTT are performed 3 to 4 hours after the thrombolytic infusion is started to confirm that the fibrinolytic systems have been activated.



### Quality and Safety Nursing Alert

*Because of the prolonged clotting time, only essential arterial punctures or venipunctures are performed in patients who have received thrombolytics, and manual pressure is applied to any puncture site for at least 30 minutes. Pulse oximetry is used to monitor changes in oxygenation. The thrombolytic infusion is discontinued immediately if uncontrolled bleeding occurs.*

## Managing Pain

Chest pain, if present, is usually pleuritic rather than cardiac in origin. A semi-Fowler position provides a more comfortable position for breathing. However, the nurse must continue to turn patients frequently and reposition them to improve V/Q. The nurse administers opioid analgesic agents as prescribed for severe pain.

## Managing Oxygen Therapy

Careful attention is given to the proper use of oxygen. The patient must understand the need for continuous oxygen therapy. The nurse assesses the patient frequently for signs of hypoxemia and monitors the pulse oximetry values to evaluate the effectiveness of the oxygen therapy. Deep breathing and incentive spirometry are indicated for all patients to minimize or prevent atelectasis and improve ventilation (see [Chapter 19](#) for discussion of incentive spirometry). Nebulizer therapy or percussion and postural drainage may be used for management of secretions.

## Relieving Anxiety

The nurse encourages the patient who is stabilized to talk about any fears or concerns related to this frightening episode, answers the patient's and family's questions concisely and accurately, explains the therapy, and describes how to recognize untoward effects early.

## Monitoring for Complications

When caring for a patient who has had PE, the nurse must be alert for the potential complication of cardiogenic shock or right ventricular failure subsequent to the effect of PE on the cardiovascular system. (Nursing

activities for managing shock are found in [Chapter 11](#); see [Chapter 25](#) for nursing management of right ventricular failure.)



## Providing Postoperative Nursing Care

If the patient has undergone surgical embolectomy, the nurse measures the patient's pulmonary arterial pressure and urinary output. The nurse also assesses the insertion site of the arterial catheter for hematoma formation and infection. Maintaining the blood pressure at a level that supports perfusion of vital organs is crucial. To prevent peripheral venous stasis and edema of the lower extremities, the nurse elevates the foot of the bed and encourages isometric exercises, the use of intermittent pneumatic compression devices, and walking when the patient is permitted out of bed. Sitting for long periods is discouraged, because hip flexion compresses the large veins in the legs.

## Promoting Home, Community-Based, and Transitional Care



### Educating Patients About Self-Care

Before hospital discharge and at follow-up visits to the clinic, the nurse educates the patient about preventing recurrence and reporting signs and symptoms. Patient education instructions, presented in [Chart 26-11](#), are intended to help prevent recurrences and side effects of treatment.

**Chart 26-11**



### HOME CARE CHECKLIST

## **Prevention of Recurrent Pulmonary Embolism**

**At the completion of education, the patient and/or caregiver will be able to:**

- State the impact of pulmonary embolism (PE) on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State changes in lifestyle (e.g., diet, activity) necessary to restore health.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Name the anticoagulant prescribed and describe relevant patient education related to taking anticoagulants described in [Chart 26-10](#).
- Describe the importance of follow-up appointments with providers.
- Describe strategies to prevent recurrent deep venous thrombosis and pulmonary emboli:
  - Continue to wear anti-embolism stockings (compression hose) as long as directed.
  - Avoid sitting with legs crossed or sitting for prolonged periods of time.
  - When traveling, change position regularly, walk occasionally, and do active exercises of moving the legs and ankles while sitting.
  - Drink fluids, especially while traveling and in warm weather, to avoid hemoconcentration due to fluid deficit.
- Describe the signs and symptoms of lower extremity circulatory compromise and potential deep venous thrombosis: calf or leg pain, swelling, pedal edema.
- Describe the signs and symptoms of pulmonary compromise related to recurrent PE (e.g., dyspnea, chest pain, anxiety, fever, tachycardia, apprehension, cough, syncope, diaphoresis, hemoptysis).
- Describe how and when to contact the primary provider if symptoms of circulatory compromise or pulmonary compromise are identified.
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

## Continuing and Transitional Care

During follow-up or home visits, the nurse monitors the patient's adherence to the prescribed management plan and reinforces previous instructions. The nurse also monitors the patient for residual effects of the PE and recovery. The patient is reminded about the importance of keeping follow-up appointments for coagulation tests, if indicated, and appointments with the primary provider.

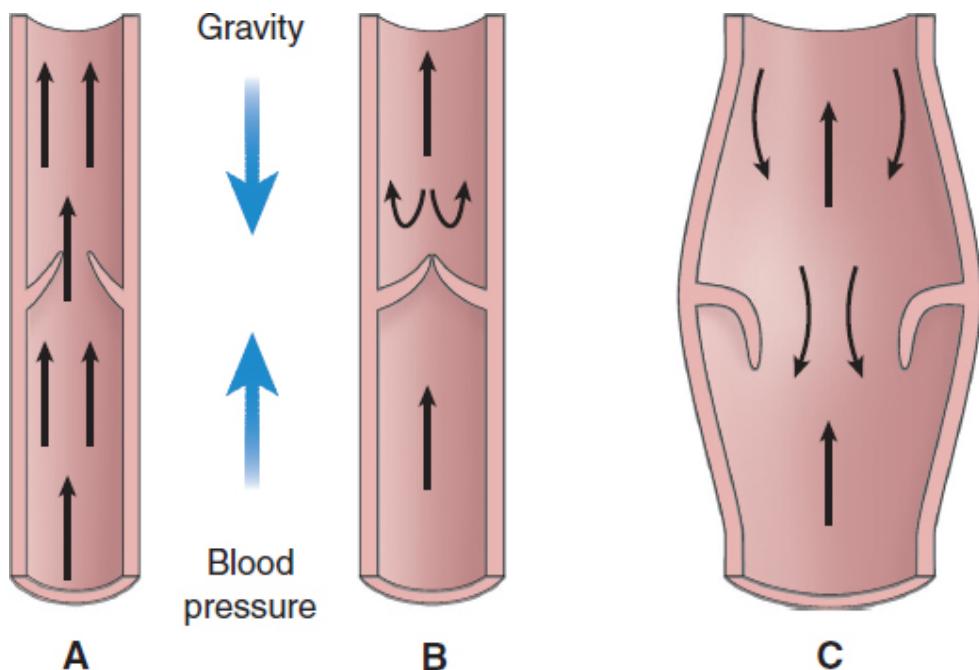
## Chronic Venous Insufficiency/Postthrombotic Syndrome

Venous insufficiency results from obstruction of the venous valves in the legs or a reflux of blood through the valves. Superficial and deep leg veins can be involved. Resultant venous hypertension can occur whenever there has been a prolonged increase in venous pressure, such as occurs with DVT. Because the walls of veins are thinner and more elastic than the walls of arteries, they distend readily when venous pressure is consistently elevated. In this state, leaflets of the venous valves are stretched and prevented from closing completely, causing a backflow or reflux of blood in the veins. Duplex ultrasonography confirms the obstruction and identifies the level of valvular incompetence. Twenty percent to 50% of patients who have had a DVT develop deep vein incompetence leading to postthrombotic syndrome (Sidawy & Perler, 2019) (see Fig. 26-15).

## Clinical Manifestations

Postthrombotic syndrome is characterized by chronic venous stasis, resulting in edema, altered pigmentation, pain, and stasis dermatitis. The patient may notice the symptoms less in the morning and more in the evening. Obstruction or poor calf muscle pumping in addition to valvular reflux must be present for the development of severe postthrombotic syndrome and stasis ulcers. Superficial veins may be dilated. The disorder is long-standing, difficult to treat, and often disabling (Kahn, Galanaud, Vedantham, et al., 2016).

Stasis ulcers develop as a result of the rupture of small skin veins and subsequent ulcerations. When these vessels rupture, red blood cells escape into surrounding tissues and then degenerate, leaving a brownish discoloration of the tissues, called *hemosiderin staining*. The pigmentation and ulcerations usually occur in the lower part of the extremity, in the area of the medial malleolus of the ankle. The skin becomes dry, cracks, and itches; subcutaneous tissues fibrose and atrophy. The risk of injury and infection of the extremities is increased.



**Figure 26-15 •** Competent valves showing blood flow patterns when the valve is open (A) and closed (B), allowing blood to flow against gravity. C. With faulty or incompetent valves, the blood cannot move toward the heart.

## Complications

Venous ulceration is the most serious complication of chronic venous insufficiency and can be associated with other conditions affecting the circulation of the lower extremities. Cellulitis or dermatitis may complicate the care of chronic venous insufficiency and venous ulcerations.

## Management

Management of the patient with venous insufficiency is directed at reducing venous stasis and preventing ulcerations. Extremities with venous insufficiency must be carefully protected from trauma; the skin is kept clean, dry, and soft. Signs of ulceration are immediately reported to the primary provider for treatment and follow-up. Measures that increase venous blood flow are antigravity activities, such as elevating the leg, and compression of superficial veins with graduated compression stockings and other compression therapies.

Elevating the legs decreases edema, promotes venous return, and provides symptomatic relief. The legs should be elevated frequently throughout the day (at least 15 to 20 minutes four times daily). At night, the patient should sleep with the foot of the bed elevated about 15 cm (6 inches). Prolonged sitting or standing in one position is detrimental; walking should be encouraged. When sitting, the patient should avoid placing pressure on the popliteal spaces, as occurs when crossing the legs or sitting with the legs dangling over the side of the bed. Constricting garments, especially socks that are tight at the top or that leave marks on the skin, should be avoided.

Compression of the legs with graduated compression stockings reduces the pooling of venous blood, enhances venous return to the heart, and is recommended for people with venous insufficiency.

## Providing Compression Therapy

### Stockings

Graduated compression stockings usually are prescribed for patients with venous disease as soon as possible after diagnosis (Bjork & Ehmann, 2019). The amount of pressure gradient is determined by the amount and severity of venous disease. For example, a pressure gradient of 20 to 30 mm Hg is prescribed for patients with asymptomatic varicose veins, whereas at least a pressure gradient of 30 to 40 mm Hg at the ankle is recommended for patients with venous stasis ulceration (Bjork & Ehmann, 2019). Compression stockings with at least 30 to 40 mm Hg pressure can be used during the 6 months post DVT to decrease symptoms and reduce the development of postthrombotic syndrome (Zierler & Dawson, 2016). These stockings should not be confused with anti-embolism stockings (i.e., thromboembolic deterrent [TED] stockings) that provide less compression (12 to 20 mm Hg at the ankle). Graduated compression stockings are designed to apply 100% of the prescribed pressure gradient at the ankle and then decrease along the

length of the stocking, reducing the caliber of the superficial veins in the leg and increasing flow in the deep veins. Each stocking should fit so that pressure is greater at the foot and ankle and then gradually declines to a lesser pressure at the knee or groin. These stockings may be knee high, thigh high, or pantyhose. Several colors, fabrics, and styles are available to promote patient adherence. Stockings should be applied after the legs have been elevated for a period, when the amount of blood in the leg veins is at its lowest.



#### **Quality and Safety Nursing Alert**

*Any type of stocking can inadvertently become a tourniquet if applied incorrectly (i.e., rolled tightly at the top). In such instances, the stockings produce—rather than prevent—venous stasis. For ambulatory patients, graduated compression stockings can be removed at night and reapplied before the legs are lowered from the bed to the floor in the morning.*

When the stockings are off, the skin is inspected for signs of irritation, and the calves are examined for tenderness. Any skin changes or signs of tenderness are reported. Stockings are contraindicated in patients with severe PAD, epifascial arterial bypass, severe cardiac insufficiency, allergy to compression material, and severe diabetic neuropathy with sensory loss or microangiopathy (Rabe, Partsch, Morrison, et al., 2020).



#### **Gerontologic Considerations**

Older patients have decreased strength and manual dexterity and may be unable to apply graduated compression stockings. If this is the case, a family member or friend should be taught to assist the patient to apply the stockings so that they do not cause excessive pressure on any part of the feet or legs. Frames and other devices have been designed to assist patients with applying stockings, and if there is any concern regarding patients' physical abilities, they should be referred to an occupational therapist who can provide examples of and training in the use of stocking assistance devices (Balcombe, Miller, & McGuiness, 2017).

#### **External Compression Devices and Bandages**

Short-stretch elastic bandages may be applied from the toes to the knee in a 50% spiral overlap. These bandages are available in a two-layer

system, which includes an inner layer of soft padding. These bandages have extension indicators that are rectangular and become squares when extended correctly, which reduces the possibility of bandaging a leg too loosely or too tightly. Three- and four-layer systems are also available (e.g., Profore, Dyna-Care), but these may be used only once compared with the two-layer system, which can be used multiple times.

Other types of compression are available. The Unna boot, which consists of a paste bandage impregnated with zinc oxide, glycerin, gelatin, and sometimes calamine, is applied without tension in a circular fashion from the base of the toes to the tibial tuberosity with a 50% spiral overlap. The foot must remain dorsiflexed at a 90-degree angle to the leg, thus avoiding excess pressure or trauma to the anterior ankle area. Once the bandage dries, it provides a constant and consistent compression to the venous system. This type of compression may remain in place for as long as 1 week, although it may be too heavy for patients who are too frail to tolerate. The Unna boot is more commonly used with venous insufficiency.

The CircAid, a nonelastic leg wrap with a series of overlapping, interlocking Velcro straps, augments the effect of muscle while the patient is walking. The CircAid is usually worn during the day. Patients may find the CircAid easier to apply and wear than the Unna boot because it is lighter, can be removed to shower, and is adjustable. However, its adjustability may also be problematic; if patients loosen the straps, the compression achieved may not be adequate.

### Intermittent Pneumatic Compression Devices

Intermittent pneumatic compression devices can be used in conjunction with elastic bandages or graduated compression stockings to support venous circulation and prevent DVT. Compression devices consist of an electric controller that is attached by air hoses to either knee-high or thigh-high sleeves. The sleeves are divided into compartments, which sequentially fill to apply pressure to the ankle, calf, and thigh at variable pressures from 30 to 70 mm Hg. Intermittent pneumatic compression devices can increase blood velocity beyond that produced by elastic bandages or stockings. Intermittent pneumatic compression devices are prescribed for patients who are not physically able to apply compression bandages or wraps, or don a pair of stockings (Nicolaides, 2020). Nursing measures in caring for patients who use these devices include ensuring that sleeves are properly encircling the extremity and the

prescribed pressures are set and not exceeded, assessing for patient comfort, and ensuring adherence to therapy.

## Leg Ulcers

A leg ulcer is an excavation of the skin surface that occurs when inflamed necrotic tissue sloughs off. In the United States the most common lower extremity ulcerations have a venous etiology (estimated between 80% and 90%), with PAD the second leading cause. The coexistence of both venous and arterial disease is estimated to be present in 26% of patients with leg ulcers (Singer, Tassiopoulos, & Kirsner, 2017).

## Pathophysiology

Inadequate exchange of oxygen and other nutrients in the tissue is the metabolic abnormality that underlies the development of leg ulcers. When cellular metabolism cannot maintain energy balance, cell necrosis (death) results. Alterations in blood vessels at the arterial, capillary, and venous levels may affect cellular processes and lead to the formation of ulcers.

## Clinical Manifestations

The characteristics of leg ulcers are determined by the cause of the ulcer (Ermer-Selton, 2016). Most ulcers, especially in older patients, have more than one cause. The symptoms depend on whether the problem is arterial or venous in origin (see [Table 26-1](#)). The severity of the symptoms depends on the extent and duration of the vascular insufficiency. The ulcer itself appears as an open, inflamed sore. The area may be exuding or covered by eschar (dark, hard crust).



**Figure 26-16 • A.** Ulcers resulting from arterial emboli. **B.** Gangrene of the toes resulting from severe arterial ischemia. **C.** Ulcer from venous stasis.

