A 45-year-old patient is brought to the emergency department (ED) complaining of pain in his left lower leg, which is swollen and painful. Ultrasound reports reveal that he has a deep vein thrombosis (DVT). He is admitted to a general medical/surgical unit for anticoagulation therapy. He is begun on a weight-based heparin intravenous (IV) drip, with concomitant oral doses of warfarin (Coumadin). When the IV heparin arrives on the unit, the nurse notes that the prescribed order dose does not match the dose on the IV bag sent from the pharmacy. The nurse sends the bag back to the pharmacy, requesting that the correct dose be sent as soon as possible. The patient is anxious to begin the heparin therapy and asks the nurse why there is a delay in starting the heparin drip.
QSEN Competency Focus: Safety

The complexities inherent in today’s health care system challenges 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 (QSEN, 2017; Cronenwett, Sherwood, Barnsteiner, et al., 2007) provides a framework for the knowledge, skills, and attitudes (KSA) 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.

Safety Definition: Minimize risk of harm to patients and providers through both system effectiveness and individual performance.

<table>
<thead>
<tr>
<th>SELECTED PRE-LICENSURE KSAs</th>
<th>APPLICATION AND REFLECTION</th>
</tr>
</thead>
</table>

**Knowledge**

Describe the factors that create a culture of safety (such as open communication strategies and organizational error reporting systems).

Identify how medication errors may occur in hospitals. Identify the different points during the process, from the time a medication is prescribed to the time it is given to a patient, that an error may occur. What system designs are in place in hospitals to prevent medication errors from occurring?

**Skills**

Communicate observations or concerns related to hazards and errors to patients, families, and the health care team.

How and to whom should the nurse report this type of “near miss” event? Although a medication error was averted, the patient’s treatment is delayed; describe how treatment delays such as these may also cause harm to patients. How should the nurse respond to the patient’s question regarding his treatment delay? Should the nurse disclose that the wrong dose was sent to the unit by the pharmacy? Should others be involved in disclosing this “near miss” event to the patient?

**Attitudes**

Value own role in preventing errors.

Reflect on how you would feel in this type of situation. Would you be pleased with yourself for discovering the error and inclined to blame the pharmacist? Or would you feel concerned that a flaw in the hospital’s system design allowed this type of “near miss” event to occur, and frightened that you could have administered the wrong dose of medication to this patient? How could you engage in system-design advocacy so that this type of safety-design flaw is rectified? How comfortable would you be in disclosing an error or potential error to a patient?

Glossary

- **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 140/90 mm Hg
- **hypotension**: a decrease in blood pressure to less than 100/60 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 S1 (atrioventricular valves) and S2 (semilunar valves)
- **opening snaps**: abnormal diastolic sound generated during opening of a rigid atriouventricular valve leaflet
- **postural (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

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

1. Describe the relationship between the anatomic structures and the physiologic function of the cardiovascular system.
2. Incorporate assessment of cardiac risk factors into the health history and physical assessment of the patient with cardiovascular disease.
3. Explain the proper techniques to perform a comprehensive cardiovascular assessment.
4. Discriminate between normal and abnormal assessment findings identified by inspection, palpation, percussion, and auscultation of the cardiovascular system.
5. Recognize and evaluate the major manifestations of cardiovascular dysfunction by applying concepts from the patient’s health history and physical assessment findings.
6. Discuss the clinical indications, patient preparation, and other related nursing implications for common tests and procedures used to assess cardiovascular function and diagnose cardiovascular diseases.
7. Compare the various methods of hemodynamic monitoring (e.g., central venous pressure, pulmonary artery pressure, and arterial pressure monitoring) with regard to indications for use, potential complications, and nursing responsibilities.
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 (see Fig. 25-1). 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.

More than 85.6 million Americans have one or more types of cardiovascular disease (CVD), including hypertension, coronary artery disease (CAD), heart failure (HF), stroke, and congenital cardiovascular defects (American Heart Association [AHA], 2016). Because of the 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.

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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 systole 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 25-1.

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 (Woods, Froelicher, Motzer, et al., 2009). Figure 25-2 identifies the pressures in each of these areas.

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, 2014).

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. 25-1).

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 valves 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. The heart has high metabolic requirements, extracting approximately 70% to 80% of the oxygen delivered (other organs extract, on average, 25%) (Woods et al., 2009). 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 Fig. 27-2 in Chapter 27).

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 (see Fig. 25-3). 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, 2014).

The electrical impulses initiated by the SA node are conducted along the myocardial cells of the atria via specialized tracts called inter nodal 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. 25-3). 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, 2014).

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) (Woods et al., 2009). 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.

As shown in Figure 25-4, 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 dysrhythmias. 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 dysrhythmias, 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 dysrhythmias. (These dysrhythmias and others are discussed in detail in Chapter 26.)

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. 25-2). 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 during diastole and systole. Each cardiac cycle has three major sequential events: diastole, atrial systole, and ventricular systole. The pressures caused by these events make blood 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.

Pressure in the atria is lower than in the ventricles because the pressure in the ventricles forces the pulmonic and aortic valves to open, and regurgitation (backflow) of blood into the atria is prevented. These events mark the onset of diastole, and the cardiac cycle is repeated.

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 (Woods et al., 2009).

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 significant elevations in BP (hypertension), 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). Less 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 cardiac muscle cells (sarcomeres), 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 [Lanoxin], 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 [Lopressor]). 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% (Woods et al., 2009). 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 29 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, 2014; Woods et al., 2009).

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, 2014; Woods et al., 2009). The structural and functional changes with aging and associated history and physical examination findings are summarized in Table 25-1.

**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 catheterization and angioplasty technically more difficult.

Women typically develop CAD 10 years later than men, as women have the benefit of the female hormone estrogen and its cardioprotective effects. 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) dilatation of the blood vessels, which enhance blood flow to the heart. As women reach menopause, around 50 years of age, estrogen levels slowly disappear and place women at higher risk for CAD. By age 45 years for men and 55 years of age for women, the risk for CAD is equivalent (McSweeney, Rosenfeld, Abel, et al., 2016).

In the past, hormone therapy (HT) was routinely prescribed for postmenopausal women with the belief that it would deter the onset and progression of CAD. However, the AHA no longer recommends its use due to strong evidence that HT is not a beneficial CAD preventative strategy for women (McSweeney et al., 2016). In fact, recent studies report that HT after menopause can be harmful to women by putting them at increased risk for stroke and venous thromboembolic events (Boardman, Hartley, Eisinga, et al., 2015).

