

PART IV

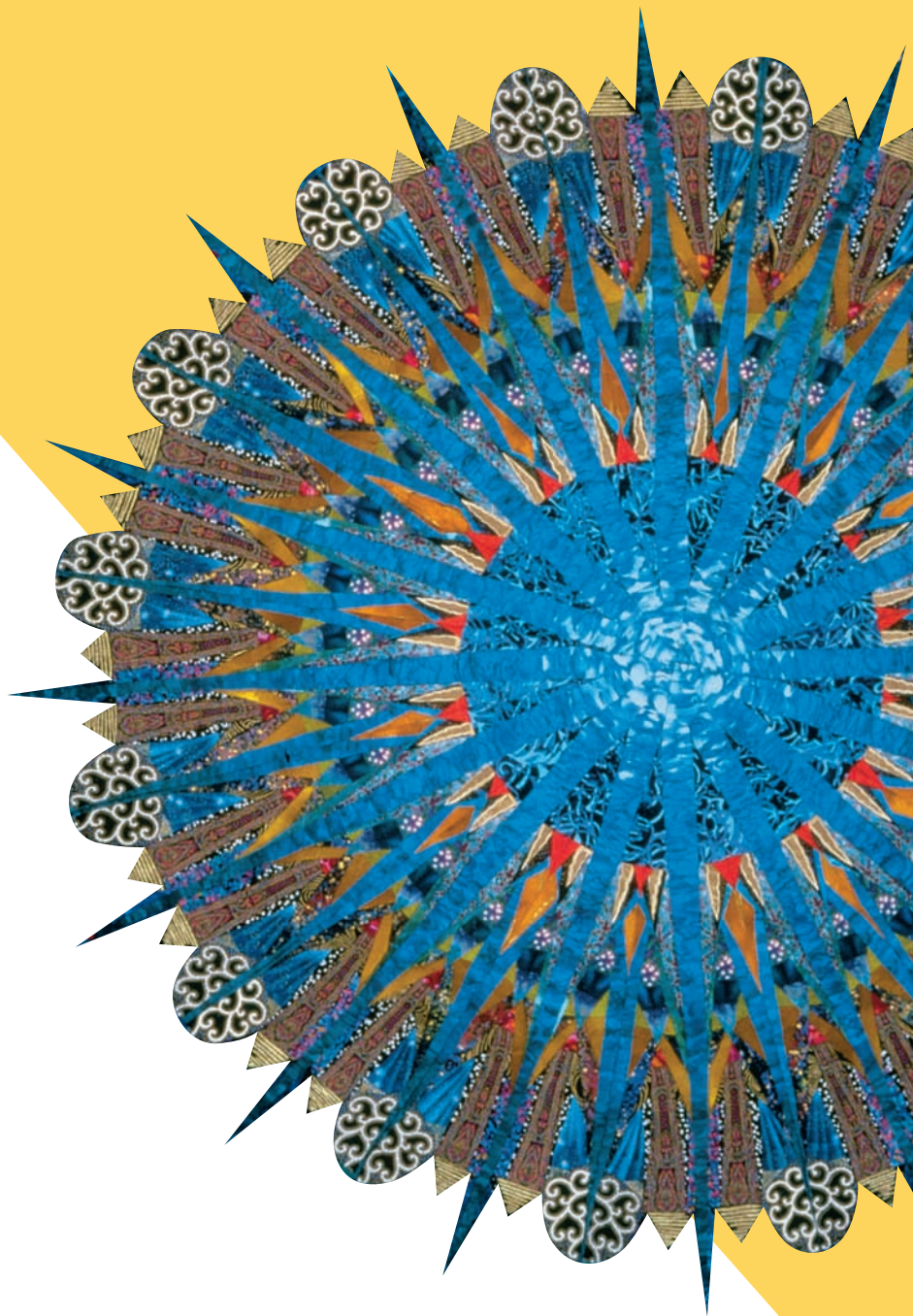
Activity and Exercise Patterns

UNIT 9
Responses to Altered Cardiac Function

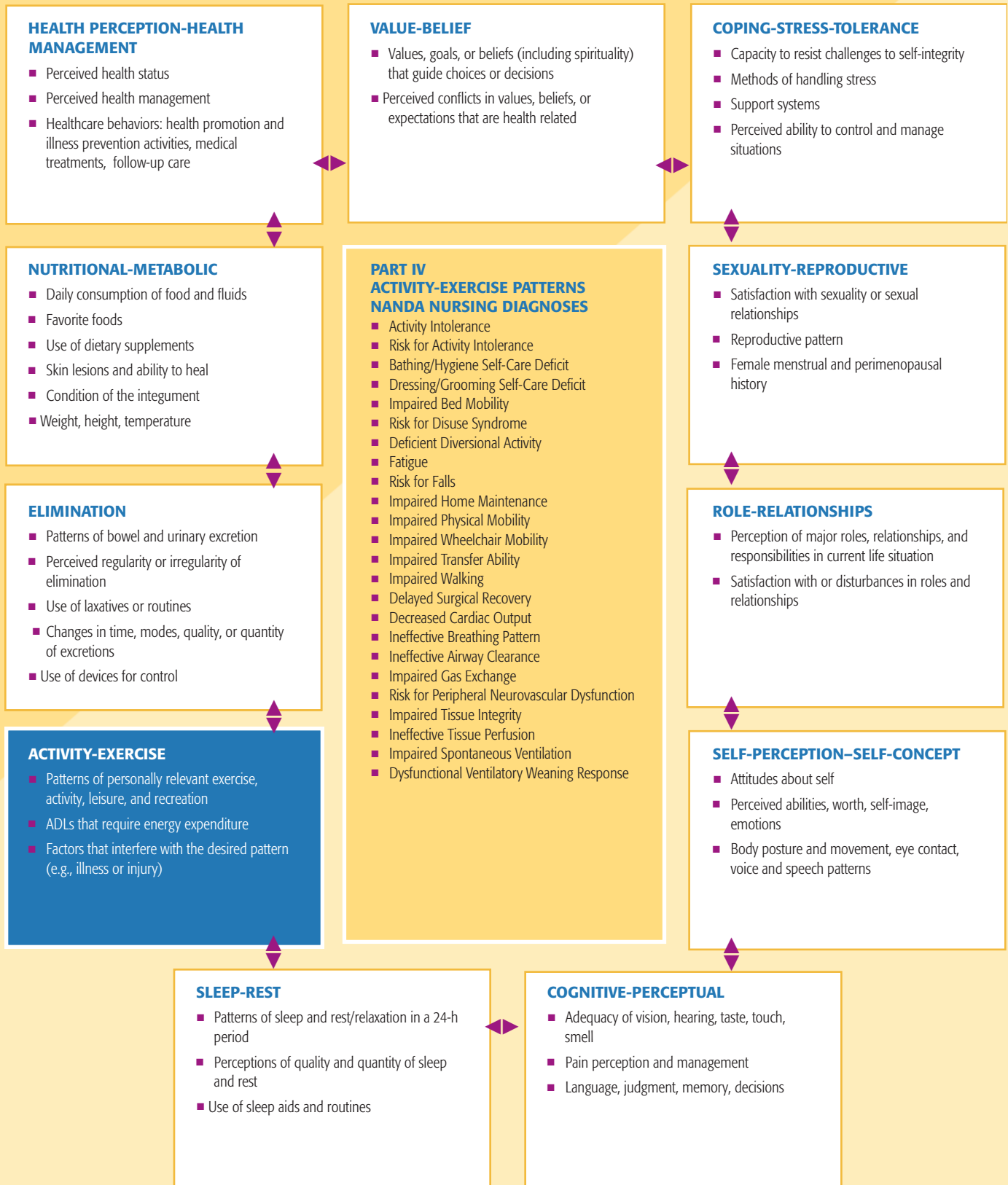
UNIT 10
**Responses to Altered Peripheral Tissue
Perfusion**