### Arterial Ulcers

Chronic arterial disease is characterized by intermittent claudication. The patient may also complain of digital or forefoot pain at rest. If the onset of arterial occlusion is acute, ischemic pain is unrelenting and rarely relieved even with opioids. Typically, arterial ulcers are small, circular, deep ulcerations on the tips of toes or in the web spaces between the toes. Ulcers often occur on the medial side of the hallux or lateral fifth toe and may be caused by a combination of ischemia and pressure (see Fig. 26-16).

### **Venous Ulcers**

Chronic venous insufficiency is characterized by pain described as aching or heavy. The foot and ankle may be edematous. Ulcerations are in the area of the medial or lateral malleolus (gaiter area) and are typically large, superficial, and highly exudative. Venous hypertension causes extravasation of blood, which discolors the area (see Fig. 26-16). Studies report the average venous ulcer requires as long as 6 to 12 months to heal completely and in patients who do not adhere to compression therapy (see discussion later in the chapter), the recurrence rate is nearly 100% within 36 months (Nicolaides, 2020). Patients with neuropathy (e.g., a common occurrence in patients with diabetes) frequently have ulcerations on the side of the foot over the metatarsal heads. These ulcers are painless and are described in further detail in Chapter 46.

## **Assessment and Diagnostic Findings**

The cause of each ulcer needs to be identified so that appropriate therapy can be prescribed. The history of the condition is important in determining arterial or venous insufficiency. The pulses of the lower extremities (femoral, popliteal, posterior tibial, and dorsalis pedis) are carefully examined. More conclusive diagnostic aids include Doppler and duplex ultrasound studies, arteriography, and venography. Cultures of the ulcer bed may be necessary to determine whether an infection is contributing to tissue destruction.

## **Medical Management**

Patients with ulcers can be effectively managed by advanced practice nurses or wound-ostomy-continence nurses in collaboration with the

patients' primary provider. All ulcers have the potential to become infected.

## Pharmacologic Therapy

Antiseptic agents, such as povidone–iodine, cadexomer iodine, and silver, inhibit growth and development of most microbes, are broad spectrum, generate relatively little antimicrobial resistance, and can be used for short periods of time. Once a wound is infected (e.g., erythema, induration, exudate, edema, wound breakdown, malodor), a systemic antibiotic is necessary (Swanson, Angel, Sussman, et al., 2016). The specific antibiotic agent selected is based on culture and sensitivity test results. Oral antibiotics usually are prescribed because topical antibiotics have not proven to be effective for leg ulcers and promote antimicrobial resistance (Swanson et al., 2016).

Pharmacologic therapy also is important in the management of wound-related pain.

## Compression Therapy

After the circulatory status of the patient is assessed and an absolute ankle pressure of greater than 60 mm Hg and an ABI that exceeds 0.80 are confirmed, compression therapy can be used up to 40 mm Hg without impeding arterial perfusion (Singer et al., 2017). See the discussion on compression therapy in the Chronic Venous Insufficiency section.

## Cleansing and Débridement

To promote healing, the wound bed is prepared by removing excessive wound exudate and nonviable tissue. The usual cleansing method is to flush the area with water or normal saline solution or clean it with a noncytotoxic wound-cleansing agent (Saf-Cleens<sup>TM</sup>, Biolex<sup>TM</sup>, Restore<sup>TM</sup>). If this is unsuccessful, therapeutic wound cleansing and/or débridement may be necessary (Swanson et al., 2016).

Therapeutic wound cleansing includes mechanical cleaning with a cleansing solution or gel such as:

- Polyhexamethylene biguanide (PHMB)
- Octenidine dihydrochloride
- Superoxidised solution with hypochlorous acid (HOCL) and sodium hypochlorite (NaOCL)
- Povidone–iodine

Many older antiseptics are no longer recommended due to the risk of tissue damage associated with their use. However, these agents may still be used for wound management in low-resource settings, where alternative, contemporary antiseptics are not always available. Solutions not recommended include:

- Hydrogen peroxide
- High-concentration sodium hypochlorite (EUSOL™, Milton, Dakin's solution)
- Chlorhexidine gluconate
- Chlorhexidine gluconate and cetrimide (Savlon™)
- High-concentration acetic acid
- Antibiotics for systemic administration
- High-concentration potassium permanganate

Débridement is the removal of nonviable tissue from wounds. Removing nonviable tissue is important, particularly for infection and biofilms (i.e., the microorganisms that grow on the surface of the wound). Biofilms have now been established as an important factor in wound healing and the need for serial débridement is important to disrupt the biofilm and allow the penetration of topical and systemic antimicrobial agent (Swanson et al., 2016).

Débridement can be accomplished by several different methods:

- Surgical débridement is the fastest method and can be performed by a primary provider under aseptic conditions using instruments to excise nonviable tissue. It is usually performed in the operating room with access to anesthesia and hemostasis.
- Conservative sharp wound débridement can be performed at the bedside or in a clinic by a primary provider, skilled advanced practice nurse or wound-ostomy-continence nurse in collaboration with the primary provider or advanced practice nurse. Instruments are used to remove loose, avascular, and insensate nonviable tissue and topical anesthetics may be used to manage procedural pain.
- Chemical débridement involves the application of chemical agents (e.g., cadexomer iodine, hypertonic saline) for the controlled removal of nonviable tissue. There may be some cytotoxic effects to healthy cells in the wound.
- Ultrasonic débridement uses ultrasonography to disrupt the attachment of the nonviable tissue.

Hydrosurgical débridement uses a water-jet-powered wound

- débridement tool to cut nonviable tissue.
- Biologic débridement, or larval therapy, involves the deliberate infestation of disinfected fly larvae that secrete a proteolytic enzyme which liquifies and ingests nonviable tissues.
- Enzymatic débridement involves the application of ointments containing enzymes. The ointment is applied to the ulcer but not to surrounding skin. Most enzymatic ointments are covered with a secondary dressing that will not soak up the ointment. The enzymatic ointment is discontinued when the nonviable tissue has been débrided, and an appropriate wound dressing is applied.
- Autolytic débridement is achieved through the application of dressings that allow the lysozymes in wound exudate to naturally break down the nonviable tissue. These include calcium alginate dressings (e.g., Kaltostat<sup>TM</sup>, Sorbsan<sup>TM</sup>) or gelling fiber dressings (e.g., Aquacel Hydrofiber<sup>TM</sup>) and may be used for débridement when absorption of exudate is needed. These dressings are changed when the exudate seeps through the secondary dressing, or at least every 7 days. Calcium alginate dressings can be used on areas that are bleeding after débriding, because the material helps stop the bleeding. As the dry fibers absorb exudate, they become a gel that can be painlessly removed from the ulcer bed. Calcium alginate and gelling fiber dressings should not be used on dry or nonexudative wounds.

Nonselective débridement by applying and removing saline dressings of fine mesh gauze (wet to dry) are not recommended for vascular wounds because of the risk of removing viable tissue and wound-related pain. Preprocedural pain management is usually necessary.

Arterial insufficiency may result in gangrene of the toe(s), or digital gangrene, which is usually caused by trauma. The toe is stubbed and then turns black (see Fig. 26-16). Usually, patients with this problem are older people without adequate circulation. Débridement is contraindicated in these instances. Although the toe is gangrenous, it is dry. Managing dry gangrene is preferable to débriding the toe and causing an open wound that will not heal because of insufficient circulation. If the toe were to be amputated, the lack of adequate circulation would prevent healing and might make higher-level amputation necessary—a below- or an above-

knee amputation. A higher-level amputation in an older adult could result in a loss of independence and possibly the need for institutional care. Dry gangrene of the toe in an older adult with poor circulation is usually left undisturbed. The nurse keeps the toe clean and dry, if it is stable, until it autoamputates or separates (without creating an open wound).

## Topical Therapy

A variety of topical agents can be used in conjunction with cleansing and débridement to promote healing of leg ulcers. The goals of treatment are to remove nonviable tissue and biofilm, and keep the ulcer clean and moist while healing takes place. The treatment should not destroy developing tissue. For topical treatments to be successful, adequate nutrition must be maintained.

## Wound Dressing

Semicoclusive or occlusive wound dressings prevent evaporative fluid loss from the wound and retain warmth; these factors favor healing. When determining the appropriate dressing to apply, the following should be considered: simplicity of application, frequency of required dressing changes, ability to absorb wound exudate, expense, and patient comfort (see [Chapter 56](#) for further discussion of wound dressings).

Knowledge deficit, frustration, fear, anxiety, and depression can decrease the patient's and family's adherence with the prescribed therapy; therefore, patient and family education is necessary before beginning and throughout the wound care program.

## Stimulated Healing

Tissue-engineered human skin equivalent (e.g., Apligraf<sup>TM</sup> [Graftskin<sup>TM</sup>]) is a skin product cultured from human dermal fibroblasts and keratinocytes used in combination with therapeutic compression. When applied, it interacts with the patient's cells within the wound to stimulate the production of growth factors. Application is not difficult, no suturing is involved, and the procedure is painless. A dermal repair scaffold (e.g., PriMatrix<sup>TM</sup>) is a bioactive and regenerative extracellular matrix that binds with the patient's own cells and growth factors. PriMatrix<sup>TM</sup> has been used successfully for tunneling wounds, as well as wounds with exposed tendon and bone, in which Apligraf<sup>TM</sup> cannot be used.

Dermagraft™, which is a human fibroblast–derived dermal replacement, demonstrates efficacy similar to Apligraf™ (Nicolaides, 2020).

## Hyperbaric Oxygenation

Hyperbaric oxygenation (HBO) may be beneficial as an adjunct treatment in patients with diabetes with no signs of wound healing after 30 days of standard wound treatment. HBO is accomplished by placing the patient into a chamber that increases barometric pressure while the patient is breathing 100% oxygen. Treatment regimens vary from 90 to 120 minutes once daily for 30 to 90 sessions. The process by which HBO is thought to work involves several mechanisms. The edema in the wound area is decreased because high oxygen tension facilitates vasoconstriction and enhances the ability of leukocytes to phagocytize and kill microbes. In addition, HBO is thought to increase diffusion of oxygen to the hypoxic wound, thereby enhancing epithelial migration and improving collagen production. The two most common adverse effects of HBO are middle-ear barotrauma and confinement anxiety. The benefit from this therapy on wound healing in patients without diabetes is unclear (Bonifant & Holloway, 2019).

## Negative Pressure Wound Therapy

Research suggests that negative pressure wound therapy (NPWT) using vacuum-assisted closure (e.g., VAC™) devices decreases time to healing in complex wounds that have not healed in a 3-week period. Groin incisions, common in vascular surgery, may be complicated by wound dehiscence, lymphatic fistula, or infections in 5% to 10% of patients. NPWT has been found to be effective in treating patients who develop postoperative groin wound infections, decreasing hospital lengths of stay, rates of graft infection, and likelihood of limb loss (Apelqvist, Willy, Fagerdahl, et al., 2017). Patients who are ambulatory may be given small, portable NPWT devices, giving them the freedom to perform their ADLs (Harding, Chrysostomou, Mohamud, et al., 2017). Additional features such as instillation therapy (Veraflow™) are now available. This therapy facilitates cleansing via instillation of fluid followed by a negative pressure cycle. Research to evaluate the microbial load and changes in the bacterial spectrum in wounds while using a growing number of NPWT devices is ongoing (Kim, Applewhite, Dardano, et al., 2018).

## NURSING PROCESS

### The Patient with Leg Ulcers

#### Assessment

A focused nursing history and assessment are important. The extent and type of pain are carefully assessed, as are the appearance and temperature of the skin of both legs. The quality of all peripheral pulses is assessed, and the pulses in both legs are compared. The legs are checked for edema. If the extremity is edematous, the degree of edema is determined. Any limitation of mobility and activity that results from vascular insufficiency is identified. The patient's nutritional status is assessed, and a history of diabetes, collagen disease, or varicose veins is obtained.

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include:

- Impaired skin integrity associated with vascular insufficiency
- Impaired mobility associated with activity restrictions of the therapeutic regimen and pain
- Impaired nutritional status associated with increased need for nutrients that promote wound healing

##### COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Infection
- Gangrene

#### Planning and Goals

The major goals for the patient may include restoration of skin integrity, improved physical mobility, adequate nutrition, and absence of complications.

#### Nursing Interventions

Caring for these patients can be challenging, whether the patient is in the hospital, in a long-term care facility, or at home. Leg ulcers are often long term and disabling causing a substantial drain on the patient's physical, emotional, and economic resources.

##### RESTORING SKIN INTEGRITY

To promote wound healing, measures are used to keep the area clean. Cleansing requires very gentle handling, a neutral skin cleanser, and lukewarm water. Positioning of the legs depends on whether the ulcer is of arterial or venous origin. If there is arterial insufficiency, the patient should be referred for evaluation of vascular reconstruction. If there is venous insufficiency, dependent edema can be avoided by elevating the lower extremities and initiating graduated compression therapy. A decrease in edema promotes the exchange of cellular nutrients and waste products in the area of the ulcer, promoting healing.

Avoiding trauma to the lower extremities is imperative in promoting skin integrity. Heel suspension devices such as protective boots may be used (e.g., Rooke vascular boots, Prevalon); they are soft and provide warmth and protection from injury and displace tissue pressure to prevent pressure injury. If the patient is on bed rest or has reduced mobility, it is important to relieve pressure on the heels to prevent heel pressure injuries. When the patient is in bed, a bed cradle can be used to relieve pressure from bed linens and to prevent anything from touching the legs. When the patient is ambulatory, all obstacles are moved from the patient's path so that the patient's legs are not bumped. Heating pads, hot-water bottles, or hot baths are avoided, because they increase the oxygen demands and thus the blood flow demands of the already compromised tissue. The patient with diabetes and neuropathy has decreased sensation; therefore, heating pads may cause a burn without the patient noticing.

#### **IMPROVING PHYSICAL MOBILITY**

Generally, physical activity is initially restricted to promote wound healing. When infection resolves and healing begins, ambulation should resume gradually and progressively. Activity promotes arterial blood flow and venous return and is encouraged after the acute phase of the ulcer process. Until full activity is resumed, the patient is advised to move about when in bed, to turn from side to side frequently, and to exercise the upper extremities to maintain muscle tone and strength. Meanwhile, diversional activities are encouraged. Consultation with an occupational therapist and physical therapist may be helpful if prolonged immobility and inactivity are anticipated.

If pain limits the patient's activity, analgesic agents may be prescribed. The wound-related pain is typically chronic and often

disabling. Analgesic agents may be taken before scheduled activities to help the patient participate more comfortably.

#### **PROMOTING ADEQUATE NUTRITION**

Nutritional deficiencies are common, requiring dietary alterations to remedy them. There is conflicting evidence that dietary supplementation aids in healing of ulcerations. Comorbidities that may contribute to ulceration may also cause ongoing inflammation, disuse atrophy, and other metabolic disturbances; these may have a greater effect on wound healing than nutritional intake. Further research is needed; however, eating a diet that is high in protein, vitamins C and A, iron, and zinc is encouraged to promote healing (Bonifant & Holloway, 2019). Particular consideration should be given to iron intake, because many patients are older adults who are at risk for iron deficiency anemia. After a dietary plan has been developed that meets the patient's nutritional needs and promotes healing, dietary instruction is provided to the patient and family.

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**

The self-care program is planned with the patient so that activities that promote arterial and venous circulation, relieve pain, and promote tissue integrity are encouraged. Reasons for each aspect of the program are explained to the patient and family. Leg ulcers are often chronic and difficult to heal; they frequently recur, even when the patient follows the plan of care. Long-term care of the feet and legs to promote healing of wounds and prevent recurrence of ulcerations is the primary goal. Leg ulcers increase the patient's risk of infection, may be painful, and may limit mobility, necessitating lifestyle changes. Participation of family members and home health care providers may be necessary for treatments such as dressing changes, reassessments, reinforcement of instruction, and evaluation of the effectiveness of the plan of care. Regular follow-up with a primary provider is necessary.