**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. An acutely ill patient with CVD who is admitted to the emergency department (ED) or intensive care unit (ICU) requires a very different assessment than a person who is being examined for a chronic stable condition. Although the key components of the cardiovascular assessment remain the same, the assessment priorities vary according to the needs of the patient. For example, an 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 within minutes of arrival to the ED. The physical assessment is ongoing and concentrates on evaluating the patient for ACS complications, such as dysrhythmias and HF, and determining the effectiveness of medical treatment.

**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. This results in prolonged delays in seeking lifesaving treatment. Major barriers to seeking prompt medical care include lack of knowledge about the symptoms of heart disease, attributing symptoms to a benign source, denying symptom significance, and feeling embarrassed about having symptoms (Gillis, Arslanian-Engoren, & Struble, 2014). 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 dysrhythmias and conduction problems (see Chapter 26); CAD (see Chapter 27); structural, infectious, and inflammatory disorders of the heart (see Chapter 28); and complications of CVD such as HF and cardiogenic shock (see Chapters 29 and 14, respectively). 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, dysrhythmias, 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)

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**TABLE 25-1 Age-Related Changes of the Cardiac System**

<table>
<thead>
<tr>
<th>Cardiovascular Structure</th>
<th>Structural Changes</th>
<th>Functional Changes</th>
<th>History and Physical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atria</td>
<td>↑ Size of left atrium Thickening of the endocardium</td>
<td>↑ Atrial irritability</td>
<td>Irregular heart rhythm from atrial dysrhythmias</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>Endocardial fibrosis Myocardial thickening (hypertrophy) Infiltration of fat into myocardium</td>
<td>Left ventricle stiff and less compliant Progressive decline in cardiac output</td>
<td>Fatigue ↓ Exercise tolerance Signs and symptoms of heart failure or ventricular dysrhythmias Point of maximal impulse palpated lateral to the midclavicular line ↓ Intensity S, S split S, S may be present</td>
</tr>
<tr>
<td>Valves</td>
<td>Thickening and rigidity of AV valves Calcification of aortic valve</td>
<td>Abnormal blood flow across valves during cardiac cycle</td>
<td>Murmurs may be present Thrill may be palpated if significant murmur is present</td>
</tr>
<tr>
<td>Conduction system</td>
<td>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</td>
<td>Slower SA node rate of impulse discharge Slowed conduction across AV node and ventricular conduction system</td>
<td>Bradycardia Heart block ECG changes consistent with slowed conduction (↑ PR interval, widened QRS complex)</td>
</tr>
<tr>
<td>Sympathetic nervous system</td>
<td>↓ Response to beta-adrenergic stimulation</td>
<td>↓ Adaptive response to exercise: contractility and heart rate slower to respond to exercise demands Heart rate takes more time to return to baseline</td>
<td>Fatigue ↓ Diminished exercise tolerance ↓ Ability to respond to stress</td>
</tr>
<tr>
<td>Aorta and arteries</td>
<td>Stiffening of vasculature ↓ Elasticity and widening of aorta Elongation of aorta, displacing the brachiocephalic artery upward</td>
<td>Left ventricular hypertrophy Progressive increase in systolic BP; slight ↑ in diastolic BP Widening pulse pressure Pulsation visible above right clavicle</td>
<td></td>
</tr>
<tr>
<td>Baroreceptor response</td>
<td>↓ Sensitivity of baroreceptors in the carotid artery and aorta to transient episodes of hypertension and hypotension</td>
<td>Baroreceptors unable to regulate heart rate and vascular tone, causing slow response to postural changes in body position</td>
<td>Postural BP changes and reports of feeling dizzy, fainting when moving from lying to sitting or standing position</td>
</tr>
</tbody>
</table>

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

• 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, dysrhythmias, hypotension, postural hypotension, vasovagal episode)

Symptoms of ACS can differ between men and women. Although chest pain or discomfort can occur in both men and women, it is more likely to be experienced by men. On the contrary, women can experience more atypical symptoms and women, it is more likely to be experienced by men. On the contrary, women can experience more atypical symptoms including fatigue, nausea, neck pain, right arm pain, jaw pain, dizziness, and syncope (McSweeney et al., 2016).

Chest Pain

Chest pain and chest discomfort are common symptoms that may be caused by a number of cardiac and noncardiac problems. Table 25-2 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 25-2.
• 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 myocardial infarction (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 dysrhythmias. Both HF and dysrhythmias can be complications of an acute MI. (See Chapter 27 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 CADs (see Chart 27-1 in Chapter 27) and any measures that they are taking to modify these risks. Some risk factors, such as increasing age, male sex, 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 (Goff, Lloyd-Jones, Bennett, et al., 2013). There is a tool available on the internet from the National Institute of Health (NIH) that is used to assess an individual’s 10 year risk for having an acute MI. It takes into consideration all of these risk factors (NIH, 2014) (see tool in Resources section at end of chapter).

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 (see Chart 25-1)?
• What are your risk factors for CAD (see Chart 27-1 in Chapter 27)?
• 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. On the contrary, patients who have this understanding may be more motivated to alter their lifestyle to avoid the risk of future cardiac events. The AHA has published lifestyle management guidelines that identify interventions and treatment goals for each of these risk factors (Eckel, Jakicic, Ard, et al., 2014). Chapter 27 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, herbs, 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?

Aspirin, a nonprescription medication, is an important antithrombotic therapy for secondary prevention in patients recovering from ACS (Amsterdam, Wenger, Brindis, et al., 2014). However, patients who are not aware of this benefit may be inclined to stop taking aspirin if they think it is a trivial medication. A careful medication history often uncovers common medication errors and causes for nonadherence 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
### TABLE 25-2 Assessing Chest Pain

<table>
<thead>
<tr>
<th>Location</th>
<th>Character</th>
<th>Duration</th>
<th>Precipitating Events and Aggravating Factors</th>
<th>Alleviating Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina Pectoris, ACS (unstable angina, MI)</td>
<td>Uncomfortable pressure, squeezing, or fullness in substernal chest area</td>
<td>Angina: 5–15 min</td>
<td>Angina: Physical exertion, emotional upset, eating large meal, or exposure to extremes in temperature</td>
<td>Angina: Rest, nitroglycerin, oxygen</td>
</tr>
<tr>
<td></td>
<td>Can radiate across chest to the medial aspect of one or both arms and hands, jaw, shoulders, upper back, or epigastrium</td>
<td>ACS: &gt;15 min</td>
<td>ACS: Emotional upset or unusual physical exertion occurring within 24 h of symptom onset Can occur at rest or while asleep</td>
<td>ACS: Morphine, reperfusion of coronary artery with thrombolytic (fibrinolytic) agent or percutaneous coronary intervention</td>
</tr>
<tr>
<td></td>
<td>Radiation to arms and hands, described as numbsness, tingling, or aching</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less common sites of pain with myocardial ischemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Sharp, severe substernal or epigastric pain</td>
<td>Intermittent</td>
<td>Sudden onset Pain increases with inspiration, swallowing, coughing, and rotation of trunk</td>
<td>Sitting upright, analgesia, anti-inflammatory medications</td>
</tr>
<tr>
<td></td>
<td>Can radiate to neck, arms, and back</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Associated symptoms include fever, malaise, dyspnea, cough, nausea, dizziness, and palpitations</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary Disorders (pneumonia, pulmonary embolism)</td>
<td>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</td>
<td>≥30 min</td>
<td>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)</td>
<td>Treatment of underlying cause</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued on page 682)
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 (assessment for obesity); 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