UNIT 11
Responses to Altered Respiratory Function

UNIT 12
**Responses to Altered Musculoskeletal
Function**



Functional Health Patterns with Related Nursing Diagnoses



UNIT 9

Responses to Altered Cardiac Function

CHAPTER 30

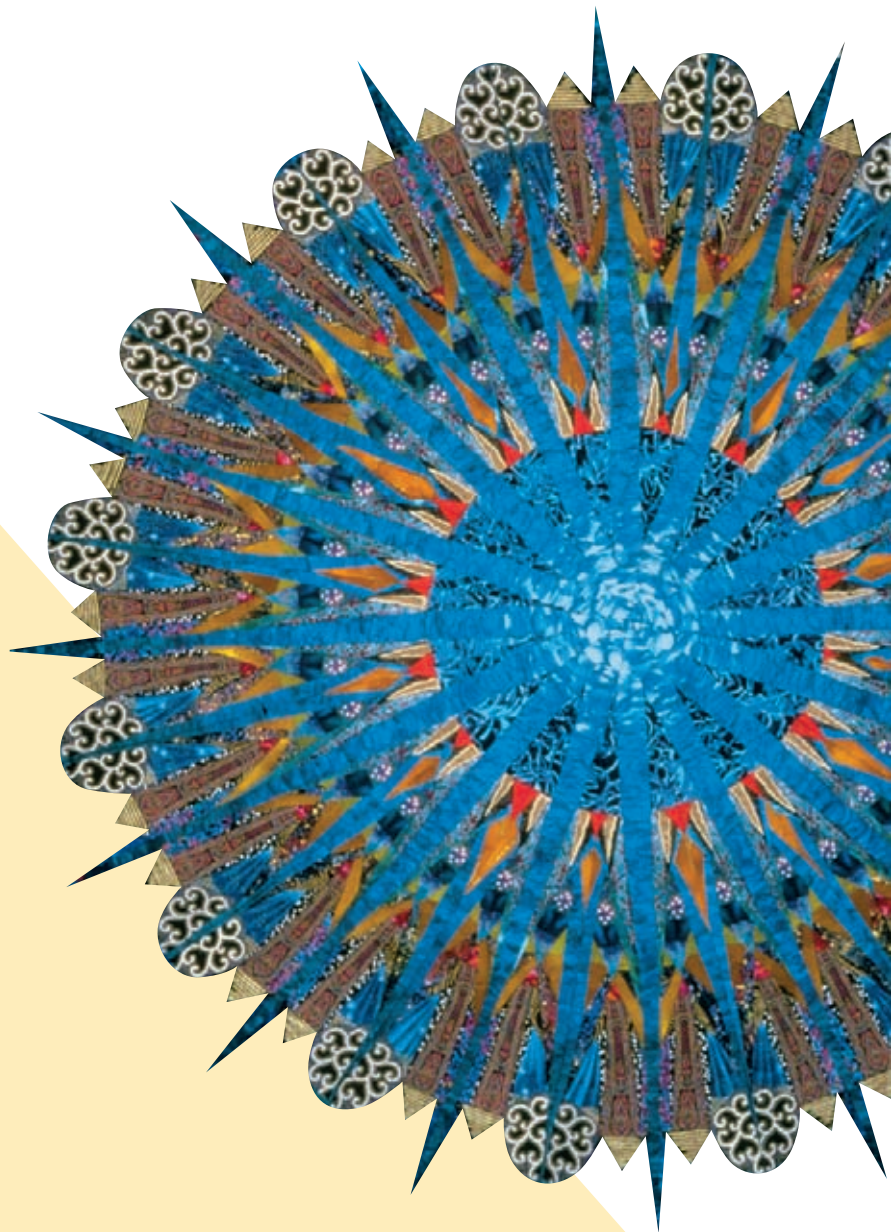
**Assessing Clients with Cardiac
Disorders**

CHAPTER 31

**Nursing Care of Clients with Coronary
Heart Disease**

CHAPTER 32

**Nursing Care of Clients with Cardiac
Disorders**



CHAPTER Assessing Clients 30 with Cardiac Disorders

LEARNING OUTCOMES

- Describe the anatomy, physiology, and functions of the heart.
- Trace the circulation of blood through the heart and coronary vessels.
- Identify normal heart sounds and relate them to the corresponding events in the cardiac cycle.
- Explain cardiac output and the influence of various factors in its regulation.
- Describe normal variations in assessment findings for the older adult.
- Identify manifestations of impaired cardiac structure and functions.

CLINICAL COMPETENCIES

- Assess an ECG strip and identify normal and abnormal cardiac rhythm.
- Conduct and document a health history for clients having or at risk for having alterations in the structure and functions of the heart.
- Conduct and document a physical assessment of cardiac status.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Stethoscope with a diaphragm and a bell
- Good light source
- Watch with a second hand
- Centimeter ruler

MEDIA LINK



Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD accompanying this textbook, and on the Companion Website at <http://www.prenhall.com/lemone>



KEY TERMS

afterload, 940
apical impulse, 952
cardiac index (CI), 941
cardiac output (CO), 940
cardiac reserve, 940
contractility, 940

dysrhythmia, 953
ejection fraction, 940
heave, 952
ischemic, 940
lift, 952
murmur, 950

preload, 940
pulsations, 952
retraction, 952
stroke volume (SV), 940
thrill, 953
thrust, 952

The heart, a muscular pump, beats an average of 70 times per minute, or once every 0.86 second, every minute of a person's life. This continuous pumping moves blood through the body, nourishing tissue cells and removing wastes. Deficits in the structure or function of the heart affect all body tissues.

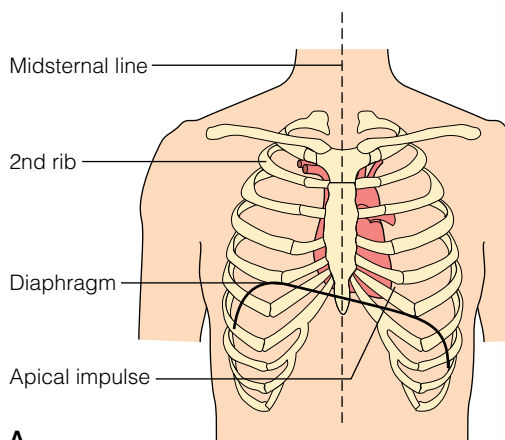
Changes in cardiac rate, rhythm, or output may limit almost all human functions, including self-care, mobility, and the ability to maintain fluid volume status, respirations, tissue perfusion, and comfort. Cardiac changes may also affect self-concept, sexuality, and role performance.