#### **Evaluation**

Expected patient outcomes may include:

1. Demonstrates restored skin integrity
  - a. Exhibits absence of inflammation
  - b. Exhibits absence of drainage
  - c. Has negative wound culture
  - d. Avoids trauma to the legs

2. Increases physical mobility
  - a. Progresses gradually to optimal level of activity
  - b. Reports that pain does not impede activity
3. Attains adequate nutrition
  - a. Selects foods high in protein, vitamins C and A, iron, and zinc
  - b. Discusses with family members dietary modifications that need to be made at home
  - c. Plans, with the family, a diet that is nutritionally sound

## Varicose Veins

Varicose veins (varicosities) are abnormally dilated, tortuous, superficial veins caused by incompetent venous valves (see [Fig. 26-15](#)). Most commonly, this condition occurs in the lower extremities, the saphenous veins, or the lower trunk, but it can occur elsewhere in the body, such as the esophagus (e.g., esophageal varices; see [Chapter 43](#)).

It is estimated that varicose veins occur in 23% of American adults, and if spider telangiectasias and reticular veins are included in these statistics, the prevalence increases to 80% of men and 85% of women (Sidawy & Perler, 2019). The condition is most common in people whose occupations require prolonged standing, such as salespeople, hairstylists, teachers, nurses and ancillary medical personnel, and construction workers. A hereditary weakness of the vein wall may contribute to the development of varicosities, and it commonly occurs in several members of the same family. Varicose veins are rare before puberty. Pregnancy may cause varicosities because of hormonal effects related to decreased venous outflow, increased pressure by the gravid uterus, and increased blood volume (Della Torre, Sutherland, & Digiovanni, 2019).

## Pathophysiology

Varicose veins may be primary (without involvement of deep veins) or secondary (resulting from obstruction of deep veins). A reflux of venous blood results in venous stasis. If only the superficial veins are affected, the person may have no symptoms but may be concerned by the appearance of the veins.

## Clinical Manifestations

Symptoms, if present, may include dull aches, muscle cramps, increased muscle fatigue in the lower legs, ankle edema, and a feeling of heaviness of the legs. Nocturnal cramps are common. When deep venous obstruction results in varicose veins, the patient may develop the signs and symptoms of chronic venous insufficiency: edema, pain, pigmentation, and ulcerations. Susceptibility to injury and infection is increased, thus increasing risk for ulceration.

## Assessment and Diagnostic Findings

Diagnostic tests for varicose veins include the duplex ultrasound scan, which documents the anatomic site of reflux and provides a quantitative measure of the severity of valvular reflux. These scans are typically performed in a reverse Trendelenburg position or with the patient standing. Venography is now rarely performed due to the availability of ultrasound. However, when it is used, it involves injecting a radiopaque contrast agent into the leg veins so that the vein anatomy can be visualized by x-ray studies during various leg movements. CT venography can be helpful, especially if the pelvic venous structures are involved.

## Prevention and Medical Management

The patient should avoid activities that cause venous stasis, such as wearing socks that are too tight at the top or that leave marks on the skin, crossing the legs at the thighs, and sitting or standing for long periods. Changing position frequently, elevating the legs 3 to 6 inches higher than heart level when they are tired, and getting up to walk for several minutes of every hour promote circulation. The patient is encouraged to walk 30 minutes each day if there are no contraindications. Walking up the stairs rather than using the elevator or escalator is helpful, and swimming is good exercise.

Graduated compression stockings, especially knee-high stockings, are useful. The patient who is overweight should be encouraged to begin a weight reduction plan.

The most common treatment options for venous insufficiency and varicose veins are thermal ablation with radiofrequency and laser therapy, micro (stab) phlebectomy, and foam sclerotherapy. Surgical

ligation and stripping are reserved for select cases not amenable to other treatment options.

## Thermal Ablation

Thermal ablation is a nonsurgical approach using thermal energy. Radiofrequency ablation uses an electrical contact inside the vein. As the device is withdrawn, the vein is sealed. Laser ablation uses a laser fiber tip that seals the vein (decompressed). Topical gel may be used first to numb the skin along the course of the saphenous vein. To protect the surrounding tissue, several small punctures are made along the vein, and 100 to 200 mL of dilute lidocaine is delivered to the perivenous space using ultrasound guidance. The goal of this tumescent anesthesia (i.e., anesthesia that causes localized swelling) is to provide analgesia, thermal protection (the cuff of fluid surrounds the veins and accompanying nerves), and extrinsic compression of the vein (Poder, Fisette, Bédard, et al., 2018). The saphenous vein is entered percutaneously near the knee using ultrasound guidance. A catheter is introduced into the saphenous vein and advanced to the saphenofemoral junction. The device is then activated and withdrawn, sealing the vein. Bandages or graduated compression stockings are applied after the procedure. A simultaneous microphlebectomy of branch varicosities may be performed; this is associated with a lower incidence of thrombophlebitis. This is also associated with quality of life improvements such as decreased leg swelling, pain, skin changes and healing of ulcerations, because all symptomatic veins may be treated at one time (Berti-Hearn & Elliott, 2019).

Cyanoacrylate embolization is approved for the treatment of an incompetent greater saphenous vein. Cyanoacrylate adhesive has been used for the treatment of arteriovenous malformations and a modified cyanoacrylate adhesive was developed that has rapid polymerization on contact with blood and tissue, flexibility sufficient to tolerate dynamic movements in the legs without generation of symptoms or be perceptible by the patient, and has a high viscosity to decrease the risk of propagation or embolization into the deep veins (Sidawy & Perler, 2019). During this procedure, a sheath is placed into the greater saphenous vein (guided by ultrasound). A catheter is advanced to the proximal saphenous vein and injections of cyanoacrylate are given, followed by local compression, and then repeated injections with repeated compression until the entire length of the target vein segment is

treated. Cyanoacrylate embolization has been associated with less procedural ecchymosis because heat is not needed during this procedure.

### **Microphebectomy**

If there are superficial varicose veins that are close to the surface, a microphebectomy may be performed. This procedure involves removal of a superficial varicosity using anywhere from one to 20 small incisions. There can be extensive bruising and risk of infection with this procedure (Sidawy & Perler, 2019).

### **Sclerotherapy**

Sclerotherapy involves injection of an irritating chemical into a vein to produce localized phlebitis and fibrosis, thereby obliterating the lumen of the vein. This treatment may be performed alone for small varicosities or may follow vein ablation, ligation, or stripping. Sclerotherapy is typically performed in a procedure room and does not require sedation. After the sclerosing agent is injected, graduated compression stockings are applied to the leg and are worn for approximately 1 week after the procedure. Ultrasound-guided foam sclerotherapy has been found to be more effective in achieving closure of the branch veins. It is also associated with decreased symptoms of leg aching, itching, and edema, less skin changes and ulcerations, and increased patient satisfaction (Berti-Hearn & Elliott, 2019). After sclerotherapy, walking is encouraged to activate the calf muscle pump and maintain blood flow in the leg.

### **Ligation and Stripping**

Surgery for symptomatic varicose veins requires that the deep veins be patent and functional. The saphenous vein is ligated high in the groin, where the saphenous vein meets the femoral vein. In addition, the vein may be stripped (removed). After the vein is ligated, an incision is made 2 to 3 cm below the knee, and a metal or plastic wire is passed the full length of the vein to the point of ligation. The wire is then withdrawn, pulling (stripping) the vein as it is removed. Pressure and elevation minimize bleeding during surgery.

## **Nursing Management**

Thermal ablation is performed in an outpatient or clinic setting. The patient is advised to maintain compression on the affected limb for at least 24 hours and then wear compression stockings while ambulatory for at least 1 week post procedure. Patients have no activity restrictions, but are advised to avoid strenuous exercise, such as weight lifting, bicycle riding, or swimming for 2 weeks. The patient is informed that bruising may occur along the course of the saphenous vein and that they may experience leg cramps for a few days and may find it difficult to straighten the knee for up to 1.5 weeks. Nonsteroidal anti-inflammatory medications such as ibuprofen and cool compresses are used as needed for pain.

Ligation and stripping can be performed in an outpatient setting, or the patient may be admitted to the hospital on the day of surgery and discharged the same or next day if a bilateral procedure is to be performed or the patient is at high risk for postoperative complications. If the procedure is performed in an outpatient setting, nursing measures are the same as if the patient were hospitalized. Bed rest is discouraged, and the patient is encouraged to ambulate as soon as sedation has worn off. The patient is instructed to walk every hour for 5 to 10 minutes while awake for the first 24 hours if they can tolerate the discomfort, and then to increase walking and activity as tolerated. Graduated compression stockings are worn continuously for about 1 week after vein stripping. The nurse assists the patient to perform exercises and move the legs. The foot of the bed should be elevated. Standing and sitting are discouraged.

### **Promoting Comfort and Understanding**

Analgesic agents are prescribed to help the patient move the affected extremities more comfortably. Dressings are inspected for bleeding, particularly in the groin, where the risk of bleeding is greatest. The nurse is alert for reported sensations of “pins and needles.” Hypersensitivity to touch in the involved extremity may indicate a temporary or permanent nerve injury resulting from surgery, because the saphenous vein and nerve are close to each other in the leg. Any of these signs or symptoms should be reported to the primary provider.

Usually, the patient may shower after 24 hours. A clean towel is used to gently pat dry, not rub, the incisions. Application of skin lotion is avoided until the incisions are completely healed to avoid infection. The patient is instructed to apply sunscreen to the incisional area prior to sun

exposure; otherwise, hyperpigmentation of the incision, scarring, or both may occur.

If the patient has undergone sclerotherapy, a burning sensation in the injected leg may be experienced for 1 to 2 days. The nurse encourages the use of a mild analgesic medication as prescribed and walking to provide relief.

## Promoting Home, Community-Based, and Transitional Care

Long-term venous compression is essential after discharge, and the patient needs to obtain adequate supplies of graduated compression stockings or elastic bandages. Exercise of the legs is necessary; the development of an individualized plan requires consultation with the patient and the health care team.

## LYMPHATIC DISORDERS

The lymphatic system consists of a set of vessels that spread throughout most of the body, as described previously in this chapter. The fluid drained from the interstitial space by the lymphatic system is called *lymph*. The flow of lymph depends on the intrinsic contractions of the lymph vessels, the contraction of muscles, respiratory movements, and gravity. The lymphatic system of the abdominal cavity maintains a steady flow of chyle (digested fatty food) from the intestinal mucosa to the thoracic duct. In other parts of the body, the lymphatic system's function is regional; the lymphatic vessels of the head, for example, empty into clusters of lymph nodes located in the neck, and those of the extremities empty into nodes of the axillae and the groin.

### Lymphangitis and Lymphadenitis

Lymphangitis is an acute inflammation of the lymphatic channels. It arises most commonly from a focal area of infection in an extremity. Usually, the infectious organism is a *hemolytic streptococcus*. The characteristic red streaks that extend up the arm or the leg from an infected wound outline the course of the lymphatic vessels as they drain.

The lymph nodes located along the course of the lymphatic channels also become enlarged, red, and tender; this is referred to as acute lymphadenitis. They can also become necrotic and form an abscess, called suppurative lymphadenitis. The nodes involved most often are those in the groin, axilla, or cervical region.

Because these infections are nearly always caused by organisms that are sensitive to antibiotics, it is unusual to see abscess formation. Recurrent episodes of lymphangitis are often associated with progressive lymphedema. After acute attacks, a graduated compression stocking should be worn on the affected extremity for several months to prevent long-term edema.

## Lymphedema

Lymphedema may be primary (congenital malformations) or secondary (acquired obstructions). Tissue swelling occurs in the extremities because of an increased quantity of lymph that results from obstruction of lymphatic vessels. It is especially marked when the extremity is in a dependent position. Initially, the edema is soft and pitting. As the condition progresses, the edema becomes firm, nonpitting, and unresponsive to treatment. The most common type is congenital lymphedema, known as lymphedema praecox, which is caused by hypoplasia of the lymphatic system of the lower extremity. This disorder is usually seen in women and first appears before age 35 (Dayan, Ly, Kataru, et al., 2018).

The obstruction may be in the lymph nodes and the lymphatic vessels. Sometimes, it is seen in the arm after an axillary node dissection (e.g., for breast cancer) and in the leg in association with varicose veins or chronic thrombophlebitis. In the latter case, the lymphatic obstruction usually is caused by chronic lymphangitis. Lymphatic obstruction caused by a parasite (filaria) is most frequently seen in the tropics. When chronic swelling is present, there may be frequent bouts of acute infection characterized by high fever and chills and increased residual edema once the inflammation has resolved. These changes can lead to chronic fibrosis, thickening of the subcutaneous tissues, and hypertrophy of the skin. This specific type of lymphedema, in which chronic swelling of the extremity recedes only slightly with elevation, is referred to as elephantiasis. There are an estimated 120 million people in the world infected by lymph-dwelling filarial parasites; of these, 40 million have lymphedema and secondary infections, creating an enormous global burden (King, Suamani, Sanuku, et al., 2018).

## Medical Management

The goal of therapy is to reduce and control the edema and prevent infection. Active and passive exercises assist in moving lymphatic fluid into the bloodstream. External compression devices milk the fluid proximally from the foot to the hip or from the hand to the axilla. When the patient is ambulatory, custom-fitted graduated compression stockings or sleeves are worn; those with the highest compression strength (exceeding 40 mm Hg) are suggested; however, many patients cannot tolerate these pressures. When the leg is affected, continuous bed rest with the leg elevated may aid in mobilizing the fluids but is not practical long term. Manual lymphatic drainage performed by specially trained therapists is designed to direct or shift the congested lymph through functioning lymphatics that have preserved drainage. Manual lymphatic drainage is performed with light touch (as opposed to deep massage) to the proximal then distal lymphatic channels. Manual lymphatic drainage is incorporated in a sequential treatment approach used in combination with multilayer compression bandages, stockings or wraps, exercises, skin care, pressure gradient sleeves, and pneumatic pumps, depending on the severity and stage of the lymphedema (Patullo & Rajagopalan, 2017).

### **Pharmacologic Therapy**

As initial therapy, the diuretic furosemide may be prescribed to prevent fluid overload due to mobilization of extracellular fluid. Diuretics have also been used along with elevation of the leg and the use of graduated compression stockings or sleeves. The use of diuretics alone has little benefit because their main action is to limit capillary filtration by decreasing the circulating blood volume. If lymphangitis or cellulitis is present, antibiotic therapy is initiated. Lymphedema significantly increases the risk for cellulitis; therefore, the patient is taught to provide meticulous skin care and inspect the skin for evidence of infection.

### **Surgical Management**

Surgery is performed if the edema is severe and uncontrolled by medical therapy, if mobility is severely compromised, or if infection persists. One surgical approach involves the excision of the affected subcutaneous tissue and fascia, with skin grafting to cover the defect. Another procedure involves the surgical relocation of superficial lymphatic vessels into the deep lymphatic system, also known as lymph node transfer, by means of a buried dermal flap to provide a conduit for lymphatic drainage (Pappalardo, Patel, & Cheng, 2018).

Lymphaticovenous bypasses also are performed with anastomosing the end of the lymphatic vessels to the side of veins to reduce lymphatic flow in the limbs (Gallagher, Marulanda, & Gray, 2018).

## Nursing Management

After surgery, antibiotics may be prescribed for 3 to 7 days (Pappalardo et al., 2018). Constant elevation of the affected extremity and observation for complications are essential. Complications may include flap necrosis, hematoma or abscess under the flap, and cellulitis. The nurse instructs the patient or caregiver to inspect the dressing daily. Unusual drainage or any inflammation around the wound margin suggests infection and should be reported to the surgeon. The patient is informed that there may be a loss of sensation in the surgical area. The patient is also instructed to avoid the application of heating pads or exposure to sun to prevent burns or trauma to the area.

## CELLULITIS

Cellulitis is the most common infectious cause of limb swelling. Cellulitis can occur as a single isolated event or a series of recurrent events. It is sometimes misdiagnosed as recurrent thrombophlebitis or chronic venous insufficiency.

## Pathophysiology

Cellulitis occurs when an entry point through broken skin allows microbes to enter and release their toxins in the subcutaneous tissues. The etiologic pathogen of cellulitis is typically either *Streptococcus* species or *Staphylococcus aureus* (Bystritsky & Chambers, 2018).