ACS, acute coronary syndrome; MI, myocardial infarction.
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 Chart 5-2: 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, and alcohol use)

**Genetics Resources**

American Heart Association, www.heart.org
Hypertrophic Cardiomyopathy Association, www.4hcm.org
Familial Hypercholesterolemia Foundation, www.thefhfoundation.org
Sudden Arrhythmia Death Syndromes, www.sads.org
See Chapter 8, Chart 8-7 for additional components of genetic counseling.

**Chart 25-1**

**GENETICS IN NURSING PRACTICE**

**Cardiovascular Disorders**

- 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

**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 platelet-inhibiting medications such as aspirin and clopidogrel (Plavix); platelet aggregation inhibitors such as abciximab (ReoPro), eptifibatide (Integrilin), and tirofiban (Aggrastat); anticoagulants, such as low–molecular-weight heparin (e.g., dalteparin [Fragmin], enoxaparin [Lovenox]), heparin, or oral anticoagulants such as warfarin (Coumadin), rivaroxaban (Xarelto), or apixaban (Eliquis).

**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 awakenings with shortness of breath, called paroxysmal nocturnal dyspnea, is an additional symptom of worsening HF. This nighttime symptom is caused by the realignment 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.

There are mounting evidence of the cardiac consequences associated with sleep-disordered breathing (SDB). Often also called obstructive sleep apnea (OSA), 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. Untreated SDB has been linked to CAD, hypertension, HF, and dysrhythmias. SDB is treated by the use of continuous positive airway pressure (CPAP) and mandibular advancement devices (MAD) (see Chapter 22). These devices maintain an open airway during sleep, preventing hypoxemia and resulting abnormal elevations in BP. The cardinal signs of SDB are loud disruptive snoring and apnea lasting 10 seconds or more. Obesity and large neck circumference are two important risk factors for SDB (Ayas, Owens, & Kheirandish-Gozal, 2015).

During the health history, the nurse assesses for SDB by asking patients at high risk if they snore loudly, have frequent bouts of awaking from sleep, awaken with a headache, or experience severe daytime sleepiness (hypersomnolence). For patients with a diagnosis of SDB, the nurse determines if the patient has been prescribed a CPAP or MAD 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 an individual’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 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. A recent Scientific Statement from the AHA highlights the importance of social support. Based on findings from recent studies, social support has been found to be closely linked to CVD outcomes. In fact, patients with lower levels of social support have an increased risk for cardiac-related mortality and stroke, compared with others having higher levels of support (Havranek, Mujahid, Barr, et al., 2015).

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. In men, erectile dysfunction may develop as a side effect of cardiac medications (e.g., beta-blockers); some men will stop taking their medication as a result. Few patients with sexual dysfunction or concerns about resuming sexual activity seek medical care (Steinke, Jaarsma, Barnason, et al., 2013). The nurse can help patients by initiating discussions about sexuality and encouraging them to discuss problems with their primary provider or cardiologist.

A common reason for decreasing sexual activity is due to concerns that people have about the effects of physical exertion on the heart. They are worried that sexual activity may cause another heart attack, sudden death, or untoward symptoms such as angina, dyspnea, or palpitations. Often couples lack adequate information about the physical demands related to sexual activity and ways in which these demands can be modified. The physiologic demands associated with sexual activity range between 3 and 5 metabolic equivalents (METs), which is similar to the METs expended during mild to moderate activity. Sharing this information may make patients and their partners more comfortable about resuming sexual activity (Steinke et al., 2013).
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.

Coping and Stress Tolerance

Anxiety, depression, and stress are known to influence both the development of and recovery from CAD and HF. High levels of anxiety are associated with an increased incidence of CAD and in-hospital complication rates after MI. Patients with a diagnosis of an acute MI and depression have an increased risk of rehospitalization, death, more frequent angina, more physical limitations, and poorer quality of life compared with patients without depression (Lichtman, Froelicher, Blumenthal, et al., 2014). Although the association between depression and CAD is not completely understood, both biologic factors (e.g., platelet abnormalities, inflammatory responses) and lifestyle factors may contribute to the development of CAD. Patients who are depressed are less motivated to adhere to recommended lifestyle changes and medical regimens necessary to prevent future cardiac events, such as an MI (Havranek et al., 2015; Lichtman et al., 2014).

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. A quick and simple screening tool recommended by the AHA is the two-question Patient Health Questionnaire (PHQ-2) (Bigger & Glassman, 2010). The nurse asks the patient the following:

Over the past 2 weeks, how often have you been bothered by either of the following problems?

• Little interest or pleasure in doing things
• Feeling down, depressed, or hopeless

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 score of 3 or higher may be experiencing major depression and should be referred to their primary providers for further evaluation and treatment.

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 distension, 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, underweight, or cachectic). The patient’s height and weight are measured to calculate BMI (weight in kilograms/square of the height in meters), as well as the waist circumference (see Chapter 5). These measures are used to determine if obesity (BMI greater than 30 kg/m²) and abdominal fat (males: waist greater than 40 in; females: waist greater than 35 in) 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 25-3 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 6 P's, are pain, pallor, 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 Fig. 29-2 in Chapter 29). 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 slight (1+) to very marked (4+) as described in Table 25-3. It is important that clinicians

### TABLE 25-3 Common Assessment Findings Associated With Cardiovascular Disease

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

PAD, peripheral arterial disease.  
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 30 for a complete description of these conditions) (Weber & Kelley, 2014).

### 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 BP in adults 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 by having a systolic BP that is consistently greater than 140 mm Hg or a diastolic BP greater than 90 mm Hg (Weber, Schiffrin, White, et al., 2014). Hypotension refers to an abnormally low systolic and diastolic BP that can result in lightheadedness or fainting. (See Chapter 31 for additional definitions, measurement, and management.)