ANATOMY, PHYSIOLOGY, AND FUNCTIONS OF THE HEART

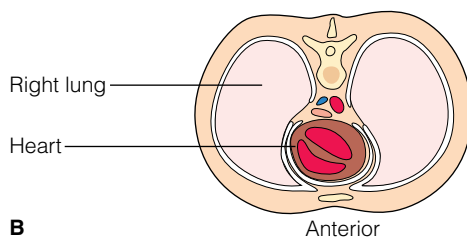
The heart is a hollow, cone-shaped organ approximately the size of an adult's fist, weighing less than 1 lb. It is located in the mediastinum of the thoracic cavity, between the vertebral column and the sternum, and is flanked laterally by the lungs. Two-thirds of the heart mass lies to the left of the sternum; the upper base lies beneath the second rib, and the pointed apex is approximate with the fifth intercostal space, midpoint to the clavicle (Figure 30-1 ■).

The Pericardium

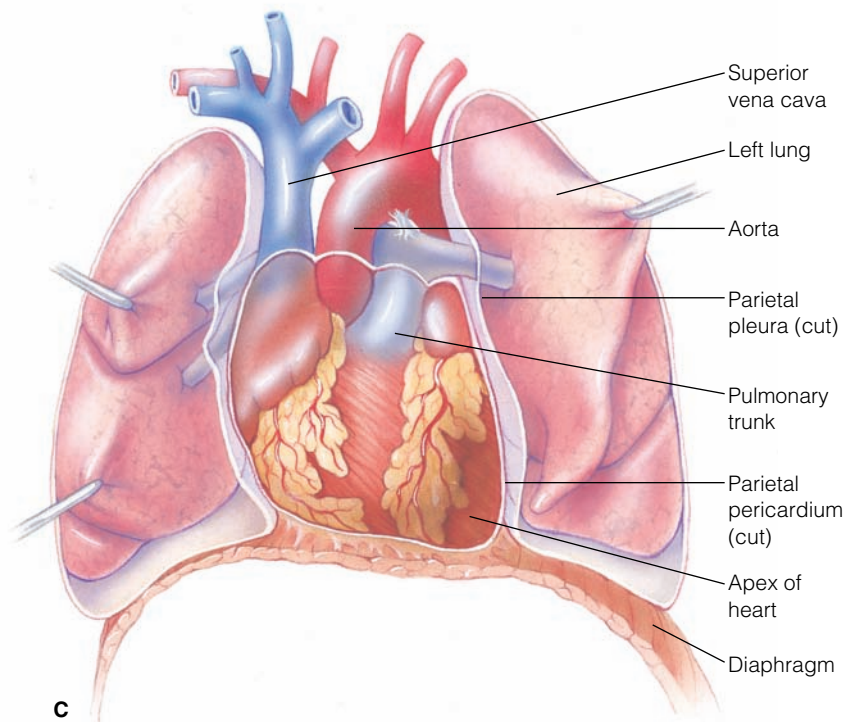
The heart is covered by the pericardium, a double layer of fibroserous membrane (Figure 30-2 ■). The pericardium encases the heart and anchors it to surrounding structures, forming the pericardial sac. The snug fit of the pericardium prevents the heart from overfilling with blood. The outermost layer is the parietal pericardium, and the visceral pericardium



A



B



C

Figure 30-1 ■ Location of the heart in the mediastinum of the thorax. *A*, Relationship of the heart to the sternum, ribs, and diaphragm. *B*, Cross-sectional view showing relative position of the heart in the thorax. *C*, Relationship of the heart and great vessels to the lungs.

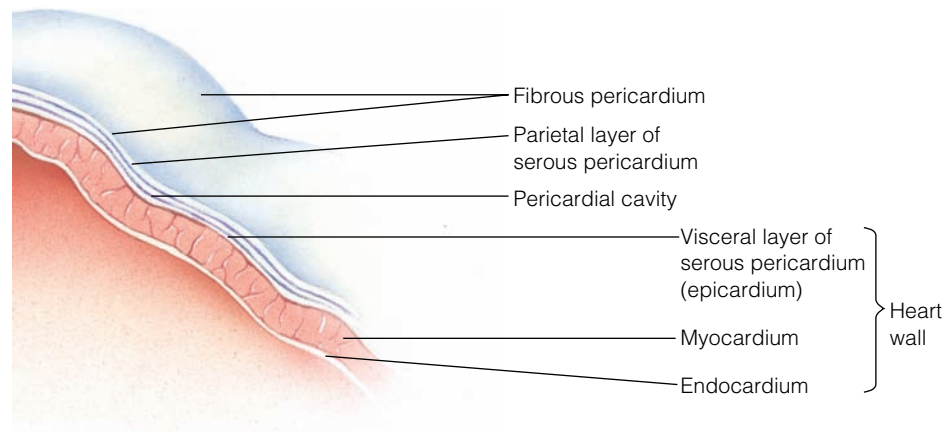


Figure 30-2 ■ Coverings and layers of the heart.

(or epicardium) adheres to the heart surface. The small space between the visceral and parietal layers of the pericardium is called the pericardial cavity. A serous lubricating fluid produced in this space cushions the heart as it beats.

Layers of the Heart Wall

The heart wall consists of three layers of tissue: the epicardium, the myocardium, and the endocardium (see Figure 30-2). The epicardium covers the entire heart and great vessels, and then folds over to form the parietal layer that lines the pericardium and adheres to the heart surface. The myocardium, which is the

middle layer of the heart wall, consists of specialized cardiac muscle cells (myofibrils) that provide the bulk of contractile heart muscle. The endocardium, which is the innermost layer, is a thin membrane composed of three layers; the innermost layer is made up of smooth endothelial cells that line the inside of the heart's chambers and great vessels.

Chambers and Valves of the Heart

The heart has four hollow chambers, two upper atria and two lower ventricles. They are separated longitudinally by the interventricular septum (Figure 30-3 ■).