## Clinical Manifestations

The onset of swelling, localized redness, warmth, and pain is frequently associated with systemic signs of fever, chills, and sweating. The redness may not be uniform and often skips areas and eventually develops a pitting “orange peel” appearance. Regional lymph nodes may also be tender and enlarged (Bystritsky & Chambers, 2018).



**Concept Mastery Alert**

Cellulitis needs to be differentiated from lymphangitis. With cellulitis, the swelling and redness is localized and anatomically nonspecific. With lymphangitis, characteristic red streaks appear denoting the outline of the lymphatic vessels that are affected.

## Medical Management

Mild cases of cellulitis can be treated on an outpatient basis with oral antibiotic therapy. If the cellulitis is severe, the patient is treated with IV antibiotics. The key to preventing recurrent episodes of cellulitis lies in adequate antibiotic therapy for the initial event and in identifying the site of microbial entry. Cracks and fissures that occur in the skin between the toes must be examined as potential sites of microbial entry. Other locations include drug use injection sites, contusions, abrasions, ulceration, ingrown toenails, and hangnails. Prophylactic compression therapy to reduce the risk of recurrence may be indicated in some cases (Webb, Neeman, Bowden, et al., 2020).

## Nursing Management

The patient is instructed to elevate the affected area 3 to 6 inches above heart level and apply cool packs to the site every 2 to 4 hours until the inflammation has resolved, and then transition to warm packs. Patients with sensory and circulatory deficits, such as those caused by diabetes and paralysis, should use caution when applying warm packs because burns may occur; it is advisable to use a thermometer or have a caregiver ensure that the temperature is not more than lukewarm. Education should focus on preventing a recurrent episode. The patient with peripheral vascular disease or diabetes should receive education or reinforcement about skin and foot care.

### CRITICAL THINKING EXERCISES

**1 pq** A 27-year-old male with Marfan syndrome presents to the emergency department where you work complaining of a sudden onset of chest pain with dyspnea and left leg weakness. You are unable to palpate pulses in his left foot. The patient's laboratory findings indicate a decline in renal function. How would you triage and prioritize your initial nursing care for this patient? The provider orders an urgent CT angiogram, which reveals a dissection of the aorta and obstructed flow to the left kidney and left leg. What are the ongoing priorities for managing this patient's care?

**2 ebp** A 32-year-old woman presents to the primary care clinic where you work with leg pain. She has recently been on a long airplane flight and has had some unilateral leg swelling with calf tightness and pain. Her only medication is an oral contraceptive agent; she is generally healthy. What are her risk factors for venous thromboembolism (VTE)? What further diagnostic studies should be considered and what evidence-based practice recommendations will guide the management of her care? What pharmacologic options might be considered for anticoagulation and what therapies are indicated to manage her leg edema?

**3 ipc** A 70-year-old male presents to a preadmission center where you work to prepare for surgical repair of an abdominal aortic aneurysm. His aneurysm is 5.8 cm which is an increase of 1 cm in the last 6 months. His hypertension is well controlled, and he has had no further angina since coronary artery bypass grafts (CABGs) 2 years ago. He has good exercise tolerance and his weight is within a normal range. What preoperative assessments are required? The patient asks what he should do if he experiences increasing abdominal, back, or flank pain before surgery. How will you respond? How will the interprofessional team manage his postoperative care and coordinate a safe discharge?

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\*Asterisk indicates nursing research.

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## Resources

- American Heart Association (AHA), [www.aha.org](http://www.aha.org)
- American Venous Forum (AVF), [www veinforum.org](http://www veinforum.org)
- Society for Vascular Medicine (SVM), [www.vascularmed.org](http://www.vascularmed.org)
- Society for Vascular Nursing (SVN), [www.svnnet.org](http://www.svnnet.org)
- Society for Vascular Surgery (SVS), [www.vascularweb.org](http://www.vascularweb.org)
- Society for Vascular Ultrasound (SVU), [www.svunet.org](http://www.svunet.org)

Vascular Cures, [www.vascularcures.org](http://www.vascularcures.org)

# 27 Assessment and Management of Patients with Hypertension

## LEARNING OUTCOMES

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

1. Compare and contrast normal blood pressure and various stages of hypertension.
2. Identify pathophysiologic processes implicated in the progression of hypertension.
3. Demonstrate the proper techniques to perform an assessment and discriminate between normal and abnormal findings identified in the patient with hypertension.
4. Discuss risk factors and treatment approaches for hypertension, including lifestyle modifications and medication therapy.
5. Use the nursing process as a framework for care of the patient with hypertension.
6. Describe hypertensive crises and their treatments.

## NURSING CONCEPTS

Assessment  
Perfusion

## GLOSSARY

**hypertensive emergency:** an emergent situation in which blood pressure is severely elevated and there is evidence of actual or probable target organ damage

**hypertensive urgency:** an urgent situation in which blood pressure is severely elevated but there is no evidence of impending or progressive target organ damage

**isolated systolic hypertension:** a disorder most commonly seen in the older adult in which the systolic pressure is greater than 140 mm Hg and the diastolic pressure is less than 80 mm Hg

**masked hypertension:** blood pressure that is typically suggestive of a diagnosis of hypertension that is paradoxically normal in health care settings

**primary hypertension:** high blood pressure with no identifiable cause (*synonym:* essential hypertension)

**rebound hypertension:** blood pressure in a patient with hypertension that is controlled with medication and becomes abnormally high with the abrupt discontinuation of that medication

**resistant hypertension:** high blood pressure treated with three or more antihypertensive medications of different classes; one of these must be a diuretic agent

**secondary hypertension:** high blood pressure from an identified cause, such as chronic kidney disease

**target organ damage:** manifestations of pathophysiologic changes in various organs as a consequence of hypertension

**white coat hypertension:** blood pressure that increases to hypertensive readings in health care settings that is paradoxically within the normal ranges in other settings

Hypertension is the most common chronic disease among adults in the United States and in the world (Whelton, Carey, Aronow, et al., 2017). It is identified as the leading risk factor for premature death, disability, and overall disease burden worldwide because it may lead to cardiovascular disease (CVD), stroke, and chronic kidney disease (CKD) when not appropriately treated (Caillon, Paradis, & Schiffrin, 2019; DePalma, Himmelfarb, MacLaughlin, et al., 2018). The overall risk of developing these CVDs, strokes, and renal disorders is low among patients with blood pressures that are consistently stable around 115/75 mm Hg; however, each increase of 20 mm Hg in the systolic blood pressure (SBP) or 10 mm Hg increase in the diastolic blood pressure (DBP) doubles the risk of death from stroke or heart disease (Lee, Kim, Kang, et al., 2018). And yet, most patients with hypertension could lower their blood pressure through lifestyle changes (e.g., diet, exercise, medication adherence, smoking cessation) and lower these associated morbid risks

(Whelton et al., 2017). This chapter presents an overview of hypertension and how it is defined and managed so that nurses may appropriately assess, monitor, educate, and intervene with patients with hypertension.

## Hypertension

For many years, patients were diagnosed with hypertension if they had chronically elevated SBPs of 140 mm Hg or higher or DBPs of 90 mm Hg or higher. These parameters, which also specified that the diagnosis of hypertension must be based on an average of two or more accurate readings taken one to 4 weeks apart, were endorsed by the *Seventh Report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure* (JNC 7) (Chobanian, Bakris, Black, et al., 2003), as well as the *Eighth Joint National Committee* (JNC 8) (James, Oparil, Carter, et al., 2014), and the *American Society of Hypertension* (ASH) and the *International Society of Hypertension* (ISH) (Weber, Schiffrin, White, et al., 2014). However, these parameters for diagnosing hypertension recently changed to less permissive parameters, and patients with average SBPs that are 130 mm Hg or higher or with average DBPs 80 mm Hg or higher may be diagnosed with hypertension, according to the *American College of Cardiology (ACC)/American Heart Association (AHA) Task Force* (Whelton et al., 2017).

The classification system for hypertension has been further revised by the ACC/AHA (Whelton et al., 2017) to include elevated, stage 1, and stage 2 categories, as displayed in [Table 27-1](#). [Table 27-1](#) compares this classification system to previous JNC 7 and JNC 8 guidelines classification systems, which are no longer followed (DePalma et al., 2018). The blood pressure categories emphasize the direct relationship between the SBP and DBP risks of morbidity, all-cause mortality, and specifically, cardiovascular mortality. Of particular note, the ACC/AHA guideline (Whelton et al., 2017) changed the previously labeled *prehypertension* category to *elevated blood pressure* category. The rationale for this change in terminology is to highlight the association between any elevated blood pressure and increased cardiovascular risk. The blood pressure readings should use the average of two or more valid, reproducible measurements obtained on more than two occasions, in most instances (see later discussion under Assessment and Diagnostic Findings).

The prevalence of hypertension among adults in the United States is substantially higher when the definition of the ACC/AHA guideline is used versus the JNC 7 or JNC 8 definition (46% vs. 32%) (Whelton et al., 2017). However, since nonpharmacologic treatment (i.e., lifestyle changes) is recommended for most adults whose blood pressures are within the elevated hypertension category, the newer guidelines that define hypertension have resulted in only a small increase in antihypertensive medication prescriptions,

overall. Indeed, it has been asserted that the greatest benefit of the ACC/AHA (2017) guideline is its greater emphasis on lifestyle interventions, which include weight loss, healthy diet, physical exercise, reduced sodium intake, increased potassium intake, and decreased alcohol intake (Ioannidis, 2018).

The prevalence of hypertension increases as people age or have other cardiovascular risk factors. Of all adults with hypertension, it is estimated that 35.3% do not know that they have this disorder. Furthermore, approximately 45.4% of people with hypertension do not have their blood pressure under control (Benjamin, Muntner, Alonso, et al., 2019). The prevalence of hypertension varies by ethnicity and gender, and is estimated at approximately 48.2% among Caucasian men, 41.3% among Caucasian women, 58.6% among African American men, 56% among African American women, 47.4% among Hispanic men, 40.8% among Hispanic women, 46.4% among Asian American men, and 36.4% among Asian American women. The prevalence of hypertension among African Americans is among the highest in the world (Benjamin et al., 2019). Moreover, African Americans tend to develop hypertension at younger ages than Caucasian Americans (Spikes, Higgins, Quyyumi, et al., 2019). [Chart 27-1](#) displays risk factors for hypertension.

Findings from the National Health and Nutrition Examination Survey (NHANES) have shown better hypertension control rates in women, in Caucasians than in African Americans and Hispanics, and in older versus younger patients. Additionally, adults of higher socioeconomic status have better control of their blood pressures compared to adults of lower socioeconomic status. Hypertension is most prevalent among adults 75 years of age and older, affecting 80% of men and 85.6% of women (Benjamin et al., 2019).

**TABLE 27-1** Comparing Blood Pressure Classifications by Key Guidelines for Adults Age 18 and Older

Systolic BP (mm Hg)		Diastolic BP (mm Hg)	ACC/AHA (2017) Guideline <sup>a</sup>	JNC 7 <sup>b</sup> and JNC 8 <sup>c</sup> Guidelines
<120	-and-	<80	Normal	Normal
120–129	-and-	<80	Elevated	Prehypertension
130–139	-or-	80–89	Stage 1 hypertension	Prehypertension
140–159	-or-	90–99	Stage 2 hypertension	Stage 1 hypertension
≥160	-or-	≥100	Stage 2 hypertension	Stage 2 hypertension

*Note:* For each guideline, if the patient's systolic and diastolic BPs fall into different categories, then the patient is classified according to the highest category. BP, blood pressure.

Adapted from <sup>a</sup>Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115; <sup>b</sup>Chobanian, A. V., Bakris, G. L., Black, H. R., et al.; National High Blood Pressure Education Program Coordinating Committee (2003). Seventh Report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 Report. *JAMA*, 289(19), 2560–2572; <sup>c</sup>James, P. A., Oparil, S., Carter, B. L., et al. (2014). 2014 evidence-based guideline for the management of high blood pressure in adults: Report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*, 311(5), 507–520.

## Chart 27-1 RISK FACTORS

## Hypertension

### Risk Factors

- Advancing adult age
- African American
- Chronic kidney disease
- Diabetes
- Drinking too much alcohol (i.e., more than two drinks per day for men and more than one drink per day for women)
- Family history
- Gender-related:
  - Men have greater risk until 64 y of age
  - Women have greater risk at 65 y of age and later
- Hypercholesterolemia
- Overweight/obesity
- Poor diet habits, particularly if it includes too much salt, as well as limited intake of vegetables, fiber, fish fats, and potassium
- Sedentary lifestyle
- Use of tobacco and nicotine products (e.g., cigarettes, e-cigarettes) and exposure to secondhand smoke
- Stress
- Sleep apnea

Adapted from American Heart Association (AHA). (2019). Know your risk factors for high blood pressure. Retrieved on 10/17/2019 at: [www.heart.org/en/health-topics/high-blood-pressure/why-high-blood-pressure-is-a-silent-killer/know-your-risk-factors-for-high-blood-pressure](http://www.heart.org/en/health-topics/high-blood-pressure/why-high-blood-pressure-is-a-silent-killer/know-your-risk-factors-for-high-blood-pressure); Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115.

Hypertension is categorized as either primary hypertension or secondary hypertension. **Primary hypertension** (also called *essential hypertension*) is diagnosed when there is no identifiable cause (Alexander, 2019). Approximately 90% to 95% of adults with hypertension have primary hypertension.

**Secondary hypertension** is defined as high blood pressure from an identifiable underlying cause. Between 5% and 10% of all adults with hypertension have secondary hypertension. Screening for secondary hypertension is indicated for new-onset, poorly controlled hypertension, in hypertension resistant to treatment with three or more drugs, with hypertension of an abrupt onset, or in patients younger than 30 years of age. In addition, a new diagnosis of hypertension with associated excessive target organ damage, such as cerebral vascular disease, retinopathy, left ventricular hypertrophy (LVH), heart failure with preserved ejection fraction, coronary artery disease, CKD, or peripheral arterial disease, could suggest secondary hypertension. [Chart 27-2](#) displays some common underlying causes of secondary hypertension.

## Pathophysiology



Blood pressure is the product of cardiac output multiplied by peripheral resistance. Cardiac output is the product of the heart rate multiplied by the stroke volume. Each time the heart contracts, pressure is transferred from the contraction of the myocardium to the blood and then pressure is exerted by the blood as it flows through the blood vessels. Hypertension can result from increases in cardiac output, increases in peripheral resistance (constriction of the blood vessels), or both. Increases in cardiac output are often related to an expansion in vascular volume. Although no precise cause can be identified for most cases of hypertension, it is understood that hypertension is a multifactorial condition. Because hypertension can be a sign, it is most likely to have many causes, just as fever has many causes (Norris, 2019). For hypertension to occur there must be a change in one or more factors affecting peripheral resistance or cardiac output. In addition, there must also be a problem with the body's control systems that monitor or regulate pressure ([Fig. 27-1](#)).

### Chart 27-2

### Common Causes of Secondary Hypertension

Chronic kidney disease  
Coarctation of the aorta  
Cushing's syndrome  
Hyperaldosteronism (primary or secondary)  
Hyperparathyroidism  
Hypo- or hyperthyroidism  
Medication abuse (nonsteroidal anti-inflammatory drugs [NSAIDs]) or substance abuse disorder (alcohol, cocaine, amphetamines)  
Obstructive sleep apnea  
Pheochromocytoma  
Preeclampsia  
Polycystic kidney disease  
Prostatism  
Renal artery stenosis

Adapted from Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115.