### Pulse Pressure

The difference between the systolic and the diastolic pressures is called the pulse pressure. It is a reflection of stroke volume, ejection velocity, and systemic vascular resistance. Pulse pressure, which normally is 30 to 40 mm Hg, indicates how well the patient maintains cardiac output. The pulse pressure increases in conditions that elevate the stroke volume (anxiety, exercise, bradycardia), reduce systemic vascular resistance (fever), or reduce distensibility of the arteries (atherosclerosis, aging, hypertension). Decreased pulse pressure reflects reduced stroke volume and ejection velocity (shock, HF, hypovolemia, mitral regurgitation) or obstruction to blood flow during systole (mitral or aortic stenosis). A pulse pressure of less than 30 mm Hg signifies a serious reduction in cardiac output and requires further cardiovascular assessment (Woods et al., 2009).

### Postural (Orthostatic) Blood Pressure Changes

There is a gravitational redistribution of approximately 300 to 800 mL of blood into the lower extremities and the gastrointestinal system immediately upon standing. These changes reduce venous 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. These compensatory mechanisms stabilize arterial BP (Arnold & Shibao, 2013).

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.

**Postural (orthostatic) 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 (Arnold & Shibao, 2013).

Postural 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 antihypertensive 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 (see Chart 25-2).

The following is an example of BP and heart rate measurements in a patient with postural 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

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**Chart 25-2**

**Assessing Patients for Postural Hypotension**

The following steps are recommended when assessing patients for postural 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 min for a total of 10 minutes to rule out postural hypotension.
- Return the patient to a supine position if postural hypotension is detected or if the patient becomes symptomatic.
- Document heart rate and BP measured in each position (e.g., supine, sitting, and standing) and any signs or symptoms that accompany the postural changes.

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 frequently raises 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.

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 PML, for a full minute while simultaneously palpating the radial pulse. Any discrepancy between contractions heard and pulses felt is noted. Disturbances of rhythm (dysrhythmias) 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 dysrhythmias 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 26 for a detailed discussion of these dysrhythmias).

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, 2014).

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 30.)

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 Fig. 30-2 in Chapter 30). 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 (Woods et al., 2009). 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 who have normal blood volume (euvolemia), the jugular veins are normally visible in the supine position with the head of the bed elevated to 30° (Bickley, 2014). Obvious distention of the veins with the patient’s head elevated 45° to 90° 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 25-5 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

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 (see Fig. 25-6).

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 heart failure. 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 25-5, 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 S₁ and S₂, are produced by closure of the AV valves and the semilunar valves, respectively. The period between S₁ and S₂ corresponds with ventricular systole (see Fig. 25-7). When the heart rate is within the normal range, systole is much shorter than the period of diastole. However, as the heart rate increases, diastole shortens.

Normally, S₁ and S₂ are the only sounds heard during the cardiac cycle (Bickley, 2014).

**S₁—First Heart Sound**

Tricuspid and mitral valve closure creates the first heart sound (S₁). The word “lub” is used to replicate its sound. S₁ is usually heard the loudest at the apical area. S₁ is easily identifiable and serves as the point of reference for the remainder of the cardiac cycle.

The intensity of S₁ increases during tachycardias or with mitral stenosis. In these circumstances, the AV valves are wide open during ventricular contraction. The accentuated S₁ occurs as the AV valves close with greater force than normal. Similarly, dysrhythmias can vary the intensity of S₁ from beat to beat due to lack of synchronized atrial and ventricular contraction.

**S₂—Second Heart Sound**

Closure of the pulmonic and aortic valves produces the second heart sound (S₂), commonly referred to as the “dub” sound. The aortic component of S₂ is heard the loudest over the aortic and pulmonic areas. However, the pulmonic component of S₂ is a softer sound and is heard best over the pulmonic area.

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 S₂. Normal physiologic splitting of S₂ 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 S₂ 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 S₂ 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 S₃ or S₄ gallops, opening snaps, systolic clicks, and murmurs. S₃ and S₄ 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.

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**Figure 25-7 • Normal heart sounds. The first heart sound (S₁) is produced by closure of the mitral and tricuspid valves (“lub”). The second heart sound (S₂) is produced by closure of the aortic and pulmonic valves (“dub”). Arrows represent the direction of blood flow.**
Figure 25-8 • Gallop sounds. An S₃ (“DUB”) is an abnormal sound heard immediately following S₂ (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. S₄ (“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.

S₃—Third Heart Sound
An S₃ (“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 S₂. “Lub-dub-DUB” is used to imitate the abnormal sound of a beating heart when an S₃ 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 S₃ (see Fig. 25-8). In older adults, an S₃ 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 S₃ is heard over the tricuspid area with the patient in a supine position. A left-sided S₃ is best heard over the apical area with the patient in the left lateral position.

S₄—Fourth Heart Sound
S₄ (“LUB”) occurs late in diastole (see Fig. 25-8). S₄ is heard just before S₁, 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. S₄, 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 S₄, although less common, is heard best over the tricuspid area with the patient in a supine position. There are times when both S₃ and S₄ 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 S₂ to be mistaken for an opening S₂ and too early in diastole to be mistaken for an S₁. The high-pitched, snapping quality of the sound is another way to differentiate an opening snap from an S₃. 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 S₁. 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, and 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 (see Chart 25-3).

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.
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 clinician.
- **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.

**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, S1 is identified and evaluated with respect to its intensity and splitting. Next, S2 is identified, and its intensity and any splitting are noted. After concentrating on S1 and S2, 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? 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 S3, an S4, 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 25-3): 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 (S1, S2) 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 S3 after treatment with a diuretic.

**Assessment of Other Systems**

**Lungs**

The details of respiratory assessment are described in Chapter 20. 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 (Inderal), may cause airway narrowing, especially in patients with underlying pulmonary disease.

Abdomen

For the patient with a cardiovascular disorder, several components of the abdominal examination are relevant:

Abdominal distension: 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.

Hepatocaval 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 hepatocaval 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 Fig. 53-8 in Chapter 53) 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.

Isolated systolic hypertension is directly correlated to the aging process, affecting more than 50% of people 65 years of age or older. By middle age, those without hypertension have a 90% lifetime risk of developing this problem. Untreated hypertension is associated with significant cardiovascular morbidity and mortality, including stroke (Rosen dorff, Lackland, Allison, et al., 2015). Another common BP problem in the older adult is postural (orthostatic) hypotension. It is a result of impaired baroreceptor function, which is necessary to regulate BP. Other factors that heighten the risk for postural hypotension include prolonged bed rest, dehydration, and many cardiovascular medications (e.g., beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, diuretics, nitrates). Postural hypotension, regardless of the cause, places older adults at risk for falls (Weber & Kelley, 2014).

An S4 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 S4 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, 2014) (see Table 25-1).

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 assist in making a diagnosis
- 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 electrocardiogram (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 27 for further discussion of cardiac biomarker analysis.)