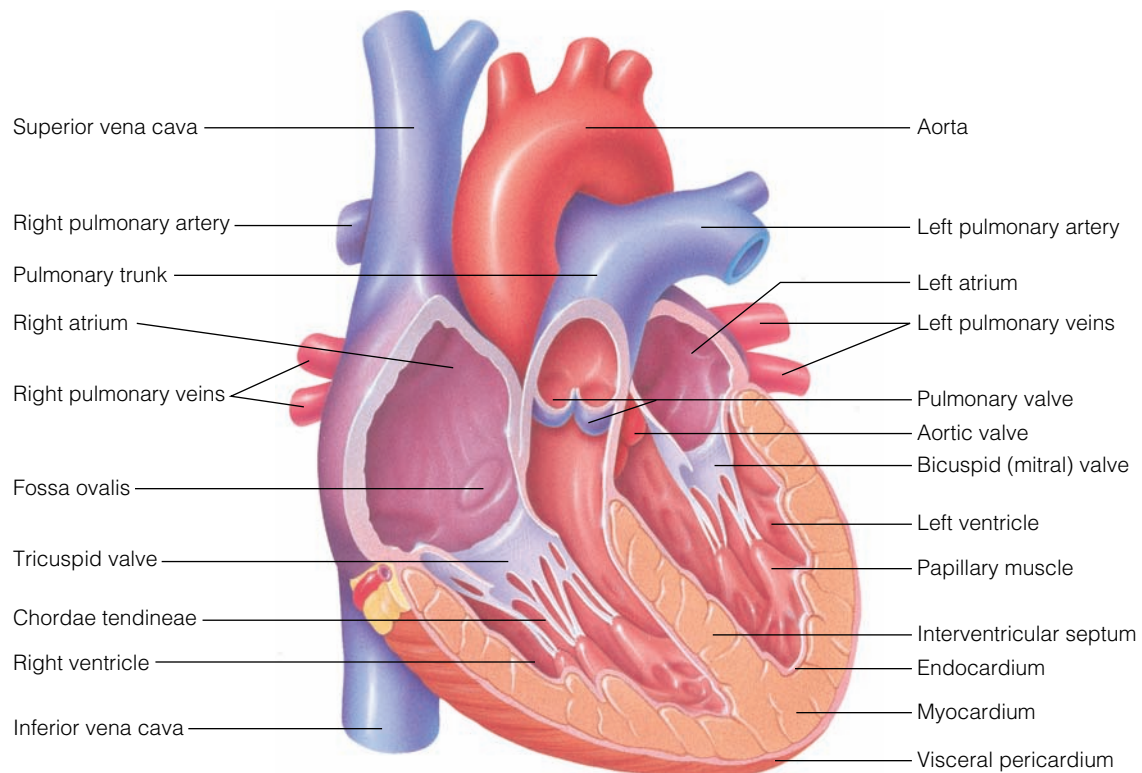


Figure 30-3 ■ The internal anatomy of the heart, frontal section.

The right atrium receives deoxygenated blood from the veins of the body: The superior vena cava returns blood from the body area above the diaphragm, the inferior vena cava returns blood from the body below the diaphragm, and the coronary sinus drains blood from the heart. The left atrium receives freshly oxygenated blood from the lungs through the pulmonary veins.

The right ventricle receives deoxygenated blood from the right atrium and pumps it through the pulmonary artery to the pulmonary capillary bed for oxygenation. The newly oxygenated blood then travels through the pulmonary veins to the left atrium. Blood enters the left atrium and crosses the mitral (bicuspid) valve into the left ventricle. Blood is then pumped out of the aorta to the arterial circulation.

Each of the heart's chambers is separated by a valve that allows unidirectional blood flow to the next chamber or great vessel (see Figure 30–3). The atria are separated from the ventricles by the two atrioventricular (AV) valves; the tricuspid valve is on the right side, and the bicuspid (or mitral) valve is on the left. The flaps of each of these valves are anchored to the papillary muscles of the ventricles by the chordae tendineae. These structures control the movement of the AV valves to prevent backflow of blood. The ventricles are connected to their great vessels by the semilunar valves. On the right, the pulmonary (pulmonic) valve joins the right ventricle with the pulmonary artery. On the left, the aortic valve joins the left ventricle to the aorta.

Closure of the AV valves at the onset of contraction (systole) produces the first heart sound, or S₁ (characterized by the syllable “lub”); closure of the semilunar valves at the onset of relaxation (diastole) produces the second heart sound, or S₂ (characterized by the syllable “dub”).

Systemic, Pulmonary, and Coronary Circulation

Because each side of the heart both receives and ejects blood, the heart is often described as a double pump. Blood enters the right atrium and moves to the pulmonary bed at almost the exact same time that blood is entering the left atrium. The circulatory system has two parts: the pulmonary circulation (moving blood through the capillary bed surrounding the lungs to link with the gas exchange system of the lungs), and the systemic circulation, which supplies blood to all other body tissues. In addition, the heart muscle itself is supplied with blood via the coronary circulation.

Systemic Circulation

The systemic circulation consists of the left side of the heart, the aorta and its branches, the capillaries that supply the brain and peripheral tissues, the systemic venous system, and the vena cava. The systemic system, which must move blood to peripheral areas of the body, is a high-pressure system.

Pulmonary Circulation

The pulmonary circulation consists of the right side of the heart, the pulmonary artery, the pulmonary capillaries, and the pulmonary vein. Because it is located in the thorax near the heart, the pulmonary circulation is a low-pressure system. Pulmonary circulation begins with the right side of the heart. De-

oxygenated blood from the venous system enters the right atrium through two large veins, the superior and inferior venae cavae, and is transported to the lungs via the pulmonary artery and its branches (Figure 30–4 ■). After oxygen and carbon dioxide are exchanged in the pulmonary capillaries, oxygen-rich blood returns to the left atrium through several pulmonary veins. Blood is then pumped out of the left ventricle through the aorta and its major branches to supply all body tissues. This second circuit of blood flow is called the systemic circulation.

Coronary Circulation

The heart muscle itself is supplied by its own network of vessels through the coronary circulation. The left and right coronary arteries originate at the base of the aorta and branch out to encircle the myocardium (Figure 30–5A ■), supplying blood, oxygen, and

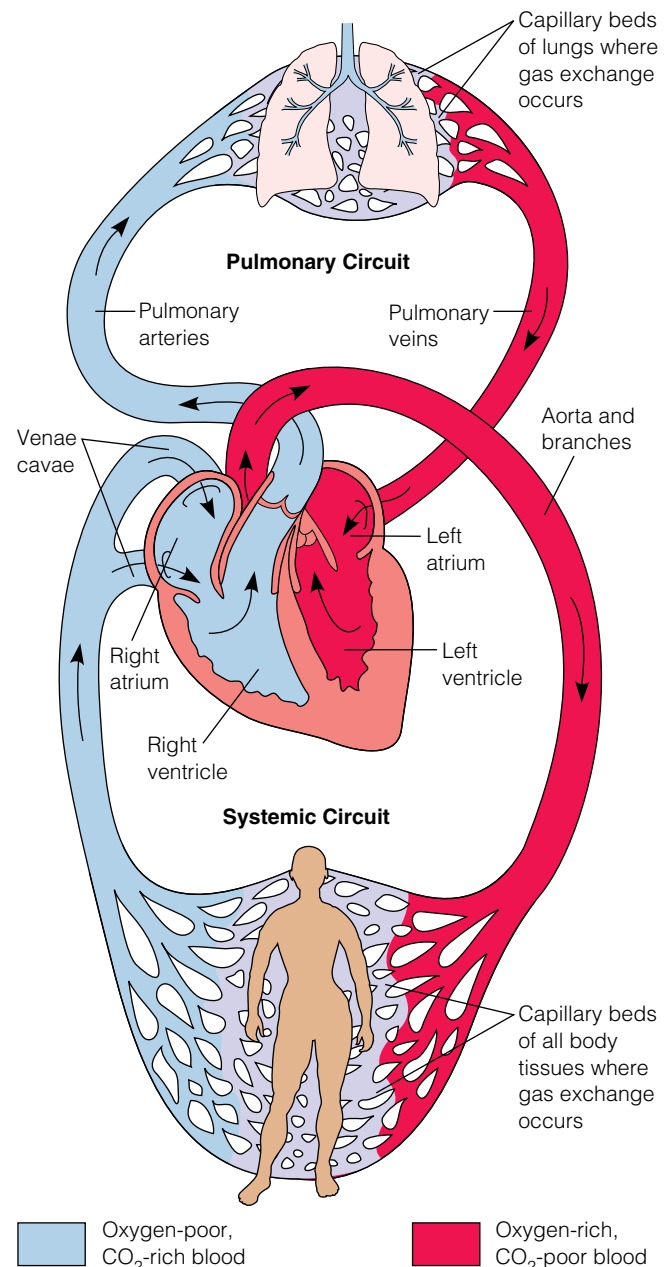


Figure 30–4 ■ Pulmonary and systemic circulation.