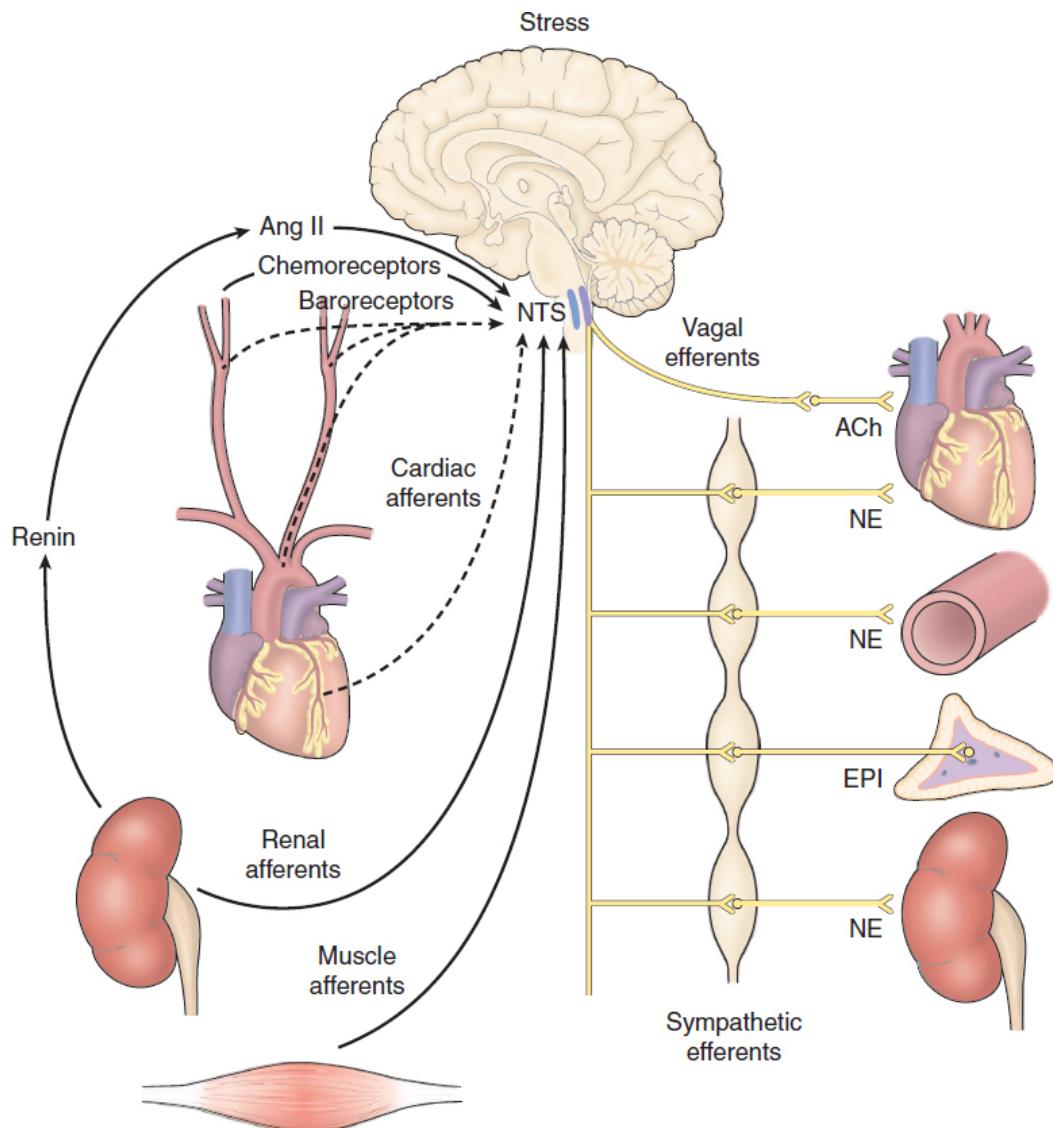
Hypertension is thought to occur as a result of a complex interaction between behavioral–social–environmental risks and genetics (Zilberman, Gaye, Berthon, et al., 2019). Behavioral–social–environmental risks may include dietary habits, including limited consumption of vegetables, fiber, fish fats, and potassium, and excessive intake of sodium; obesity; poor physical fitness; and excessive alcohol intake (Whelton et al., 2017).

Although single-gene mutations associated with hypertension have been identified, most types of hypertension are thought to be polygenic (i.e., mutations in more than one gene) (Whelton et al., 2017). The tendency to develop hypertension can be inherited; however, genetic profiles alone cannot predict who will and will not develop hypertension. The role of genetics in hypertension is complex and not fully understood at the present time.

To date, over 1000 genetic variants have been identified that may contribute to hypertension; however, collectively they explain only about 6% of the trait variance (Zilberman et al., 2019).

Many physiologic precedents that can lead to hypertension have been identified (Caillon, Mian, Fraulob-Aquino, et al., 2017; Caillon et al., 2019; Norris, 2019):

- Increased sympathetic nervous system activity related to dysfunction of the autonomic nervous system



**Figure 27-1 •** Central and reflex mechanisms involved in the neural control of blood pressure. Dotted arrows represent inhibitory neural influences, and solid arrows represent excitatory neural influences on sympathetic outflow. ACH, acetylcholine; Ang II, angiotensin II; EPI, epinephrine; NE, norepinephrine; NTS, nucleus tractus solitarius. Adapted from Kaplan, N. M., & Victor, R. G. (2015). *Kaplan's clinical hypertension* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

- Increased renal reabsorption of sodium, chloride, and water related to a genetic variation in the pathways by which the kidneys handle sodium
- Increased activity of the renin–angiotensin–aldosterone system, resulting in expansion of extracellular fluid volume and increased systemic vascular resistance

- Decreased vasodilation of the arterioles related to dysfunction of the vascular endothelium
- Resistance to insulin action, which may be a common factor linking hypertension, type 2 diabetes, hypertriglyceridemia, obesity, and glucose intolerance
- Activation of the innate and adaptive components of the immune response that contribute to vascular inflammation and dysfunction



## Gerontologic Considerations

Structural and functional changes in the heart, blood vessels, and kidneys contribute to increases in blood pressure that occur with aging. These changes include accumulation of atherosclerotic plaque, fragmentation of arterial elastins, increased collagen deposits, impaired vasodilation, and renal dysfunction. The result of these changes is decreased elasticity or stiffening of the major blood vessels, particularly the aorta, and volume expansion (Eliopoulos, 2018; Fajemiroye, da Cunha, Saavedra-Rodríguez, et al., 2018). Hence, both SBP and DBP increase linearly up to the sixth decade of life. At that time, among most adults, DBP gradually decreases while SBP continues to rise. Thus, **isolated systolic hypertension** is the predominant form of hypertension in older people (Whelton et al., 2017). Results from randomized controlled studies have demonstrated that lowering blood pressure in older adults with isolated systolic hypertension is effective in reducing the incidence of adverse cardiovascular events and death (Whelton et al., 2017).

## Clinical Manifestations

Physical examination may reveal no abnormalities other than elevated blood pressure. People with hypertension may be asymptomatic and remain so for many years. Hypertension is known as the “silent killer” because it typically has no warning signs or symptoms, and many people do not know they have it. However, when specific signs and symptoms appear, they usually indicate vascular damage, with specific manifestations related to the organs served by the involved vessels. These specific manifestations of pathophysiologic changes in various organs as a consequence of hypertension are referred to as **target organ damage**. Retinal changes such as hemorrhages, exudates (fluid accumulation), arteriolar narrowing, and cotton-wool spots (small infarctions) may occur. In severe hypertension, papilledema (swelling of the optic disc) may be seen (Weber & Kelley, 2018). Coronary artery disease with angina and myocardial infarction (MI) are common consequences of hypertension. LVH occurs in response to the increased workload placed on the ventricle as it contracts against higher systemic pressure. When heart damage is extensive, heart failure follows. Pathologic changes in the kidneys (indicated by

increased blood urea nitrogen [BUN] and serum creatinine levels) may manifest as nocturia. Cerebrovascular involvement may lead to a transient ischemic attack (TIA) or stroke, manifested by alterations in vision or speech, dizziness, weakness, a sudden fall, or transient or permanent hemiplegia (paralysis on one side). Cerebral infarctions account for most of the strokes in patients with hypertension (Norris, 2019).

## Assessment and Diagnostic Findings

The first step of diagnosis is an accurate blood pressure measurement (see [Chart 27-3](#) for an overview of appropriate BP measurement equipment, instructions, and interpretation for both the patient and the clinician). It is important to use an average of at least two blood pressure readings on at least two occasions to confirm the diagnosis of hypertension for most patients. The notable exception is when a patient's average BP is greater than or equal to 160/100 mm Hg, confirmed by at least two accurate readings on one occasion (see later discussion) (Muntner, Shimbo, Carey, et al., 2019; Whelton et al., 2017).

Blood pressure measurement within a clinical setting is often not an accurate reading; therefore, home blood pressure measurement (HBPM) or ambulatory blood pressure measurement (ABPM) are considered more accurate reflections of the blood pressure status. HBPM and ABPM are used not only to confirm the diagnosis of hypertension in most cases, but also to evaluate whether success has been achieved with treatments, such as lifestyle modifications and prescription medications (see later discussion) (Whelton et al., 2017).

Utilizing HBPM and ABPM measurements have led to recognizing other manifestations of blood pressure. Examples of these alternative manifestations of hypertension include masked hypertension and white coat hypertension. Patients with **masked hypertension** exhibit elevated blood pressure at levels typically consistent with hypertension in settings outside the hospital or clinic, while their blood pressure is seemingly normal in health care settings. In contrast, patients with **white coat hypertension** have blood pressure readings that would suggest a diagnosis of hypertension when they are in health care settings (e.g., clinics), but are within the normal ranges in other settings. If untreated, the patient with masked hypertension can go on to experience adverse cardiovascular events (e.g., MI, strokes) and mortality. On the other hand, the patient with white coat hypertension may receive treatment that is not warranted (Cohen, Lotito, Trivedi, et al., 2019).



## Measuring Blood Pressure

### Equipment

#### For the Patient at Home

- Automatic or semiautomatic upper-arm electronic device with digital display of readings

#### For the Practitioner

- Preferably, a validated electronic oscillometric device; if not available, a recently calibrated aneroid sphygmomanometer
- Appropriately sized arm cuff

### Instructions for the Patient

- Avoid eating, smoking, drinking caffeinated beverages, and physical activity for 30 min before blood pressure (BP) is measured.
- Empty bladder.
- Sit quietly for 5 min before the measurement.
- Sit comfortably, with back supported, with the forearm supported at heart level on a firm surface, with both feet on the ground; avoid talking while the measurement is being taken.

### Instructions for the Practitioner

- Select the size of the cuff based on the size of the patient. (The cuff size should have a bladder width of at least 40% of limb circumference and length 80–100% of limb circumference.) Small adult cuffs are 12 cm wide and 22 cm long, average adult cuffs are 16 cm wide and 30 cm long, large adult cuffs are 16 cm wide and 36 cm long, and extra-large adult cuffs are 16 cm wide and 42 cm long. Using a cuff that is too small will give a higher BP measurement, and using a cuff that is too large results in a lower BP measurement compared to one taken with a properly sized cuff.
- Wrap the cuff firmly around the arm. Center the cuff bladder directly over the brachial artery.
- Position the patient's arm at the level of the heart.
- If an aneroid sphygmomanometer is used, palpate the systolic pressure before auscultating. This technique helps to detect the presence of an auscultatory gap more readily.
- Ask the patient to sit quietly while the BP is measured, because the BP can increase when the patient is engaged in conversation.
- Initially, record BP results of both arms and take subsequent measurements from the arm with the higher BP. Normally, the BP should vary by no more than 5 mm Hg between arms.
- Take two readings 1–2 min apart and use the average of these measurements.

- Record the site where the BP was measured and the position of the patient (i.e., right arm).
- Inform the patient of their BP value and what it means. Emphasize the need for periodic reassessment, and encourage patients who measure BP at home to keep a written record of readings.

### Interpretation

Assessment is based on the average of at least two readings. (If two readings differ by more than 5 mm Hg, additional readings are taken and an average reading is calculated from the results.)

Adapted from Muntner, P., Shimbo, D., Carey, R. M., et al. (2019). Measurement of blood pressure in humans: A scientific statement from the American Heart Association. *Hypertension*, 73(5), e35–e66; Padwal, R., Campbell, N. R. C., Schutte, A. E., et al. (2019). Optimizing observer performance of clinic blood pressure measurement: A position statement from the Lancet Commission on Hypertension Group. *Journal of Hypertension*, 37(9), 1737–1745.

A thorough health history and physical examination are necessary to ensure successful diagnosis and treatment. The onset of high blood pressure and the patient's health history can be used to determine whether the patient might have primary hypertension or secondary hypertension (see [Chart 27-2](#)).

Abnormal findings from the physical examination could suggest either target organ damage or secondary hypertension. The physical examination should include palpation of all peripheral pulses. Absent, weak, or delayed femoral pulses could suggest coarctation of the aorta or severe peripheral vascular disease. The neck should be examined for carotid bruits, distended veins, or an enlarged thyroid gland. The upper abdomen should be auscultated for the presence of a renal artery bruit that could be suggestive of renal artery stenosis. A careful cardiac examination is also needed to evaluate for signs of LVH. LVH signs include displacement of the apex, a sustained and enlarged apical impulse, and the presence of an S<sub>4</sub> cardiac sound (see [Chapter 21](#)) (Weber & Kelley, 2018).

Occasionally, signs of hypertension can be discovered during a fundoscopic eye examination manifested as hypertensive retinopathy (e.g., retinal hemorrhages, microaneurysms, cotton-wool spots, papilledema); these findings are associated with an increased cardiovascular risk (e.g., stroke). Acute or chronic ocular changes can be the initial finding in asymptomatic patients and typically require a referral to an ophthalmologist. Long-standing, untreated hypertension can cause heart failure, CKD (elevated BUN and creatinine), and increased risk for cerebrovascular disease (e.g., TIAs, strokes) (Weber & Kelley, 2018).

Laboratory tests are also performed to assess for possible target organ damage and to screen for primary hypertension or secondary hypertension.

These typically include urinalysis, blood chemistry (i.e., analysis of sodium, potassium, creatinine, fasting glucose, cholesterol levels), and a 12-lead electrocardiogram. LVH can be assessed by echocardiography. Renal damage may be suggested by elevations in BUN and creatinine levels or by microalbuminuria or macroalbuminuria. Additional studies, such as creatinine clearance, renin level, urine tests, and 24-hour urine protein, may be performed. Optional testing may include uric acid and urine albumin to creatinine ratio (Whelton et al., 2017).

## Medical Management

The goal of hypertension treatment is to prevent complications (i.e., target organ damage) and death by maintaining a blood pressure lower than 130/80 mm Hg. Findings from a systematic review and meta-analysis demonstrated that hypertension treatment that effectively achieves the aim of BP control to normal levels is associated with lower mortality and lower rates of CVD (Brunstrom & Carlberg, 2018). The optimal treatment plan is one that is inexpensive, simple, and causes the least possible disruption in the patient's life.

The ACC/AHA Guidelines (Whelton et al., 2017) have developed a series of recommendations for prevention, treatment, and management of hypertension. In addition, these guidelines specify that a diagnosis of hypertension must be made based on accurate blood pressure measurements (see [Chart 27-3](#)). As noted previously, an average of at least two blood pressure readings on at least two occasions should be used to confirm the diagnosis of hypertension for most patients. After having the BP measured to screen for hypertension, a patient not previously diagnosed with hypertension and with a normal BP (i.e., SBP less than 120 mm Hg and DBP less than 80 mm Hg) can be advised to have the BP reevaluated in 1 year. A patient without a prior diagnosis of hypertension with an elevated BP (i.e., SBP 120 to 129 mm Hg and DBP less than 80 mm Hg) should be advised to follow up with additional BP readings within 3 to 6 months. A patient with a BP that could be consistent with hypertension; that is, with an SBP greater than or equal to 130 mm Hg or a DBP greater than or equal to 80 mm Hg should follow-up with additional BP readings within 1 month's time to either confirm or rule out the diagnosis (Muntner et al., 2019; Whelton et al., 2017). So that patients with suspected white coat hypertension or masked hypertension may be accurately diagnosed, blood pressure readings should be based on HBPM or ABPM. Patients not instructed to follow up with additional BP readings to confirm a diagnosis of hypertension are patients with average BP readings greater than or equal to 160/100 mm Hg on one occasion; these patients are diagnosed with hypertension and begin treatment with antihypertensive medications (Muntner et al., 2019; Whelton et al., 2017).