Blood Chemistry, Hematology, and Coagulation Studies

Table 25-4 provides information about some common serum laboratory tests and the implications for patients with CVD.
TABLE 25-4  Common Serum Laboratory Tests and Implications for Patients With Cardiovascular Disease

<table>
<thead>
<tr>
<th>Laboratory Test Reference Range</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood Chemistries</strong></td>
<td></td>
</tr>
<tr>
<td>Blood urea nitrogen (BUN): 10–20 mg/dL</td>
<td>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.</td>
</tr>
<tr>
<td>Calcium (Ca²⁺): 8.5–10.5 mg/dL</td>
<td>Calcium is necessary for blood coagulability, neuromuscular activity, and automaticity of the nodal cells (sinus and atrioventricular nodes). Hypocalcemia: Decreased calcium levels slow nodal function and impair myocardial contractility. The latter effect increases the risk for heart failure. Hypercalcemia: 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.</td>
</tr>
<tr>
<td>Creatinine: 0.7–1.4 mg/dL</td>
<td>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.</td>
</tr>
<tr>
<td>Magnesium (Mg²⁺): 1.8–3.0 mg/dL</td>
<td>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. Hypomagnesemia: Decreased magnesium levels are due to enhanced renal excretion of magnesium from the use of diuretics or digitalis therapy. Low magnesium levels predispose patients to atrial or ventricular tachycardias. Hypermagnesemia: 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.</td>
</tr>
<tr>
<td>Potassium (K⁺): 3.5–5 mEq/L</td>
<td>Potassium has a major role in cardiac electrophysiologic function. Hyperkalemia: Decreased potassium levels due to administration of potassium-excreting diuretics can cause many forms of dysrhythmias, including life-threatening ventricular tachycardia or ventricular fibrillation, and predispose patients taking digitalis preparations to digitalis toxicity. Hypokalemia: 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 dysrhythmias.</td>
</tr>
<tr>
<td>Sodium (Na⁺): 135–145 mEq/L</td>
<td>Low or high serum sodium levels do not directly affect cardiac function. Hyponatremia: Decreased sodium levels indicate fluid excess and can be caused by heart failure or administration of thiazide diuretics. Hypernatremia: Increased sodium levels indicate fluid deficits and can result from decreased water intake or loss of water through excessive sweating or diarrhea.</td>
</tr>
<tr>
<td><strong>Coagulation Studies</strong></td>
<td></td>
</tr>
<tr>
<td>Partial thromboplastin time (PTT): 60–70 s</td>
<td>PT or 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 &gt;50 s (1 dose) or &gt;100 s (4 dose).</td>
</tr>
<tr>
<td>Activated partial thromboplastin time (aPTT): 20–39 s</td>
<td></td>
</tr>
<tr>
<td>Prothrombin time (PT): 9.5–12 s</td>
<td>PT measures the extrinsic pathway activity and is used to monitor the level of anticoagulation with warfarin (Coumadin). 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.</td>
</tr>
<tr>
<td>International normalized ratio (INR): 1</td>
<td></td>
</tr>
<tr>
<td><strong>Hematologic Studies</strong></td>
<td></td>
</tr>
<tr>
<td>Complete blood count (CBC)</td>
<td>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. 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. Low hemoglobin and hematocrit levels have serious consequences for patients with cardiovascular disease, such as more frequent angina episodes or acute myocardial infarction.</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>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 (Plavix), and intravenous glycoprotein IIb/IIIa inhibitors (abciximab [ReoPro], eptifibatide [Integrilin], and tirofiban [Aggrastat]). When these medications are given, it is essential to monitor for thrombocytopenia (low platelet counts).</td>
</tr>
<tr>
<td>Male: 42–52%</td>
<td>WBC counts are monitored in immunocompromised patients, including patients with heart transplants or in situations where there is concern for infection (e.g., after invasive procedures or surgery).</td>
</tr>
<tr>
<td>Female: 35–47%</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>Male: 13–18 g/dL</td>
<td></td>
</tr>
<tr>
<td>Female: 12–16 g/dL</td>
<td></td>
</tr>
<tr>
<td>Platelets: 150,000–450,000 (mm³)</td>
<td></td>
</tr>
<tr>
<td>White blood cell (WBC) count: 4500–110,000 (mm³)</td>
<td></td>
</tr>
</tbody>
</table>

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 tool in the setting of HF. The 12-lead ECG is used to diagnose dysrhythmias, 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. It 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.

New CAD prevention guidelines no longer focus on achieving specific cholesterol levels as there is inadequate evidence to support this practice. The mainstay of cholesterol lowering therapy is lifestyle changes. Medical management focuses on determining an individual’s 10-year risk for atherosclerotic vascular disease to identify those most likely to benefit from taking prescribed statins, a class of cholesterol-lowering medications (Stone, Robinson, Lichtenstein, et al., 2014) (see Chapter 27 for more details).

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 (Stone et al., 2014).

**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 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 clinician 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 (Woods et al., 2009).

**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 B6, and vitamin B12 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 mcg/mL), borderline (12 to 15 mcg/mL), and high risk (greater than 15 mcg/mL) (Woods et al., 2009).

**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 (see Chapter 26 for electrode placement). Recordings of the electrical current flowing between two electrodes is 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 the 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 dysrhythmias, 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.
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 digitals and antiarrhythmic agents) are noted on the ECG requisition. (See Chapter 26 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 dysrhythmias. 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 27). 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)

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 dysrhythmias and conduction disorders using 1 or 2 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.

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 accurately placed on the chest wall. Chart 25-4 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 5-lead system can monitor the activity of the anterior wall of the left ventricle. Figure 25-9 presents diagrams of electrode placement.

The two ECG leads most often selected for continuous ECG monitoring are leads II and V1. Lead II provides the best visualization of atrial depolarization (represented by the P wave). Lead V1 best records ventricular depolarization and is most helpful when monitoring for certain dysrhythmias (e.g., premature ventricular contractions, tachycardias, bundle branch blocks) (see Chapter 26).

Ambulatory Electrocardiography

Ambulatory electrocardiography is a form of continuous or intermittent ECG home monitoring. It is used to identify the etiology of syncope or palpitation caused by dysrhythmias, detect episodes of myocardial ischemia, evaluate effectiveness of treatment of HF and dysrhythmias, as well as evaluate the

Chart 25-4  Applying Electrodes

The monitoring system requires an adequate electrical signal to analyze the patient’s cardiac rhythm. 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.

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. Newer devices transmit ECGs through the use of wireless technology to a secure website (Leahy & Davenport, 2015; Romero, 2013; Walsh, Topol, & Steinbuhl, 2014).