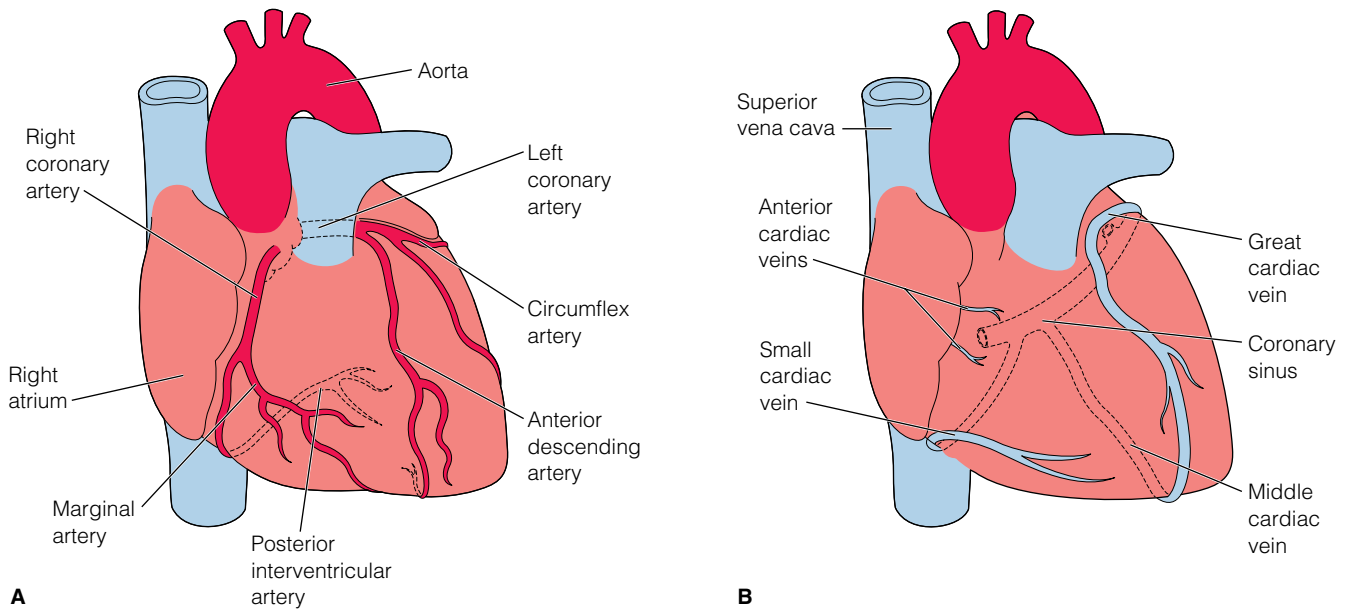


Figure 30-5 ■ Coronary circulation. *A*, Coronary arteries; *B*, coronary veins.

nutrients to the myocardium. The left main coronary artery divides to form the anterior descending and circumflex arteries. The anterior descending artery supplies the anterior interventricular septum and the left ventricle. The circumflex branch supplies the left lateral wall of the left ventricle. The right coronary artery supplies the right ventricle and forms the posterior descending artery. The posterior descending artery supplies the posterior portion of the heart. While ventricular contraction delivers blood through the pulmonary circulation and the systemic circulation, it is during ventricular relaxation that the coronary arteries fill with oxygen-rich blood. After the blood perfuses the heart muscle, the cardiac veins drain the blood into the coronary sinus, which empties into the right atrium of the heart (Figure 30-5B).

Blood flow through the coronary arteries is regulated by several factors. Aortic pressure is the primary factor. Other factors include the heart rate (most flow occurs during diastole,

when the muscle is relaxed), metabolic activity of the heart, and blood vessel tone (constriction).

The Cardiac Cycle and Cardiac Output

The contraction and relaxation of the heart constitutes one heartbeat and is called the cardiac cycle (Figure 30-6 ■). Ventricular filling is followed by ventricular systole, a phase during which the ventricles contract and eject blood into the pulmonary and systemic circuits. Systole is followed by a relaxation phase known as diastole, during which the ventricles refill, the atria contract, and the myocardium is perfused. Normally, the complete cardiac cycle occurs about 70 to 80 times per minute, measured as the heart rate (HR).

During diastole, the volume in the ventricles is increased to about 120 mL (the end-diastolic volume), and at the end of systole, about 50 mL of blood remains in the ventricles (the

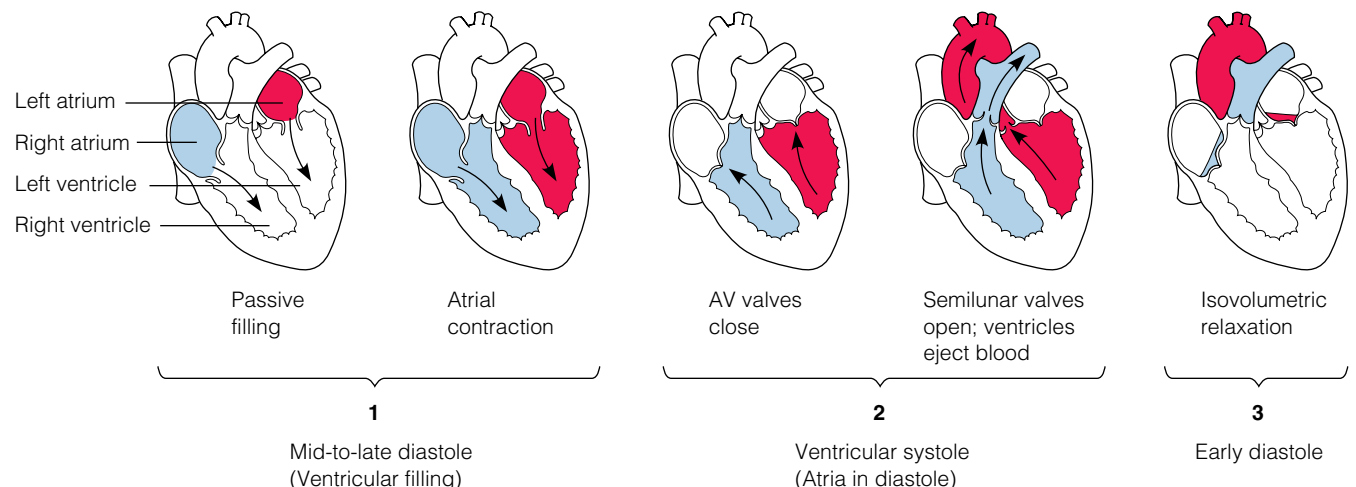


Figure 30-6 ■ The cardiac cycle has three events: (1) ventricular filling in mid-to-late diastole, (2) ventricular systole, and (3) isovolumetric relaxation in early diastole.

end-systolic volume). The difference between the end-diastolic volume and the end-systolic volume is called the **stroke volume (SV)**. Stroke volume ranges from 60 to 100 mL/beat and averages about 70 mL/beat in an adult. The **cardiac output (CO)** is the amount of blood pumped by the ventricles into the pulmonary and systemic circulations in 1 minute. Multiplying the stroke volume (SV) by the heart rate (HR) determines the cardiac output: $CO \times HR = SV$. The **ejection fraction** is the stroke volume divided by the end-diastolic volume and represents the fraction or percent of the diastolic volume that is ejected from the heart during systole (Porth, 2005). For example, an end-diastolic volume of 120 mL divided by a stroke volume of 80 mL equals an ejection fraction of 66%. The normal ejection fraction ranges from 50% to 70%.