All patients who report lifestyle choices that may put them at risk for hypertension should be counseled to adopt lifestyle changes, as appropriate. These lifestyle changes could include weight loss, dietary changes, physical activity modifications, decreased alcohol consumption, and smoking cessation ([Table 27-2](#)). In particular, the *Dietary Approach to Stop Hypertension* (DASH) diet has been one of the most effective diets in lowering BP; if used in conjunction with weight loss, this diet can lower SBP by 11 to 16 mm Hg (Campbell, 2017) ([Table 27-3](#)). In addition to this dietary advice, patients should be counseled to incorporate a low sodium (less than 2 g/day), high potassium (3500 to 5000 mg/day) diet; this dietary combination is more effective than following either a lone low sodium or high potassium diet (Perez & Chang, 2014). A high potassium diet must be avoided in patients with CKD, however.

Patients suspected to have secondary hypertension must be accurately screened and the disorder that caused the high blood pressure must be properly treated in order to bring the patient's blood pressure into normal parameters (see [Chart 27-2](#)). The recommended treatment for patients with elevated blood pressure but who are not diagnosed with hypertension is lifestyle changes, not antihypertensive medications, with follow-up in 3 to 6 months, as noted previously, to not only reevaluate the blood pressure but to see if it has responded positively to lifestyle modifications (Whelton et al., 2017).

The primary provider is advised by the ACC/AHA Guidelines (Whelton et al., 2017) to screen the patient diagnosed with Stage 1 hypertension for risk of having adverse cardiac events (e.g., stroke, MI) within the next 10 years by using the online *ASCVD Risk Estimator Plus*. This tool is published by the ACC and is free to use (links to this tool are provided in the Resources section at the end of this chapter). This tool screens patients based on factors that include blood pressure readings, age, gender, lipid panel results, use of medications, smoking status, and whether or not they have concomitant diabetes. The risk of having an adverse cardiac event is then determined as low, borderline, or high. Those patients with a score of 10 or higher (consistent with mid-borderline risk) should be prescribed an antihypertensive medication, as should any patient diagnosed with stage 2 hypertension. All patients should be advised to institute relevant lifestyle changes, regardless of stage and use of antihypertensive medications.

**TABLE 27-2** Lifestyle Modifications to Prevent and Manage Hypertension<sup>a</sup>

Modification	Recommendation	Impact on SBP Reduction <sup>b</sup>	Impact on SBP Reduction <sup>b</sup>
		Patients without Hypertension	Patients with Hypertension
Weight reduction	Maintain normal body weight (body mass index 18.5–24.9 kg/m <sup>2</sup> ). Ideal body weight is best goal; but aim for at least 1 kg (2.2 lb) weight loss. Expect ~ 1 mm Hg SBP decrease per 1 kg reduction in weight.	-2–3 mm Hg	-5 mm Hg
Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat.	-3 mm Hg	-11 mm Hg
Dietary sodium reduction	Sodium <2 g/day is optimal goal; but aim for at least 1000 mg/day reduction. Check sodium amount on food labels.	-2–3 mm Hg	-5–6 mm Hg
Dietary potassium increase	Preferred potassium intake is 3500–5000 mg/day. Choose high potassium foods; check potassium amount on food labels.	-2 mm Hg	-4–5 mm Hg
Physical activity	Engage in: Regular aerobic physical activity such as brisk walking 90–150 min weekly Regular dynamic resistance training 90–150 min weekly Regular isometric resistance training at least three times weekly	-2–4 mm Hg -2 mm Hg -4 mm Hg	-5–8 mm Hg -4 mm Hg -5 mm Hg
Moderation of alcohol consumption	Limit consumption to ≤2 drinks (e.g., 24-oz beer, 10-oz wine, or 3-oz 80-proof whiskey) per day in most men and to ≤1 drink per day in women.	-3 mm Hg	-4 mm Hg

<sup>a</sup>For overall cardiovascular risk reduction, stop smoking.

<sup>b</sup>The effects of implementing these modifications are dose and time dependent and could be greater for some individuals.

DASH, dietary approaches to stop hypertension; SBP, systolic blood pressure.

Adapted from Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A

report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115.

## Pharmacologic Therapy

Research findings have demonstrated that appropriately prescribing antihypertensive pharmacologic agents lowers BP, and reduces the risk of CVD, cerebrovascular disease, and death (Whelton et al., 2017). Many classes of medications are available for hypertension management (Table 27-4). The medications that have been shown to prevent CVD are recommended as first-line agents for most patients. This first-line group includes thiazide or thiazide-type diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and calcium channel blockers (CCBs). African American patients with hypertension and without heart failure or CKD should be prescribed either a thiazide diuretic or a CCB as a first-line agent (not an ACE inhibitor or an ARB). The recommended first-line antihypertensive agents for patients with select comorbid disorders or who are pregnant are displayed in Table 27-5.

**TABLE 27-3** The DASH (Dietary Approaches to Stop Hypertension) Diet

Food Group	Number of Servings Daily
Grains and grain products	7 or 8
Vegetables	4 or 5
Fruits	4 or 5
Low-fat or fat-free dairy foods	2 or 3
Lean meat, fish, and poultry	≤2
Nuts, seeds, and dry beans	4 or 5 servings weekly

**Note:** The diet is based on 2000 calories/day.

Adapted from U.S. Department of Health and Human Services. (2003). Your guide to lowering your blood pressure with DASH: DASH eating plan. Retrieved on 9/27/2019 at: [www.nhlbi.nih.gov/health/public/heart/hbp/dash/new\\_dash/pdf](http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash/pdf)

Patients are first prescribed low doses of medication. If blood pressure does not fall to less than 130/80 mm Hg, the dose is increased gradually and additional medications are included as necessary to achieve control. The simplest treatment schedule possible is ideal as it promotes adherence to the regimen (e.g., one pill once each day, two or more agents combined into a single pill).

**Resistant hypertension** is diagnosed when a patient takes at least three antihypertensive medications from different classes (including a diuretic) and the blood pressure is still not controlled (i.e., not less than 130/80 mm Hg). A patient with controlled blood pressure but who requires at least four antihypertensive medications in order to maintain that control is also

considered to have resistant hypertension (Whelton et al., 2017). Risk factors for resistant hypertension include older age, being African American, and having obesity, CKD, or diabetes. Treatment of patients with suspected resistant hypertension first revolves around ensuring that they are indeed adhering to their prescribed medication regimen, including ensuring that their finances do not preclude them from purchasing their prescriptions, that they understand the purpose of the medications, and that medication side effects are tolerable. Patients with suspected resistant hypertension should also be evaluated for possible secondary hypertension (Whelton et al., 2017).

**TABLE 27-4** Oral Medication Therapy for Hypertension

Medications	Major Actions	Advantages and Contraindications	Effects and Nursing Considerations
<b>First-Line Antihypertensive Agents</b>			
<b>Thiazide or Thiazide-Type Diuretics</b>			
chlorthalidone <sup>a</sup> hydrochlorothiazide indapamide metolazone <i><sup>a</sup>preferred agent for its long half-life.</i>	<p>Decrease of blood volume, renal blood flow, and cardiac output.</p> <p>Depletion of extracellular fluid.</p> <p>Negative sodium balance (from natriuresis), mild hypokalemia.</p> <p>Directly affect vascular smooth muscle.</p>	<p>Relatively inexpensive.</p> <p>Effective orally.</p> <p>Effective during long-term administration.</p> <p>Mild side effects.</p> <p>Enhance other antihypertensive medications.</p> <p>Counter sodium retention effects of other antihypertensive medications.</p> <p><i>Contraindications:</i> Gout, known sensitivity to sulfonamide-derived medications, severely impaired kidney function, and history of hyponatremia.</p>	<p>Side effects include dry mouth, thirst, weakness, drowsiness, lethargy, muscle aches, muscular fatigue, tachycardia, GI disturbance.</p> <p>Orthostatic hypotension may be potentiated by alcohol, barbiturates, opioids, or hot weather.</p> <p>Because thiazides cause loss of sodium, potassium, and magnesium, and increase in uric acid and calcium, monitor for signs of electrolyte imbalance.</p> <p>Encourage intake of potassium-rich foods.</p> <p><i>Gerontologic considerations:</i> Risk of orthostatic hypotension.</p>
<b>ACE Inhibitors</b>			
benazepril captopril enalapril fosinopril lisinopril moexipril perindopril quinapril	<p>Inhibit conversion of angiotensin I to angiotensin II.</p> <p>Lower total peripheral resistance.</p>	<p>Angioedema is a rare but potentially life-threatening complication.</p> <p><i>Contraindications:</i> Concomitant use of an ARB or a renin inhibitor or a</p>	<p>Can cause hyperkalemia.</p> <p>Side effect can include cough.</p> <p><i>Gerontologic considerations:</i> Require reduced dosages and the</p>

ramipril trandolapril	potassium-sparing diuretic or potassium supplements; bilateral renal artery stenosis, pregnancy; history of angioedema with prior use of an ACE inhibitor.	addition of loop diuretics when there is renal dysfunction.  May cause upregulation of ACE2 receptors, making patients more susceptible to infection with SARS-CoV-2; however, may also mitigate deleterious effects of COVID-19.
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### Angiotensin Receptor Blockers

azilsartan candesartan eprosartan irbesartan losartan olmesartan telmisartan valsartan	Block the effects of angiotensin II at the receptor.  Reduce peripheral resistance.	Minimal side effects.  <i>Contraindications:</i> Concomitant use of an ACE inhibitor or a renin inhibitor or a potassium-sparing diuretic or potassium supplements; bilateral renal artery stenosis; history of angioedema with prior use of an ARB; pregnancy, lactation, renovascular disease.	Monitor for hyperkalemia.  Can be prescribed for patients with a history of angioedema from ACE inhibitor; however, must wait 6 wks to take after ACE inhibitor stopped.  May cause upregulation of ACE2 receptors, making patients more susceptible to infection with SARS-CoV-2; however, may also mitigate deleterious effects of COVID-19.
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### Calcium Channel Blockers—Dihydropyridines

amlodipine felodipine isradipine nicardipine SR nifedipine LA nisoldipine	Inhibit calcium ion influx across membranes.  Vasodilatory effects on coronary arteries and	Rapid action.  Effective by oral or sublingual route.  No tendency to slow SA nodal activity or prolong AV node conduction.	Can cause pedal edema, which is more common in women.  Administer on empty stomach; recommend
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peripheral arterioles.	Useful drug in treating isolated systolic hypertension.	eating small, frequent meals if complaint of nausea.
Decrease cardiac work and energy consumption, increase delivery of oxygen to myocardium.	<i>Contraindication:</i> HFrEF (but can use amlodipine or felodipine, if necessary).	Use with caution in patients with diabetes. Muscle cramps, joint stiffness, sexual dysfunction may disappear if dose decreased. Report irregular heartbeat, constipation, shortness of breath, edema. May cause dizziness.

### Calcium Channel Blockers—Nondihydropyridines

diltiazem ER	Inhibit calcium ion influx.	Avoid concomitant dosing with beta-blockers.	Do not discontinue suddenly.
verapamil IR			Observe for hypotension.
verapamil SR	Reduce cardiac afterload.	<i>Contraindications:</i> HFrEF; sinus node dysfunction, AV block.	Report irregular heartbeat, dizziness, edema.
verapamil—delayed-onset ER	Slow velocity of conduction of cardiac impulse.		Instruct on regular dental care because of potential gingivitis. Metabolized via cytochrome p450 system; therefore, many potential drug interactions.

### Second-Line Antihypertensive Agents

#### Diuretics—Loop

bumetanide	Volume depletion.	Preferred diuretics for patients with symptomatic HF and for patients with moderate to severe CKD.	Risk of volume and electrolyte depletion; monitor for hypokalemia. <i>Gerontologic considerations:</i>
furosemide	Block reabsorption of sodium, chloride, and water in renal tubules.		
torsemide			

		<i>Contraindications:</i> Same as for thiazide diuretics.	Risk for orthostatic hypotension.
<b>Diuretics—Potassium-Sparing</b>			
amiloride triamterene	Block sodium reabsorption.  Act on distal tubule independently of aldosterone.	Not particularly effective antihypertensive drugs when prescribed as lone agents; can be effective when prescribed with a thiazide diuretic in patients with hypokalemia; causes potassium retention.  <i>Contraindications:</i> Significant CKD, severe hepatic disease, hyperkalemia.	Drowsiness, lethargy, headache.  Monitor for hyperkalemia if given with ACE inhibitor or ARB.  Diarrhea and other GI symptoms—administer medication after meals.
<b>Diuretics—Aldosterone Antagonists</b>			
eplerenone spironolactone	Competitive inhibitors of aldosterone binding.	Indicated for patients with primary aldosteronism and resistant hypertension.  <i>Contraindications:</i> Hyperkalemia and impaired renal function.	Drowsiness, lethargy, headache.  Monitor for hyperkalemia if given with ACE inhibitor or ARB.  Diarrhea and other GI symptoms—administer medication after meals.  Avoid the use of potassium supplements or salt substitutes.  Spironolactone may cause gynecomastia.
<b>Beta-Blockers—Cardioselective</b>			
atenolol betaxolol bisoprolol metoprolol tartrate metoprolol succinate	Selectively block the beta-1 adrenergic receptors of the sympathetic nervous system, slowing the heart rate and lowering	Not recommended as first-line antihypertensive agents unless the patient has HF or CAD.  Bisoprolol or metoprolol succinate preferred	Avoid sudden discontinuation.  Side effects may include insomnia, lassitude, weakness, fatigue and

	the blood pressure.	agents for patients with HFrEF. These agents are preferred over noncardioselective beta-blockers if patient has asthma, reactive airway disease, or COPD.	occasionally nausea, vomiting, and epigastric distress.
<i>Contraindications:</i> Heart block, symptomatic bradycardia.			
<b>Beta-Blockers—Cardioselective and Vasodilatory</b>			
nebivolol	Blocks beta-1 adrenergic receptors and induces nitric oxide vasodilation.	Similar to other beta-blockers with additional capacity for vasodilation.	Avoid sudden discontinuation. Side-effect profile similar to other beta-blockers.
<i>Contraindications:</i> Similar to beta-blockers but with greater risk of severe bradycardia, heart block, cardiogenic shock, decompensated cardiac failure, sinus node dysfunction.			
<b>Beta-Blockers—Noncardioselective</b>			
nadolol propranolol propranolol LA timolol	Nonselectively block the beta-adrenergic receptors of the sympathetic nervous system with intended effects of slowing the heart rate and lowering the blood pressure.	<i>Contraindications:</i> Asthma, reactive airway disease, COPD, heart block, symptomatic bradycardia.	Avoid sudden discontinuation. Side effects may include insomnia, lassitude, weakness, fatigue and occasionally nausea, vomiting, and epigastric distress.
<b>Beta-Blockers—Intrinsic Sympathomimetic Activity</b>			
acebutolol penbutolol pindolol	Block both beta-1 and beta-2 receptors. Also has antiarrhythmic activity by slowing	<i>Contraindications:</i> Avoid use in patients with HFrEF.	Avoid sudden discontinuation. Side-effect profile similar to other beta-blockers.

atrioventricular conduction.

### Beta-Blockers—Combined Alpha- and Beta-Receptor Blockers

carvedilol	Block alpha- and beta-adrenergic receptors.	Carvedilol is a preferred agent for patient with HFrEF.	Avoid sudden discontinuation.
carvedilol phosphate CR		<i>Contraindications:</i> Asthma, reactive airway disease, COPD, heart block, symptomatic bradycardia, cardiogenic shock, severe tachycardia.	Side-effect profile similar to other beta-blockers.
labetalol	Cause peripheral dilation and decrease peripheral vascular resistance.		

### Direct Renin Inhibitor

Aliskiren	Blocks the conversion of angiotensinogen to angiotensin I by inhibiting the activity of the enzyme renin.	Cannot be given in combination with ACE inhibitors or ARBs.  Very long acting.  Contraindicated in pregnancy.	Monitor for hyperkalemia, especially for patients with CKD, or patients taking potassium supplements.
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### Alpha-1 Blockers

doxazosin	Peripheral vasodilator	May be second-line agent in men with BPH.	Associated with orthostasis, especially in older adults.
prazosin			
terazosin	acting directly on the blood vessel; action similar to direct vasodilators.	<i>Contraindication:</i> CAD.	