Continuous Monitors

Commonly called Holter monitors, these small portable recorders are connected to chest electrodes (number varies based on model used) that record the ECG using several leads onto a digital memory device. The patient wears the recorder for 24 to 48 hours and in certain cases up to 2 weeks. If the patient experiences symptoms they are instructed to activate the event marker. The patient is also asked to keep a diary to note the date and time of symptoms or performance of unusual activities. The diary is used by the primary provider to correlate symptoms with detected dysrhythmias. Once monitoring is completed the patient returns the device 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. The effectiveness of this form of monitoring is dependent upon the patient’s adherence with keeping a diary and marking events. Holter monitors do not provide real-time ECG recordings, which is another limitation.

A novel alternative to the use of the Holter monitor is ECG patch monitoring, which uses blue-tooth 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 up to 14 days and then returns it to the manufacturer for analysis. These devices, which are available from several manufactures, have one major limitation. The ECG patch is capable of monitoring just one ECG lead, which makes it less sensitive to dysrhythmia detection (Walsh et al., 2014).

Continuous Real-Time Monitors

This technology allows the ECG to be monitored continuously at a remote central monitoring station. The patient has three electrodes applied to the chest or wears an electrode belt that is attached to a small sensor. The sensor transmits each heartbeat to a small monitor. When a dysrhythmia is detected, the system automatically transmits the ECG to a monitoring center either through the patient’s telephone line when at home or through wireless communications systems when outside of the home. The patient can also trigger activation of ECG transmissions. The ECG is then analyzed real time by technicians who communicate abnormal findings to the patient’s primary provider. This method of monitoring enhances detection and early treatment of dysrhythmias that might otherwise be diagnosed only after the patient develops serious symptoms (Walsh et al., 2014).

Cardiac Event Recorders

Cardiac event recorders allow patients to record the electrical activity of the heart when they experience 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. Newer technology has the capability of wireless transmission of the events automatically through the use of cellular networks. There are three main forms of recorders.

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 hand held device over the chest. The recorded event is then transmitted to the primary provider by telephone.

The continuous looping memory monitor, a small battery operated device, can record and store the ECG for short periods of time. The monitor is attached to the patient with chest electrodes or a wrist band. Patients are instructed to activate ECG recordings by the push of a button. The device records the patient’s ECG for a predetermined time before and after the device activation. Some of these devices are programmable to detect bradycardia, tachycardia, and irregular rhythms and do not require patient interaction. It 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. An adaptor, attached to the smart phone, converts the phone into a monitoring device. The patient places a finger in the device that digitally records lead I of the ECG. The ECG can be viewed immediately and also be sent as a PDF file via a secure server to the patient’s primary provider (Walsh et al., 2014).

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 dysrhythmias. 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 dysrhythmias without the need for patient interaction (see Chapter 26 for further discussion) (Leahy & Davenport, 2015).

An implantable loop recorder is another type of a cardiac implantable electronic device. This small device, composed of a battery and microchips, is injected under the skin. Compared with event recorders described above, this device offers several advantages It can record ECGs continuously for up to 3 years. It also can eliminate 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 (Romero, 2013).

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 various forms of cardiac monitoring (Hannibal, 2014; Sendelbach & Funk, 2013).

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 dysrhythmias 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. 25-9). See Chart 25-5 for a Nursing Research Profile on electrode placement, skin preparation, and alarm fatigue.

Electrodes and lead connections need to be positioned correctly. Improper positioning can result in artifact that mimics ischemia or dysrhythmias. Two leads should be selected that provide the best tracing for dysrhythmia monitoring, which are usually lead II and the chest lead V5. Electrical equipment in use around the patient should be inspected to be certain that it is functioning properly and has been recently checked.

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**Chart 25-5**

<table>
<thead>
<tr>
<th>NURSING RESEARCH PROFILE</th>
<th>Electrode Placement, Skin Preparation, and Alarm Fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Purpose</strong></td>
<td>Electrocardiographic (ECG) monitoring in the inpatient setting is a common practice for patients at risk for cardiac dysrhythmias, ST-segment changes, or prolongation of the QT interval. It is used for the early detection and notification of an abnormality in the patient’s ECG. When this situation occurs, the monitor sounds an alarm, which can vary in intensity and duration based on its clinical significance. Unfortunately, many things can cause the monitor to alarm inappropriately. Muscle tremors, patient movement, artifact from inadequate skin preparation, and improper ECG electrode placement are the most common reasons for these false alarms. Nurses faced with excessive alarms become desensitized to these sounds, causing alarm fatigue. Alarm fatigue results in delayed responses to alarms and missing alarms, which may have serious life-threatening consequences for the patient. The Joint Commission has set a goal for 2017 that no patient should be harmed by false alarms. Therefore, the purpose of this study was to evaluate the effect of proper skin preparation and ECG electrode placement on alarm rates. The nurse researchers hypothesized that using proper techniques would reduce alarms on a telemetry unit.</td>
</tr>
<tr>
<td><strong>Design</strong></td>
<td>The conceptual framework for this study was the American Association of Critical Care Nurses Synergy Model for Patient Care. This model links competent nursing practice with positive patient outcomes. A prospective descriptive design was used to test the effect that proper skin preparation and ECG electrode placement had on the number of alarms on a telemetry unit in a suburban Veterans Affairs Medical Center. The hospital’s Institutional Review Board ruled that the study was quality improvement and subjects were not required to provide consent to participate. After admission, the number of alarms was counted for 24 hours on 25 patients who had ECG electrodes placed using usual care. After 24 hours, the ECG electrodes were removed, the skin prepared (clipping hair if necessary, cleansing skin with soap and water, drying the skin with a wash cloth that causes mild skin abrasions), and reapplying the electrode using proper anatomic locations. The number of alarms was then recounted for 24 hours.</td>
</tr>
<tr>
<td><strong>Findings</strong></td>
<td>After the reapplication of ECG electrodes, nine patients were discharged before 24 hours and one patient refused to have his leads changes. Therefore, the final sample was composed of 15 men; the majority were white (93%) and had a mean age of 71.2 (±12.7) years. The initial alarm count for the first 24 hours was between 2 and 1341, compared with 1 and 992 alarms in the second 24 hours period. The mean number of alarms was significantly lower in patients after the second 24 hours, as compared with the first 24 hours. The use of proper skin preparation and lead placement was associated with a 44% reduction in the mean number of alarms.</td>
</tr>
<tr>
<td><strong>Nursing Implications</strong></td>
<td>The results of this study demonstrate that the use of guidelines for skin preparation and electrode placement recommended by the American Association of Critical Care Nurses results in safer and more effective ECG monitoring practices. The use of evidence-based ECG monitoring practices by nurses results in fewer false alarms and minimizes the risks to patients associated with alarm fatigue.</td>
</tr>
</tbody>
</table>
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 (Drew, Harris, Ze’gre-Hemsey, et al., 2014).