The average adult cardiac output ranges from 4 to 8 L/min. Cardiac output is an indicator of how well the heart is functioning as a pump. If the heart cannot pump effectively, cardiac output and tissue perfusion are decreased. Body tissues that do not receive enough blood and oxygen (carried in the blood on hemoglobin) become **ischemic** (deprived of oxygen). If the tissues do not receive enough blood flow to maintain the functions of the cells, the cells die (cellular death results in necrosis or infarction).

Activity level, metabolic rate, physiologic and psychologic stress responses, age, and body size all influence cardiac output. In addition, cardiac output is determined by the interaction of four major factors: heart rate, preload, afterload, and contractility. Changes in each of these variables influence cardiac output intrinsically, and each also can be manipulated to affect cardiac output. The heart's ability to respond to the body's changing need for cardiac output is called **cardiac reserve**.

Heart Rate

Heart rate is affected by both direct and indirect autonomic nervous system stimulation. Direct stimulation is accomplished through the innervation of the heart muscle by sympathetic and parasympathetic nerves. The sympathetic nervous system increases the heart rate, whereas the parasympathetic vagal tone slows the heart rate. Reflex regulation of the heart rate in response to systemic blood pressure also occurs through activation of sensory receptors known as baroreceptors or pressure receptors located in the carotid sinus, aortic arch, venae cavae, and pulmonary veins.

If heart rate increases, cardiac output increases (up to a point) even if there is no change in stroke volume. However, rapid heart rates decrease the amount of time available for ventricular filling during diastole. Cardiac output then falls because decreased filling time decreases stroke volume. Coronary artery perfusion also decreases because the coronary arteries fill primarily during diastole. Cardiac output decreases during bradycardia if stroke volume stays the same, because the number of cardiac cycles is decreased.

Contractility


Contractility is the inherent capability of the cardiac muscle fibers to shorten. Poor contractility of the heart muscle reduces the forward flow of blood from the heart, increases the ventricu-

lar pressures from accumulation of blood volume, and reduces cardiac output. Increased contractility may stress the heart.

Preload

Preload is the amount of cardiac muscle fiber tension, or stretch, that exists at the end of diastole, just before contraction of the ventricles. Preload is influenced by venous return and the compliance of the ventricles. It is related to the total volume of blood in the ventricles: The greater the volume, the greater the stretch of the cardiac muscle fibers, and the greater the force with which the fibers contract to accomplish emptying. This principle is called Starling's law of the heart.

This mechanism has a physiologic limit. Just as continuous overstretching of a rubber band causes the band to relax and lose its ability to recoil, overstretching of the cardiac muscle fibers eventually results in ineffective contraction. Disorders such as renal disease and congestive heart failure result in sodium and water retention and increased preload. Vasoconstriction also increases venous return and preload.


Too little circulating blood volume results in a decreased venous return and therefore a decreased preload. A decreased preload reduces stroke volume and thus cardiac output. Decreased preload may result from hemorrhage or maldistribution of blood volume, as occurs in third spacing (see Chapter 10 ).

Afterload

Afterload is the force the ventricles must overcome to eject their blood volume. It is the pressure in the arterial system ahead of the ventricles. The right ventricle must generate enough tension to open the pulmonary valve and eject its volume into the low-pressure pulmonary arteries. Right ventricle afterload is measured as pulmonary vascular resistance (PVR). The left ventricle, in contrast, ejects its load by overcoming the pressure behind the aortic valve. Afterload of the left ventricle is measured as systemic vascular resistance (SVR). Arterial pressures are much higher than pulmonary pressures; thus, the left ventricle has to work much harder than the right ventricle.

Alterations in vascular tone affect afterload and ventricular work. As the pulmonary or arterial blood pressure increases (e.g., through vasoconstriction), PVR and/or SVR increases, and the work of the ventricles increases. As workload increases, consumption of myocardial oxygen also increases. A compromised heart cannot effectively meet this increased oxygen demand, and a vicious cycle ensues. By contrast, a very low afterload decreases the forward flow of blood into the systemic circulation and the coronary arteries.

Clinical Indicators of Cardiac Output

For many critically ill clients, invasive hemodynamic monitoring catheters are used to measure cardiac output in quantifiable numbers. However, advanced technology is not the only way to identify and assess compromised blood flow. Because cardiac output perfuses the body's tissues, clinical indicators of low cardiac output may be manifested by changes in organ function that result from compromised blood flow. For example, a decrease in blood flow to the brain presents as a change in level of consciousness. Other manifestations of decreased cardiac output are discussed in Chapters 10 and 31 .

Cardiac index (CI) is the cardiac output adjusted for the client's body size, also called the client's body surface area (BSA). Because it takes into account the client's BSA, the cardiac index provides more meaningful data about the heart's ability to perfuse the tissues and therefore is a more accurate indicator of the effectiveness of the circulation.

BSA is stated in square meters (m^2), and cardiac index is calculated as CO divided by BSA. Cardiac measurements are considered adequate when they fall within the range of 2.5 to 4.2 L/min/ m^2 . For example, two clients are determined to have a cardiac output of 4 L/min. This parameter is within normal limits. However, one client is 5 feet, 2 inches (157 cm) tall and weighs 120 lb (54.5 kg), with a BSA of 1.54 m^2 . This client's cardiac index is $4 \div 1.54$, or 2.6 L/min/ m^2 . The second client is 6 feet, 2 inches (188 cm) tall and weighs 280 lb (81.7 kg), with a BSA of 2.52 m^2 . This client's cardiac index is $4 \div 2.52$, or 1.6 L/min/ m^2 . The cardiac index results show that the same cardiac output of 4 L/min is adequate for the first client but grossly inadequate for the second client.

The Conduction System of the Heart

The cardiac cycle is perpetuated by a complex electrical circuit commonly known as the intrinsic conduction system of the heart. Cardiac muscle cells possess an inherent characteristic of self-excitation, which enables them to initiate and transmit impulses independent of a stimulus. However, specialized areas of myocardial cells typically exert a controlling influence in this electrical pathway.

One of these specialized areas is the sinoatrial (SA) node, located at the junction of the superior vena cava and right atrium (Figure 30-7 ■). The SA node acts as the normal "pacemaker"

of the heart, usually generating an impulse 60 to 100 times per minute. This impulse travels across the atria via internodal pathways to the atrioventricular (AV) node, in the floor of the interatrial septum. The very small junctional fibers of the AV node slow the impulse, slightly delaying its transmission to the ventricles. It then passes through the bundle of His at the atrioventricular junction and continues down the interventricular septum through the right and left bundle branches and out to the Purkinje fibers in the ventricular muscle walls.

This path of electrical transmission produces a series of changes in ion concentration across the membrane of each cardiac muscle cell. The electrical stimulus increases the permeability of the cell membrane, creating an action potential (electrical potential). The result is an exchange of sodium, potassium, and calcium ions across the cell membrane, which changes the intracellular electrical charge to a positive state. This process of depolarization results in myocardial contraction. As the ion exchange reverses and the cell returns to its resting state of electronegativity, the cell is repolarized, and cardiac muscle relaxes. The cellular action potential serves as the basis for electrocardiography (ECG), a diagnostic test of cardiac function.