### Central Alpha<sub>2</sub>-Agonists and Other Centrally Acting Drugs

clonidine	<u>clonidine:</u>	Generally last-line agents —sometimes can be effective when other medications fail to lower blood pressure.	Dry mouth, drowsiness, sedation, and occasional headaches and fatigue.
clonidine patch	Exact mode of action is not understood, but acts through the central nervous system, apparently through centrally mediated alpha-adrenergic stimulation in the brain, producing blood pressure reduction.	Methyldopa may be drug of choice during pregnancy.	Anorexia, malaise, and vomiting with mild disturbance of liver function have been reported.
guanfacine		<i>Contraindication:</i> Severe coronary artery disease.	
methyldopa			Rebound hypertension or hypertensive
	<u>guanfacine:</u>		

	<p>Stimulates central alpha<sub>2</sub>-adrenergic receptors.</p> <p><u>methyldopa:</u> Dopa decarboxylase inhibitor; displaces norepinephrine from storage sites.</p>		<p>crisis is relatively common with withdrawal of clonidine; medication dosage should be tapered down when discontinuing clonidine and BP monitored carefully.</p>
<b>Direct Vasodilators</b>			
hydralazine minoxidil	<p>Direct vasodilatory action on smooth muscle of blood vessels, causing decreased peripheral vascular resistance.</p>	<p>Typically used in combination with other medications (diuretics, beta-blockers). Used also in pregnancy-induced hypertension.</p> <p><i>Contraindications:</i> Angina or coronary disease, heart failure, hypersensitivity.</p>	<p>Sodium and fluid retention and reflex tachycardia are common effects; headache, flushing, and dyspnea may occur.</p> <p>Hydralazine may produce lupus erythematosus-like syndrome.</p> <p>Minoxidil may cause hirsutism.</p>

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; AV, atrioventricular; BP, blood pressure; BPH, benign prostatic hyperplasia; CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; COVID-19, coronavirus disease 2019; CR, controlled release; ER, extended release; GI, gastrointestinal; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; IR, intermediate release; LA, long acting; SA, sinoatrial; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SR, sustained release.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Guo, J., Huang, Z., Lin, L., et al. (2020). Coronavirus disease 2019 (COVID-19) and cardiovascular disease: A viewpoint on the potential influence of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers on onset and severity of severe acute respiratory syndrome coronavirus 2 infection. *Journal of the American Heart Association*, 9, e016219. doi:10.1161/JAHA.120.016219; Sommerstein, R., Kochen, M. M., Messerli, F. H., et al. (2020). Coronavirus disease 2019 (COVID-19): Do angiotensin-converting enzyme inhibitors/angiotensin receptor blockers have a biphasic effect? *Journal of the American Heart Association*, 9, e016509. doi:10.1161/JAHA.120.016509; Vaduganathan, M., Vardeny, O., Michel, T., et al. (2020). Renin-angiotensin-aldosterone system inhibitors in patients with COVID-19. *The New England Journal of Medicine*, 382(17), 1653–1659; Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American

Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115.

**TABLE 27-5** Oral Antihypertensive Medications for Patients with Select Comorbid Diseases or Who Are Pregnant

Comorbid Disease or Special Patient Group	First-Line Antihypertensive Agents	Second-Line Antihypertensive Agents Comments
Stable coronary artery disease (e.g., myocardial infarction, angina) without heart failure	Beta-blockers, specifically carvedilol, metoprolol tartrate, metoprolol succinate, nadolol, bisoprolol, propranolol, or timolol; or ACE inhibitors; or ARBs.	Dihydropyridine calcium channel blockers may be prescribed if BP goal is not met and patient has continued angina.  Dihydropyridine calcium channel blockers, thiazide diuretics, or aldosterone receptor antagonist diuretics may be prescribed if BP goal is not met.
Heart failure with reduced ejection fraction (HF <sub>r</sub> EF)	ACE inhibitors; ARBs; angiotensin receptor-neprilysin inhibitor (i.e., sacubitril-valsartan); aldosterone receptor antagonist diuretics; other diuretics; or beta-blockers, specifically carvedilol, metoprolol succinate, or bisoprolol.	Nondihydropyridine calcium channel blockers are NOT recommended.  ACE inhibitors and ARBs should not be prescribed concomitantly.
Heart failure with preserved ejection fraction (HF <sub>p</sub> EF)	Diuretics should be prescribed to control hypertension when volume overload is present.	Nondihydropyridine calcium channel blockers are NOT recommended.
Chronic kidney disease (CKD) or kidney transplant	ACE inhibitors.	ARBs may be prescribed if patient is intolerant of ACE inhibitors.  ACE inhibitors and ARBs should not be prescribed concomitantly.
Diabetes	Diuretics, calcium channel blockers, ACE inhibitors, or ARBs.	ACE inhibitors or ARBs are preferred if albuminuria is present.
History of atrial fibrillation	ARBs can prevent recurrence of atrial fibrillation and achieve BP control.	
Metabolic syndrome	The best first-line antihypertensive agent is not clear.	Caution with prescriptions of thiazide diuretics because they are associated with increased insulin resistance, dyslipidemia, increased uric

		acid and progression to diabetes.
Pregnancy	Stop ACE inhibitors, ARBs, and/or direct renin inhibitors due to teratogenic effects. Transition to methyldopa, nifedipine, and/or labetalol.	Beta-blockers or calcium channel blockers appear superior to other options for preventing preeclampsia.

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure.

Adapted from DePalma, S. M., Himmelfarb, C. D., MacLaughlin, E. J., et al. (2018). Hypertension guideline update: A new guideline for a new era. *Journal of the American Academy of Physician Assistants*, 31(6), 16–22; Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*, 71(6), e13–e115.



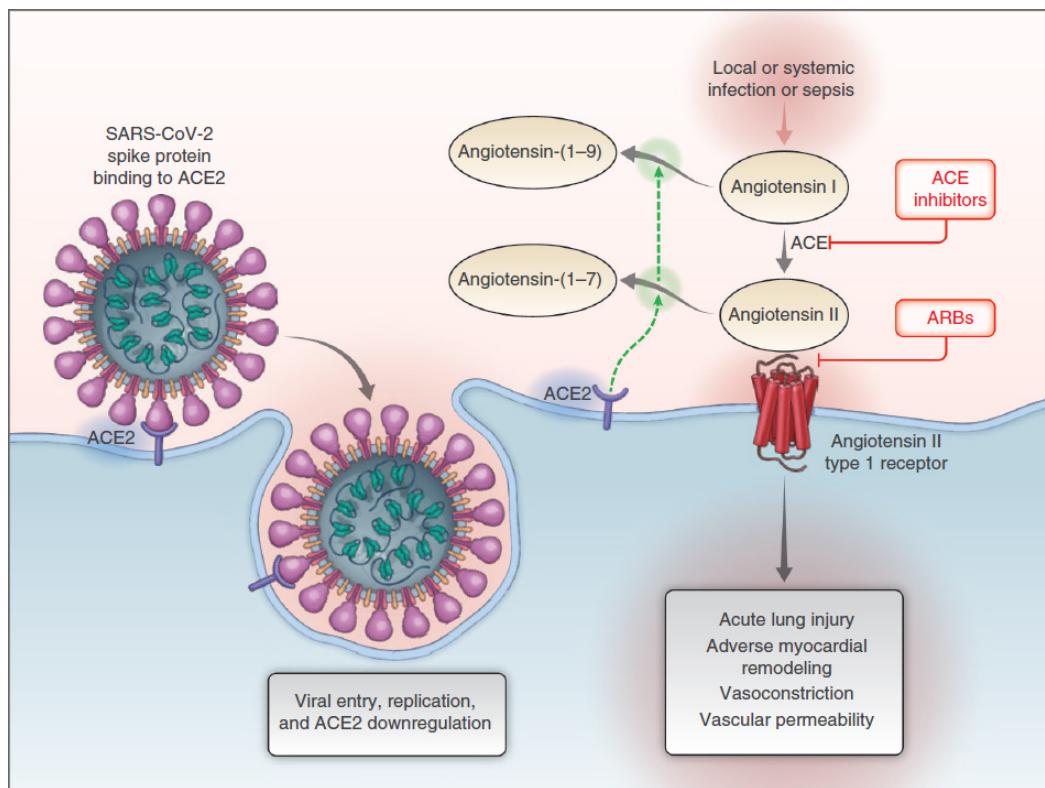
## COVID-19 Considerations

The coronavirus disease 2019 (COVID-19) pandemic began in Wuhan, China, in late 2019. Since that time, several risks for both severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and pathogenesis to coronavirus disease (COVID-19) have been posed. Epidemiologic findings from early data in China suggest that having a history of hypertension could be an important risk factor for becoming infected with SARS-CoV-2 as well as for being hospitalized to manage COVID-19 (Guo, Huang, Lin, et al., 2020; Sommerstein, Kochen, Messerli, et al., 2020; Vaduganathan, Vardeny, Michel, et al., 2020; Yang, Tan, Zhou, et al., 2020).

Because it is a virus, the SARS-CoV-2 pathogen must replicate within host cells. SARS-CoV-2 gains entry into host cells through the ACE2 cellular surface receptors, which are key to regulating the renin–angiotensin–aldosterone system (Fig. 27-2). ACE2 converts angiotensin II to angiotensin 1–7. ACE2 receptors are particularly abundant in type II alveolar cells (also called type II pneumocytes), vascular endothelial cells, and central nervous system tissue cells.

Patients who take ACE inhibitors or ARBs to manage hypertension tend to have an upregulation of ACE2 receptors (i.e., an increase in numbers of ACE2 receptors). Therefore, it is hypothesized that having an abundance of ACE2 receptors makes individuals with hypertension who take these medications more susceptible to SARS-CoV-2 infection; this hypothesis seems to explain the prevalence of patients with hypertension who also have COVID-19. However, it has also been found that, once infected with SARS-CoV-2, host cells will then downregulate ACE2 receptors, interfering with conversion of angiotensin II to angiotensin 1–7 (Vaduganathan et al., 2020). Angiotensin II

and angiotensin 1–7 have opposing effects. While angiotensin II causes vasoconstriction, high blood pressure, thrombosis, fibrosis, and inflammation, angiotensin 1–7 causes vasodilation, lower blood pressure, antithrombosis, and anti-apoptosis, and has anti-inflammatory effects (Guo et al., 2020). Thus, once infected with SARS-CoV-2, patients who take ACE inhibitors or ARBs might have a protective advantage over patients who do not take these medications, as patients who take ACE inhibitors or ARBs theoretically should have more ACE2 receptors (Sommerstein et al., 2020).



**Figure 27-2 •** Interaction between SARS-CoV-2 and the renin-angiotensin-aldosterone system. Shown is the initial entry of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) into cells, primarily type II pneumocytes, after binding to its functional receptor, angiotensin-converting enzyme 2 (ACE2). After endocytosis of the viral complex, surface ACE2 is further downregulated, resulting in unopposed angiotensin II accumulation. Local activation of the renin-angiotensin-aldosterone system may mediate lung injury responses to viral insults. ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker. Reprinted with permission from Vaduganathan, M., Vardeny, O., Michel, T., et al. (2020). Renin-angiotensin-aldosterone system inhibitors in patients with COVID-19. *The New England Journal of Medicine*, 382(17), 1653–1659.

Findings from a retrospective, single center study of patients hospitalized with COVID-19 in Wuhan, China, support the hypothesis that the use of ACE inhibitors or ARBs has a protective effect on patients with hypertension. In particular, patients with COVID-19 and hypertension who took these medications had significantly lower concentrations of C-reactive protein and procalcitonin (i.e., both are measures of inflammation) than patients with COVID-19 and hypertension who did not take these medications. In addition, fewer patients who took ACE inhibitors or ARBs were critically ill, and they had a lower mortality rate (Yang et al., 2020). Data to date supports continued use of both ACE inhibitors and ARBs in patients with hypertension during the COVID-19 pandemic (Guo et al., 2020; Sommerstein et al., 2020; Vaduganathan et al., 2020).

## Gerontologic Considerations

The target blood pressure for all adults with hypertension is less than 130/80 mm Hg, regardless of age, including older adults (Whelton et al., 2017). There is a caveat, however, that community-dwelling, ambulatory older adults should be carefully monitored for adverse effects of prescribed antihypertensive medications, which can include falls, orthostatic hypotension, and reduced renal function. The usual adage for older adults commencing antihypertensive medications is to *start low and go slow*; that is, the medication regimen starts with a low medication dosage that is slowly increased over time as needed. For older adults with multiple comorbidities and limited life expectancy, advanced cognitive impairment, or who have had frequent falls, a less aggressive blood pressure target may be reasonable based on clinical judgment and patient preference (DePalma et al., 2018). Nonetheless, the *American College of Physicians (ACP)* and *American Academy of Family Physicians (AAFP)* recommend antihypertensive pharmacologic treatment of patients 60 years and older with a history of having had a stroke or a TIA to target SBP less than 140 mm Hg to reduce the risk of recurrent stroke (Qaseem, Wilt, Rich, et al., 2017).

## NURSING PROCESS

### The Patient with Hypertension



#### Assessment

Nurse-led hypertension management has demonstrated greater rates of blood pressure control than when nurses are not leading these management efforts (Himmelfarb, Commodore-Mensah, & Hill, 2016). In many health care settings, nurses provide the BP assessment, the education, and the counseling while actively engaging the patient to promote adherence to the treatment plan (Himmelfarb et al., 2016). Assessment includes BP assessment using the equipment, instructions, and interpretive guidelines displayed in [Chart 27-3](#), and reviewing the patient's ambulatory or home blood pressure measurement technique and verifying its accuracy.

A complete history is obtained to assess for other cardiovascular risk factors and for signs and symptoms that indicate target organ damage (i.e., whether specific tissues are damaged by the elevated blood pressure). Manifestations of target organ damage may include angina; shortness of breath; alterations in speech, vision, or balance; nosebleeds; headaches; dizziness; or nocturia. The patient's partner may be helpful in identifying whether the patient may be experiencing obstructive sleep apnea (OSA), if the patient does not report being diagnosed or treated for OSA.

During the physical examination, the nurse must also pay specific attention to the rate, rhythm, and character of the apical and peripheral pulses to detect the effects of hypertension on the heart and blood vessels. A thorough assessment can yield valuable information about the extent to which the hypertension has affected the body and any other personal, social, or financial factors. For example, a patient's ability to adhere to an antihypertensive medication regimen may be influenced by the patient's financial resources to buy the medication and also by limited health insurance. Findings from research suggest that health beliefs, the presence of depressive symptoms, social support, and the presence of concomitant comorbidities might be associated with adherence to antihypertensive medication prescriptions (Spikes et al., 2019) (see the Nursing Research Profile in [Chart 27-4](#)).

#### Diagnosis

##### NURSING DIAGNOSES

Based on the assessment data, nursing diagnoses may include the following:

- Lack of knowledge regarding the relation between the treatment regimen and control of the disease process

- Impaired ability to manage regime as evidenced by difficulty adhering
  - to prescribed regimen (e.g., lifestyle changes, antihypertensive medication prescriptions)

#### **COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**

Potential complications may include the following:

- Left ventricular hypertrophy
- Myocardial infarction

**Chart 27-4**



#### **NURSING RESEARCH PROFILE**

## Medication Adherence among African Americans with Hypertension

Spikes, T., Higgins, M., Quyyumi, A., et al. (2019). The relationship among health beliefs, depressive symptoms, medication adherence, and social support in African Americans with hypertension. *Journal of Cardiovascular Nursing*, 34(1), 44–51.