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 dysrhythmia alarms indicating that the patient is tachycardic, bradycardic, or experiencing another potentially life-threatening dysrhythmia. Timely responses to all alarms can prevent serious consequences, including death.

Hospital-acquired infections may be prevented by keeping lead wire 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 demand, or “stress.” The cardiac stress test procedures—the exercise stress test and the pharmacologic stress test—are noninvasive ways to evaluate the response of the cardiovascular system to stress. The stress test helps determine the following: (1) presence of CAD, (2) cause of chest pain, (3) functional capacity of the heart after an MI or heart surgery, (4) effectiveness of antianginal or antiarrhythmic medications, (5) occurrence of dysrhythmias, and (6) specific goals for a physical fitness program. Contraindications to stress testing include acute MI within 48 hours, unstable angina, uncontrolled dysrhythmias with hemodynamic compromise, severe aortic stenosis, acute myocarditis or pericarditis, and decompensated HF (Fletcher, Ades, Kligfield, et al., 2013). Because 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.

Stress testing is often combined with echocardiography or radionuclide imaging, techniques used to capture images of the heart. Cardiac imaging is performed during the resting state and immediately after stress testing.

#### Exercise Stress Testing

**Procedure**

During an exercise stress test, the patient walks on a treadmill (most common) or pedals a stationary bicycle. Exercise intensity progresses according to established protocols. The protocol selected for the test is based on the purpose of the test and the physical fitness level and health of the patient (Fletcher et al. 2013). During the test, the following are monitored: 2 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 terminated when the target heart rate is achieved or if the patient experiences signs of myocardial ischemia. Further diagnostic testing, such as a cardiac catheterization, may be warranted if the patient develops chest pain, extreme fatigue, a decrease in BP or pulse rate, serious dysrhythmias, or ST-segment changes on the ECG during the stress test.

**Nursing Interventions**

In preparation for the exercise stress test, the patient is instructed to fast for at least 3 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 not to take certain cardiac medications, such as beta-adrenergic blocking agents, before the 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. 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 (Persantine), adenosine (Adenocard), or regadenoson (Lexiscan) given as an IV infusion are used to mimic the effects of exercise by maximally dilating normal coronary arteries. 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 liquids or food that contain chocolate or caffeine for 24 hours. This restriction also includes caffeine-free coffee, tea, carbonated beverages, as well as medications containing caffeine (e.g., Anacin). 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 help 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 used most often because it is more widely available and is technically easier to perform than PET. In addition, the diagnostic and prognostic value of SPECT is better established than PET (Fletcher et al., 2013).

Procedure

SPECT is a painless, noninvasive procedure that involves the injection of one of three commercially available tracers (technetium [Tc]-99m sestamibi, Tc-99m-tetrofosmin, or thallium-201).

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 tracers into pictures of the heart. The procedure takes approximately 30 minutes. The second scan is repeated following the 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 produces better pictures than SPECT, is faster, and uses lower doses of radiation. However, its use is limited because not all facilities have a PET scanner and the technical equipment needed to produce the tracers (Fletcher et al., 2013).

Procedure

During PET, tracers are given by injection; one compound is used to determine blood flow in the myocardium, and...
takes multiple slices at the same time. This technology helps analyze the slices to create three-dimensional images. Multiplex mathematical and computer algorithms are used to specific areas of the heart and surrounding structures. Computed tomography to provide accurate cross-sectional “virtual” slices of Cardiac CT scanning is a form of cardiac imaging that uses Procedure

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 helps to produce clear images by eliminating artifact created by the beating heart and respirations (Stone et al., 2014). There are two types of cardiac CT scanning that 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 (CABG), 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, Davenport, Ellis, et al., 2015).

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 (Lloyd-Jones, 2015).

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 time 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 are 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. MRA cannot be performed on patients who have a pacemaker, 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., NicoDerm, Androderm, Transderm Nitro, Transderm Scop, Catapres-TTS) 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 “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 alternate 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 moderate sedation are used during TEE because of the discomfort associated with the positioning of the transducer in the esophagus (refer to Chapter 18 for further discussion of moderate sedation). 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, dysrhythmias, 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 (SpO₂). During the recovery period, the patient must maintain bed rest with the head of the bed elevated to 45°. Following the moderate sedation policy of the agency, the nurse monitors the patient for dyspnea and assesses vital signs, SpO₂, 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 27).

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 catheterization, 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 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 procedure (Jorgensen, 2013). See Chart 53-5 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 dysrhythmias. 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, and bleeding disorder). The use of smaller (4 or 6 Fr) arterial catheters is 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 27).

### 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 an antecubital 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 dysrhythmias (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 [Benadryl]) and corticosteroids (e.g., prednisone). Patients at risk for CIN receive pre- and postprocedure preventive strategies. IV hydration increases vascular volume and facilitates removal of contrast from the kidneys. Administration of sodium bicarbonate and the antioxidant acetylcysteine (Mucomyst), both urine alkalizing agents, aids in protecting the renal tubules from an acidic environment (Jorgensen, 2013).
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 brachial artery or a femoral artery and advances it into the aorta and left ventricle. Potential complications include dysrhythmias, 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 viewed on fluoroscopy 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 dysrhythmias.

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 Termuo 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.

For the femoral approach, manual pressure may be used alone or in combination with mechanical compression devices such as the FemoStop™ (placed over the puncture site for 30 minutes). 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 (Quick-Seal), collagen (VasoSeal), sutures (Perclose, Techstar), 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 a dysrhythmia 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 dictated 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 dysrhythmias 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 bed rest for 2 to 6 hours after the procedure. Duration of bed rest is dependent upon location of arterial approach, size of the catheter used during the procedure, 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°. 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. The patient may be permitted to ambulate within 2 hours. If the radial artery was accessed, the patient remains on bed rest for 2 hours or until the effects of sedation have dissipated. The patient may sit up in bed. A hemostasis band or pressure dressing may be applied over the catheter access site. Patients are instructed to avoid sleeping on the affected arm for 24 hours and to avoid repetitive movement of the affected extremity for 24 to 48 hours (Rosendorf, 2013). 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 contrast-induced nephropathy 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 (see Chart 25-6).