The Action Potential

Movement of ions across cell membranes causes the electrical impulse that stimulates muscle contraction. This electrical activity, called the *action potential*, produces the waveforms represented on ECG strips.

In the resting state, positive and negative ions align on either side of the cell membrane, producing a relatively negative charge within the cell and a positive extracellular charge (Figure 30-8 ■). The cell is said to be polarized. The negative resting membrane

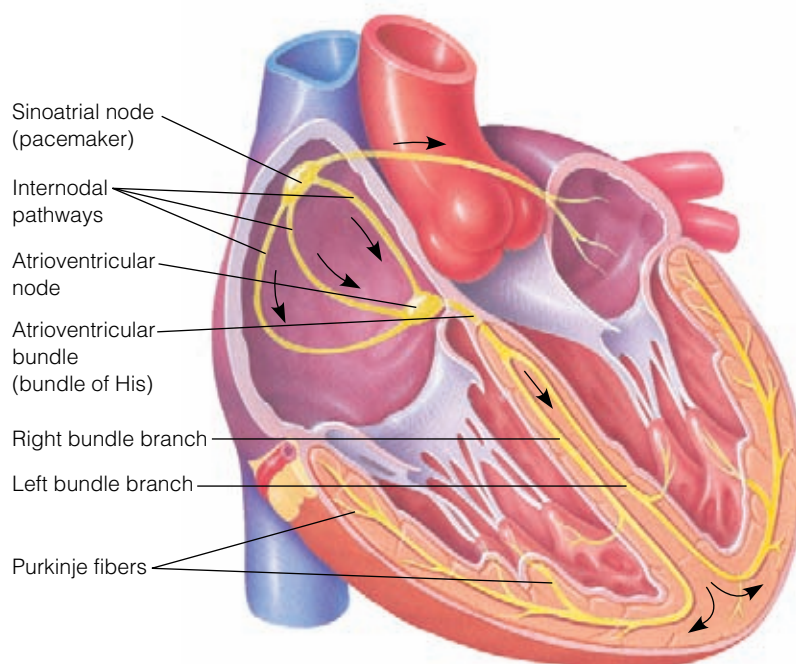


Figure 30-7 ■ The intrinsic conduction system of the heart.

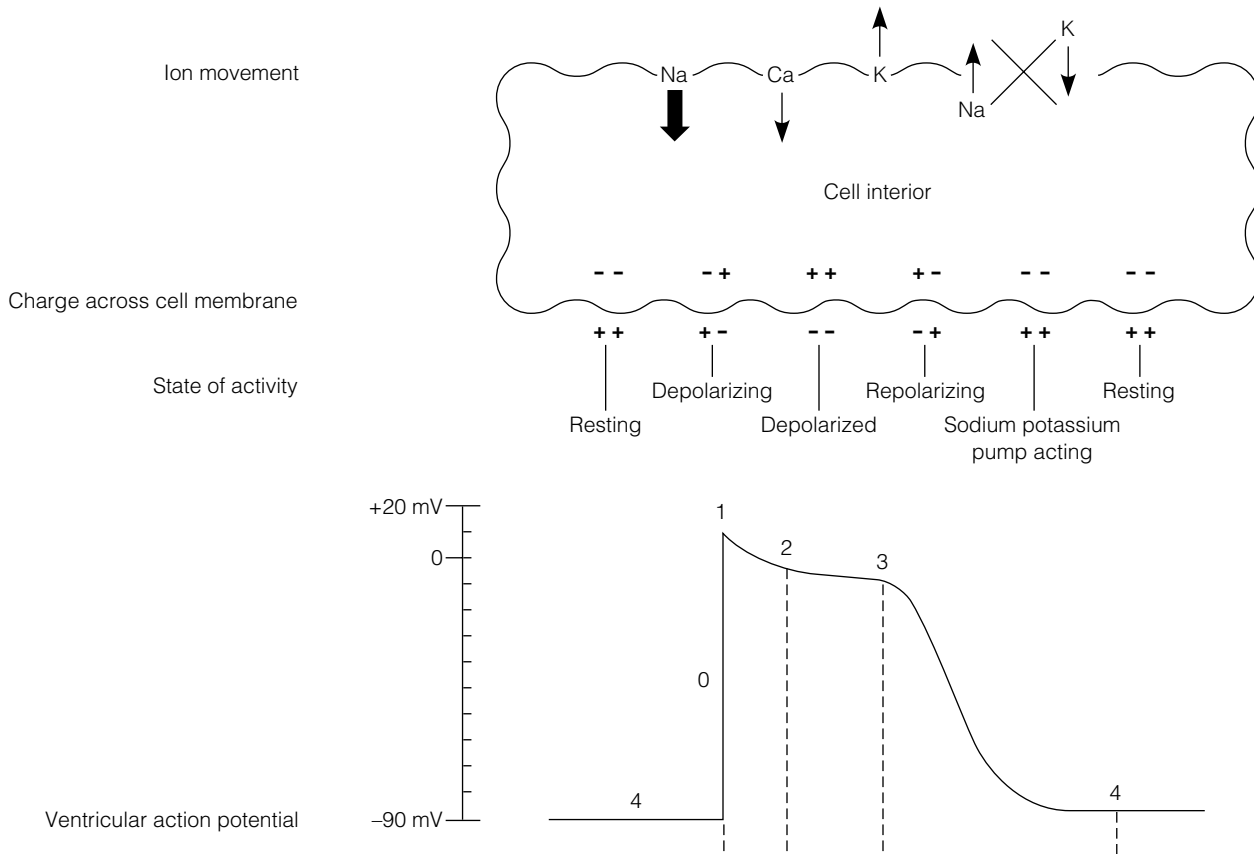


Figure 30-8 ■ Action potential of a cardiac muscle cell. In the resting state (phase 4), the cell membrane is polarized; the cell's interior has a negative charge compared to that of extracellular fluid. On depolarization (phase 0), sodium ions diffuse rapidly across the cell membrane into the cell, and calcium channels open. In the fully depolarized state (phase 1), the cell's interior has a net positive charge compared to its exterior. During the plateau period (phase 2), calcium moves into the cell and potassium diffusion slows, prolonging the action potential. In phase 3, calcium channels close, the sodium-potassium pump removes sodium from the cell, and the cell membrane again becomes polarized with a net negative charge.

potential is maintained at about -90 millivolts (mV) by the sodium-potassium pump in the cell membrane.

Depolarization

Two types of ion channels function to produce the electrical changes that occur during the depolarization phase: the fast sodium channels and the slow calcium channels. A fast action potential occurs in atrial and ventricular muscle cells and the Purkinje conduction system and uses the fast sodium channels. The slow type occurs in the SA and AV nodes, which use the slow calcium channels. The action potential for contraction of the heart is initiated in the SA node. When a resting cell is stimulated by an electrical charge from a neighboring cell or by a spontaneous event, its cell membrane permeability changes. Sodium ions enter the cell, and the membrane becomes less permeable to potassium ions. Addition of positively charged ions to intracellular fluid changes the membrane potential from negative to slightly positive at $+20$ to $+30$ mV. This change in the electrical charge across the cell membrane is called depolarization.