### Purpose

African American adults are disproportionately affected by hypertension. Not only is the prevalence of hypertension greater among African Americans than among Americans of other ethnic groups, African Americans tend to become hypertensive at earlier ages. They also tend to be at higher risk of having complications from hypertension earlier in life, including cardiovascular diseases such as strokes and heart failure, and chronic kidney disease. Evidence from previous research suggests that many African Americans hold different beliefs about the causes and consequences of hypertension than do patients with hypertension from other ethnic groups. These health beliefs are thought to affect adherence to prescribed antihypertensive medications. Furthermore, previous research suggests that depressive symptoms and social support may also affect adherence to prescribed medications. Therefore, the purpose of this study was to find associations between hypertension beliefs, depressive symptoms, social support, and medication adherence among African Americans with hypertension.

### Design

This was a cross-sectional study that sampled African Americans with metabolic syndrome and hypertension managed with prescription antihypertensive medications ( $N = 120$ ). Participants completed a series of surveys, including the medication taking subscale of the Hill-Bone Compliance to High Blood Pressure Therapy Scale, three select subscales of the Beliefs related to High Blood Pressure in African Americans Scale, the Beck Depression Index, and the Enhancing Recovery in Coronary Heart Disease Social Support Inventory.

### Findings

Participants were mostly female (77%), with a mean age of 49.9 ( $\pm 8.6$ ) y. Slightly more than half of participants (54%) reported another comorbid condition in addition to having hypertension and metabolic syndrome. Approximately 37.5% of participants ( $n = 45$ ) were deemed nonadherent to their prescribed antihypertensive regimen. Beliefs about high blood pressure, depressive symptoms, and social support were not found to correlate with antihypertensive medication adherence. However, participants with other comorbidities were found to have 2.63 times greater odds of adhering to their prescribed antihypertensive medications.

### Nursing Implications

Findings from this study suggest that African Americans with hypertension may be more likely to adhere to their prescribed therapy if they have other comorbidities. The role of comorbidities among patients with hypertension needs to be further explored to determine how and why it might affect medication adherence. Nurses who manage the care of patients with hypertension should be cognizant that those without comorbidities might be at greater risk for nonadherence than those with multiple concomitant health disorders.

- Heart failure
- Cerebrovascular disease (TIA or stroke)
- Chronic kidney disease/end-stage renal disease
- Retinal hemorrhage

### Planning and Goals

The major goals for the patient include understanding of the disease process and its treatment, participation in a self-care health management program, and absence of complications.

### Nursing Interventions

The objective of nursing care for patients with hypertension focuses on lowering and controlling the blood pressure without adverse effects or undue cost. To achieve these goals, the nurse's role is to support and educate the patient about the treatment regimen, including making lifestyle changes, taking medications as prescribed, and scheduling regular follow-up appointments with the patient's primary provider to monitor progress or identify and treat any complications of disease or therapy.

#### INCREASING KNOWLEDGE

The patient needs to understand the disease process and how lifestyle changes and medications can control hypertension. The nurse needs to emphasize the concept of controlling hypertension rather than curing it. The nurse can encourage the patient to consult a dietitian to help develop a plan for improving nutrient intake or for weight loss. Explaining that it takes 2–3 mo for the taste buds to adapt to changes in salt intake may help the patient adjust to reduced salt intake and consider herbs and seasonings that add flavor without adding salt. The patient should be advised to limit alcohol intake, and tobacco and nicotine should be avoided because anyone with high blood pressure is already at risk for heart disease, and use of cigarettes and electronic nicotine delivery systems (ENDS) including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars, amplifies this risk.

#### PROMOTING EFFECTIVE HEALTH MANAGEMENT

Deviating from the therapeutic health management program is a significant problem for people with hypertension and other chronic conditions requiring lifetime management. Blood pressure control is achieved by only 54% of patients (Whelton et al., 2017). Rates of blood pressure control are lowest among Mexican American men, at only 39% overall (Himmelfarb et al., 2016). Effective health management is more likely, however, when patients actively participate in self-care, including self-monitoring of blood pressure and diet, possibly because patients receive immediate feedback and have a greater sense of control. Nurse-led wellness programs that are tailored to the patients' behaviors and eating and exercise practices are more effective than generic programs. Patients with hypertension must make considerable effort to adhere to recommended lifestyle modifications (see [Table 27-2](#)) and to take regularly prescribed medications. The effort needed to follow the therapeutic plan may seem unreasonable to some, particularly when they have no symptoms without medications but do have side effects with medications. Continued education and encouragement are usually needed to enable patients to formulate an acceptable plan that helps them live with their hypertension and adhere to the treatment plans. Compromises may have to be made about some aspects of therapy to achieve higher-priority goals.

The nurse can assist with behavior change by supporting patients in making small changes with each visit that moves them toward their goals. Another important factor is following up at each visit to see how the patient has progressed with the plans made at the prior visit. If the patient has had difficulty with the plan, the patient and nurse should work together to develop an alternative or modification that the patient believes will be more successful. Support groups for weight control, smoking cessation, and stress reduction may be beneficial for some patients; others can benefit from the support of family and friends. The nurse assists the patient to develop and adhere to an appropriate exercise regimen, because regular activity is a significant factor in reducing blood pressure (Himmelfarb et al., 2016).

#### **PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE**

If asked to participate in a blood pressure screening program, the nurse should ensure that proper blood pressure measurement technique is being used (see [Chart 27-3](#)), that validated electronic devices are used or, if aneroid sphygmomanometers are used, that they are properly calibrated, and that provision has been made to provide follow-up for any person identified as having an elevated blood pressure level. Adequate time should also be allowed to educate each person screened about what the blood pressure numbers mean. Each person should be given a written record of their blood pressure at the screening.



### Educating Patients About Self-Care.

The therapeutic regimen is the responsibility of the patient in collaboration with the primary provider. The nurse can help the patient achieve blood pressure control through education about managing blood pressure (see earlier discussion), setting goal blood pressures, and providing assistance with social support. Involving family members in education programs enables them to support the patient's efforts to control hypertension. The American Heart Association and the National Heart, Lung, and Blood Institute both provide printed and electronic patient education materials (see Resources section).

Providing written information about the expected effects and side effects of medications is important. When side effects occur, patients need to understand the importance of reporting them and to whom they should be reported. Patients need to be informed that **rebound hypertension** can occur. This phenomenon is characterized by a pathologically high blood pressure exhibited by patients who suddenly stop taking prescribed antihypertensive medications. Thus, patients should be advised to have an adequate supply of medication, particularly when traveling and in case of emergencies such as natural disasters. If traveling by airplane, patients should pack the medication in their carry-on luggage. All patients should be informed that some medications, such as beta-blockers, might cause sexual dysfunction and that other medications are available if problems with sexual function or satisfaction occur. The nurse can encourage and educate patients to measure their blood pressure at home. This practice involves patients in their own care and emphasizes that failing to take medications may result in an identifiable rise in blood pressure. Patients need to know that blood pressure varies continuously and that the range within which their pressure varies should be monitored.



### Gerontologic Considerations.

Adherence to the therapeutic program may be more difficult for the older adult. The medication regimen may be difficult to remember, and the expense can be a challenge. Simplification of the medication regimen to treatment with a single antihypertensive medication, if possible, is helpful. Many older adults are taking other prescription and over-the-counter medications, and verifying that there are no medication interactions is important. As noted previously, community-dwelling, ambulatory older adults should be carefully monitored for adverse effects of prescribed antihypertensive medications, which can include falls, orthostatic hypotension, and reduced renal function (e.g., the nurse should assess patient's urinary output and weight for changes from baseline). Special care must be taken to ensure the older patient understands the medication regimen, can see and read the instructions, open the medication container, and can get the prescription

refilled. The older adult's family or caregivers should be included in the educational program so that they understand the patient's needs, can encourage adherence to the treatment plan, and know when and whom to call if problems arise or further information is needed.

**Continuing and Transitional Care.** Regular follow-up care is imperative so that blood pressure control can be achieved. A history and physical examination should be completed at each clinic visit. The history should include all data pertaining to any potential problem, specifically medication-related problems such as orthostatic hypotension (experienced as dizziness or lightheadedness on standing). The patient should also bring a home blood pressure log and their own home blood pressure machine to verify accuracy and technique of home blood pressure monitor use. Patients and caregivers should also be given updated information at each visit on blood pressure medication, side effects, and important side effects to report immediately, such as low blood pressure or orthostatic hypotension. Low blood pressure readings can be due to impaired cardiovascular reflexes brought on by diuretics and other medication interactions.



#### **Quality and Safety Nursing Alert**

*The patient and caregivers should be cautioned that antihypertensive medications might cause hypotension. Low blood pressure or orthostatic hypotension should be reported immediately. Older adults have impaired cardiovascular reflexes and thus are more sensitive to the extracellular volume depletion caused by diuretics and to the sympathetic inhibition caused by adrenergic antagonists. The nurse educates patients to change positions slowly when moving from a lying or sitting position to a standing position. The nurse also counsels older adult patients to use supportive devices such as hand rails and walkers as necessary to prevent falls that could result from dizziness.*

#### **MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

Target organ damage is a potential adverse effect of long standing or poorly controlled hypertension. When the patient returns for follow-up care, all body systems must be assessed to detect any evidence of vascular damage. An eye examination with an ophthalmoscope is particularly important because retinal blood vessel damage indicates similar damage elsewhere in the vascular system. The patient is questioned about blurred vision, spots in front of the eyes, and diminished visual acuity. The heart, nervous system, and kidneys are also carefully assessed. Any significant findings are promptly reported to determine whether additional diagnostic studies are

required. Based on the findings, medications may be changed to improve blood pressure control.

### Evaluation

Expected patient outcomes may include:

1. Reports knowledge of disease management sufficient to maintain adequate tissue perfusion
  - a. Maintains blood pressure at less than 130/80 mm Hg with lifestyle modifications, medications, or both
  - b. Demonstrates no symptoms of angina, palpitations, or vision changes
  - c. Has stable BUN and serum creatinine levels
  - d. Has palpable peripheral pulses
2. Effectively manages health program
  - a. Adheres to the dietary regimen as prescribed: reduces calorie, sodium, and fat intake; increases fruit and vegetable intake
  - b. Exercises regularly
  - c. Takes medications as prescribed and reports any side effects
  - d. Measures blood pressure routinely
  - e. Abstains from tobacco, nicotine, and excessive alcohol intake
  - f. Keeps follow-up appointments
3. Has no complications
  - a. Reports no changes in vision
  - b. Exhibits no retinal damage on vision testing
  - c. Maintains pulse rate and rhythm and respiratory rate within normal ranges
  - d. Reports no dyspnea or edema
  - e. Maintains urine output consistent with intake
  - f. Has renal function test results within normal range
  - g. Demonstrates no motor, speech, or sensory deficits
  - h. Reports no headaches, dizziness, weakness, changes in gait, or falls

## Hypertensive Crises

Two classes of hypertensive crisis that require immediate intervention include hypertensive emergency and hypertensive urgency, which occur when the SBP exceeds 180 mm Hg or the DBP exceeds 120 mm Hg. Hypertensive emergencies and urgencies may occur in patients with secondary hypertension, and in those whose hypertension has been poorly controlled, whose hypertension has been undiagnosed, or in those who have abruptly

discontinued their medications (i.e., rebound hypertension). Once the hypertensive crisis has been managed, a complete evaluation is performed to review the patient's ongoing treatment plan, and strategies to prevent the occurrence of subsequent hypertensive crises are implemented (Whelton et al., 2017).



## Hypertensive Emergency

**Hypertensive emergency** is severe BP elevation (SBP greater than 180 mm Hg or DBP greater than 120 mm Hg) with new or worsening target organ damage. Some examples of target organ damage that may occur include hypertensive encephalopathy, ischemic stroke, MI, heart failure with pulmonary edema, dissecting aortic aneurysm, and renal failure. The 1-year mortality rate is more than 79% and median survival is 10.4 months if left untreated (Whelton et al., 2017). The patient needs to be admitted to the intensive care unit for continuous monitoring of BP and parenteral administration of an appropriate antihypertensive medication (Whelton et al., 2017).

A rapid and focused assessment is necessary to determine possible causes and target organ involvement. For patients with suspected aortic dissection, the management goal is to reduce the SBP to less than 120 mm Hg within the first hour of treatment (Fukui, 2018; Whelton et al., 2017). For those patients with suspected severe preeclampsia/eclampsia or pheochromocytoma crises, the management goal is to reduce the SBP to less than 140 mm Hg within the first hour of treatment (Lim, 2018; Whelton et al., 2017). The treatment management goal for other patients with hypertensive emergencies is to reduce the SBP by no more than 25% within the first hour of treatment, and then, if the patient is stable, to 160/100 mm Hg within the next 2 to 6 hours with an eventual goal of a normal, controlled blood pressure within 24 to 48 hours of when treatment commenced (Whelton et al., 2017). The antihypertensive medications of choice are those that have immediate onsets of action, and can include intravenous drugs such as nicardipine, clevidipine, labetalol, esmolol, nitroglycerin, and nitroprusside (Whelton et al., 2017). To date, there is a dearth of research findings that demonstrate the superiority of any antihypertensive medications in treating hypertensive emergencies (Whelton et al., 2017).



## Hypertensive Urgency

**Hypertensive urgency** is severe BP elevation (SBP greater than 180 mm Hg or DBP greater than 120 mm Hg) in stable patients without target organ damage as evidenced based on clinical examination and results of laboratory

studies. Many times, patients with a hypertensive urgency are nonadherent with antihypertensive therapy, resulting in rebound hypertension. The underlying reason for nonadherence should be explored (e.g., finances, anxiety, misunderstandings, miscommunication, drug side effects, or recreational drug use) and the team approach used and resources mobilized to prevent nonadherence from continuing or recurring. Restarting antihypertensive medication therapy or increasing dosages are indicated in treating these patients (Whelton et al., 2017).

Extremely close monitoring of the patient's blood pressure and cardiovascular status is required during treatment of hypertensive emergencies and urgencies (see [Chapter 21](#) for discussion of cardiovascular assessment). The exact frequency of monitoring is a matter of clinical judgment and varies with the patient's condition. Taking vital signs every 5 minutes is appropriate if the blood pressure is changing rapidly; taking vital signs at 15- or 30-minute intervals in a more stable situation may be sufficient. A precipitous drop in blood pressure can occur that would require immediate action to restore blood pressure to an acceptable level.

### CRITICAL THINKING EXERCISES

**1 pq** You are working as a nurse in a clinic that serves both an assisted living and a skilled nursing facility. One of your patients is an 80-year-old woman who is a new resident at the assisted living facility. When you take her blood pressure, you note that it is 110/70 mm Hg. While talking with her, you find that she reports episodes of dizziness and has fallen twice in the past 2 weeks. She tells you that she takes her medications as prescribed and has had no other concerns. What additional assessment data do you need to obtain? What is your priority plan of action?

**2 ipc** You are employed as an occupational health nurse in a manufacturing facility. A 32-year-old man who operates a forklift presents to be treated after injuring his forearm at work. During your assessment, he reports that he takes a beta-blocker for his hypertension and his blood pressure is 108/77 mm Hg. He also tells you that he is dedicated to staying healthy, has no underlying cardiac disease, and routinely exercises at the gym. He has noted that he cannot get his heart rate up no matter how long he works out and feels tired all the time. What further follow-up would you recommend and with whom?

**3 ebp** You are working in a clinic and a 50-year-old man presents for his annual examination. He takes no medications at present but reports that last year he was told that his blood pressure was elevated and that he needed to start taking an antihypertensive medication. He never filled that prescription because he had no symptoms and thought that if he had high blood pressure, he would feel bad. His blood pressure is 140/90 mm Hg and the rest of his examination is unremarkable. What education would you provide for him? What is the strength of the evidence that supports your education plan and follow-up with this patient?

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\*Asterisk indicates nursing research.

\*\*Double asterisk indicates classic reference.

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## Resources

- ACC ASCVD Risk Estimator Plus online application, [www.tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate](http://www.tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate)  
 American Heart Association, [www.heart.org](http://www.heart.org)
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[www.nhlbi.nih.gov/files/docs/public/heart/dash\\_brief.pdf](http://www.nhlbi.nih.gov/files/docs/public/heart/dash_brief.pdf)
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