**Electrophysiologic Testing**

The electrophysiology study (EPS) is an invasive procedure that plays a major role in the diagnosis and management of serious dysrhythmias. EPS may be indicated for patients with syncope, palpitations, or both, and for survivors of cardiac arrest from ventricular fibrillation (sudden cardiac death) (Woods et al., 2009). 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 dysrhythmia (e.g., ventricular tachycardia, ventricular fibrillation) can be induced; to evaluate AV node function; to evaluate the effectiveness of antiarrhythmic medications in suppressing the dysrhythmia; or to determine the need for other therapeutic interventions, such as a pacemaker, ICD, or radiofrequency ablation. (See Chapter 26 for a detailed discussion of EPS.)

**Chart 25-6**

**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 arm or wrist artery was used: For the next 48 hours, avoid lifting anything heavier than 5 lb and avoid repetitive movement of your affected hand and wrist.

- 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 future heart problems, such as quitting smoking, lowering your cholesterol level, initiating dietary changes, beginning an exercise program, 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.


**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 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 (which may include heparin), 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 (see Fig. 25-10). 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 to 60°; however, the system must be repositioned to the phlebostatic axis to ensure an accurate reading (Urdan, Stacy, & Lough, 2014).

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. Comprehensive guidelines for the prevention of these infections have been published by Centers for Disease Control and Prevention (CDC) (O'Grady, Alexander, Burns, et al., 2011). To minimize the risk of infection, a group of evidence-based interventions, called a care bundle, should be implemented (see Chart 14-2).

The CDC has additional infection control guidelines that pertain to the ongoing care of these patients, including skin care, dressing changes, and pressure monitoring systems that are outlined in Table 25-5.

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 are 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.

Before insertion of a CVP catheter, the site is prepared as recommended by the CDC (see Chart 14-2). 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.

Figure 25-10 • 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°. Note the phlebostatic axis changes as the HOB is elevated; thus, the stopcock and transducer must be repositioned after each position change.
TABLE 25-5 Nursing Interventions to Prevent Intravascular Catheter-Related Bloodstream Infections

<table>
<thead>
<tr>
<th>Topic</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand hygiene</td>
<td>• Wash hands with soap and water or use alcohol-based hand rubs before and after contact with the catheter for any reason.</td>
</tr>
<tr>
<td>Dressing</td>
<td>• Wear clean or sterile gloves when changing the dressing. • Cleanse the skin during dressing changes with a ≥0.5% chlorhexidine preparation with alcohol. • 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. • Change gauze dressings every 2 days or transparent dressings at least every 7 days and whenever dressings become damp, loosened, or visibly soiled. • Do not use topical antibiotic ointment or creams on insertion sites.</td>
</tr>
<tr>
<td>Catheter site</td>
<td>• 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.</td>
</tr>
<tr>
<td>Pressure monitoring system</td>
<td>• Keep all components of the pressure monitoring system sterile. • Replace transducers, tubing, continuous-flush device, and flush solution every at 96-hour intervals. • Do not infuse dextrose containing solutions through the monitoring system.</td>
</tr>
<tr>
<td>Bathing</td>
<td>• Do not submerge the catheter or catheter site in water. • Showering is permitted if the catheter and related tubing are placed in an impermeable cover.</td>
</tr>
<tr>
<td>Patient education</td>
<td>• Ask patients to report any new discomforts from the catheter site.</td>
</tr>
</tbody>
</table>


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 is outlined in Table 25-5.

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, vasocative 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 (see Fig. 25-11). 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 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 (1.5-mL capacity). The valve opens and closes the balloon inflation lumen.

Figure 25-11 • 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 lumens 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.
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. 25-11).

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 (see Fig. 25-12). Fluoroscopy

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**Figure 25-12** 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.
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 dysrhythmias, 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. 25-2 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, 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 pulmonary artery pressure 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. 25-10). Serious complications include pulmonary artery rupture, pulmonary thromboembolism, pulmonary infarction, catheter kinking, dysrhythmias, 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. Traditionally, collateral circulation to the involved extremity was assessed by using the Allen test. To perform the Allen test, the hand is elevated and the patient is asked to make a fist for 30 seconds. 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 6 seconds, the circulation to the hand may be adequate enough to tolerate placement of a radial artery catheter. Evidence suggests that pulse oximetry and plethysmography are additional reliable methods for assessing circulation to the hand (Rosendorff, 2013).

**Nursing Interventions**

Site preparation and care are the same as for CVP catheters. The catheter flush solution is the same as for 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 almost 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., 2014).

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, various dysrhythmias, hemodynamic instability, and those who may be concomitantly using an intra-aortic balloon pump (see Chapter 29).

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., 2014).

In patients who are sedated, intubated, and on mechanical ventilation, the Fick principle, which uses carbon dioxide (CO₂) measures, is an additional method used to estimate cardiac output. To obtain cardiac output in this select patient population, a rebreathing loop is attached to the ventilator along with an infrared CO₂ sensor, an airflow sensor, and pulse oximeter. Continuous readings of cardiac output may be updated every 3 minutes with the use of this device.
CRITICAL THINKING EXERCISES

1 Your patient is being admitted to the cardiac ICU directly from the chronic HF clinic with acute decompensated HF. He is a 74-year-old man with a history of several MI s and an ejection fraction of 28%. The clinic nurse tells you that he is not responding to diuretics due to poor renal function. The cardiologist is planning to insert a pulmonary artery catheter to monitor the patient's response to IV dobutamine, prescribed to strengthen myocardial contractility. What information will you need to gather during the admission history and physical assessment? What laboratory results will you review to determine the patient's renal function? Why is a pulmonary artery catheter indicated for monitoring this patient? What parameters will you monitor to determine the patient's response to dobutamine? How will you determine that the catheter remains in a stable position in the pulmonary artery?

2 After working on a medical nursing unit for a year, you are transferring to a new position in the diagnostic imaging department. As part of your responsibilities you provide preprocedure telephone calls to patients undergoing diagnostic cardiac catheterizations. How will you determine if patients are at risk for contrast agent allergies or contrast-induced nephropathy? What information will you include in your conversation with patients regarding what to expect during the procedure? What information will you teach the patient about pre- and postprocedure care? What is the strength of the evidence that guides your teaching strategies? How will you know your teaching was effective?

3 During morning cardiology rounds, the team tells your patient that he will be scheduled for a stress test with imaging the following day. As they leave the patient’s bedside, the cardiologist asks you to prepare the patient for the stress test. What additional information do you need to discuss with the team before they leave the unit? What priority health history and physical assessment findings of the patient will be used to determine if a pharmacologic or exercise stress test is appropriate for this patient? Discuss the differences between these two stress tests. How do these differences affect patient preparation and education?

REFERENCES

*Asterisk indicates nursing research. **Double asterisk indicates classic reference.

Books


Journals and Electronic Documents


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American College of Cardiology (ACC), www.acc.org
American Heart Association (AHA), www.heart.org/HEARTORG/
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