As the cell becomes more positive, it reaches a point called the threshold potential. When the threshold potential is

reached, an action potential is generated. The response to the action potential in the myocardial muscle cells causes a chemical reaction of calcium within the cell. This, in turn, causes actin and myosin filaments to slide together, producing cardiac muscle contraction. The action potential spreads to surrounding cells, causing a coordinated muscle contraction. As soon as the myocardium is completely depolarized, repolarization begins.

Repolarization

Repolarization returns the cell to its resting, polarized state. During rapid repolarization, fast sodium channels close abruptly, and the cell begins to regain its negative charge. During the plateau phase, muscle contraction is prolonged as slow calcium-sodium channels remain open. When these channels close, the sodium-potassium pump restores ion concentration to normal resting levels. The cell membrane is then polarized, ready for the cycle to start again. Each heartbeat represents one cardiac cycle, with one depolarization and repolarization cycle and one complete cardiac muscle contraction and relaxation (systole and diastole).

Normally, only pacemaker cells demonstrate automaticity. Pacemaker cells have a resting potential that is much less neg-

ative (-70 to -50 mV) than other cardiac muscle cells. Their threshold potential also is lower than that of other myocardial cells. These differences result from constant leakage of sodium and potassium ions into the cell.

Myocardial cells have a unique protective property, the refractory period, during which they resist stimulation. This property protects cardiac muscle from spasm and tetany. During the absolute refractory period, depolarization will not occur no matter how strongly the cell is stimulated. It is followed by the relative refractory period, during which a greater than normal stimulus is required to generate another action potential. During the supernormal period that follows, a mild stimulus will cause depolarization. Many cardiac dysrhythmias are triggered during the relative refractory and supernormal periods.

ASSESSING CARDIAC FUNCTION

Cardiac function is assessed by findings from diagnostic tests, a health assessment interview to collect subjective data, and a physical assessment to collect objective data. Sample documentation of an assessment of cardiac function is included in the box below.

Diagnostic Tests

The results of diagnostic tests of cardiac function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease, and to help nurses monitor the client's responses to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the heart are described on pages 944–946 and summarized in the following bulleted list. More information is included in the discussion of specific disorders in Chapters 31 and 32 ∞.

- The primary test used to identify the risk of coronary artery disease (CAD) or to monitor treatment for alterations in lipid levels is a measurement of lipid components of cholesterol, triglycerides, and lipoproteins in the blood.
- Noninvasive tests of cardiac structure and function include a chest x-ray and stress/exercise tests. The treadmill test is the most basic exercise test, with diagnostic ability to measure cardiac perfusion enhanced by administering IV ra-

dioisotopes during the test. A treadmill exercise test is often combined with other tests to evaluate cardiac function under stress. The exercise thallium or technetium test is probably the most useful noninvasive test to monitor and diagnose CAD.

- Abnormal areas of the heart may be identified and evaluated by an MRI to locate areas of myocardial infarction, a CT scan to quantify calcium deposits in coronary arteries, or a PET test to evaluate myocardial perfusion and myocardial metabolic function.
- Echocardiograms are conducted in conjunction with Dopplers and color flow imaging to produce audio and graphic data about the motion, wall thickness, and chamber size of the heart and of the blood flow and velocity.
- A transesophageal echocardiogram (TEE) allows visualization of structures adjacent to the esophagus to visualize cardiac and extracardiac structures, including mitral valve and aortic valve pathology, left atrium intracardiac thrombosis, acute dissection of the aorta, endocarditis, and ventricular function during and after surgery.
- A cardiac catheterization with either coronary angiography or coronary arteriography may be performed to identify CAD or cardiac valvular disease, to determine pulmonary artery or heart chamber pressures, to obtain a myocardial biopsy, to evaluate artificial valves, or to do angioplasty or stent of an area of CAD.
- Pericardiocentesis is a procedure done to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. It may also be an emergency procedure to treat cardiac tamponade.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for assessing for medication use that may affect the outcome of the tests, for supporting the client during the examination as necessary, for documenting the procedures as appropriate, and for monitoring the results of the tests.

Genetic Considerations

When conducting a health assessment interview and physical assessment, it is important for the nurse to consider genetic influences on health of the adult. During the health assessment interview, ask about family members with health problems affecting cardiac function, or of a family history of high cholesterol levels or early onset coronary artery disease. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the box on page 950). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counseling and evaluation. Chapter 8 ∞ provides further information about genetics in medical-surgical nursing.

The Health Assessment Interview

A health assessment interview to determine problems with cardiac structure and function may be conducted during a health screening, may focus on a chief complaint (such as

SAMPLE DOCUMENTATION Assessment of Cardiac Function

56-year-old male admitted to cardiac critical care unit from ED to rule out myocardial infarction. States he has pain in the middle of his chest that is "like a heavy pressure"; 6 on a 10-point scale. Skin cool, slightly moist. BP 190/94 right arm and 186/92 left arm (both reclining). Apical pulse 92, regular and strong. No pulse deficit. Respirations 28. Apical impulse non-palpable, no visible heaves or thrusts. S_1 and S_2 auscultated without murmurs or clicks. S_4 noted.


DIAGNOSTIC TESTS of Cardiac Disorders
NAME OF TEST Lipids

PURPOSE AND DESCRIPTION Blood lipids are cholesterol, triglycerides, and phospholipids. They circulate bound to proteins, and so are known as lipoproteins. Lipids are measured to evaluate risk for CAD and to monitor effectiveness of anti-cholesterol medications.

Normal values:

Cholesterol: 140–200 mg/dL

Triglycerides: 40–190 mg/dL

HDL: Men=37–70 mg/dL

Women=40–88 mg/dL

LDL: <130 mg/dL

(Note: Normal values may vary by laboratory.)

RELATED NURSING CARE Cholesterol levels alone may be measured at any time of the day, regardless of food or fluid intake. When measuring triglycerides and lipoproteins (HDL and LDL), fasting for 12 hours (except for water) with no alcohol intake for 24 hours prior to the test is recommended.

NAME OF TEST Electrocardiogram (ECG)

PURPOSE AND DESCRIPTION See Boxes 30–1 and 30–2.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Chest x-ray

PURPOSE AND DESCRIPTION An x-ray of the thorax can illustrate the contours, placement, and chambers of the heart. It

may be done to identify heart displacement or hypertrophy, or fluid in the pericardial sac.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Stress/exercise tests

■ Treadmill test

PURPOSE AND DESCRIPTION Stress testing is based on the theory that CAD results in depression of the ST segment with exercise. Depression of the ST segment and depression or inversion of the T wave indicates myocardial ischemia. When the client is walking on a treadmill machine, the work rate of the heart is changed every 3 minutes for 15 minutes by increasing the speed and degree of incline by 3% each time. Clients

exercise until they are fatigued, develop symptoms, or reach their maximum predicted heart rate.

RELATED NURSING CARE *For all stress/exercise tests:* Ask the client to wear comfortable shoes, and to avoid food, fluids, and smoking for 2 to 3 hours before the test, assess for events that contraindicate the tests: recent myocardial infarction; severe, unstable angina; controlled dysrhythmias; congestive heart failure; or recent pulmonary embolism.

NAME OF TEST Thallium/technetium stress test (myocardial imaging perfusion test, cardiac blood pool imaging)**PURPOSE AND DESCRIPTION** *Thallium stress test:*

Thallium-201, a radioisotope that accumulates in myocardial cells, is used during the stress test to evaluate myocardial perfusion. Second scans are done 2 to 3 hours later when the heart is at rest; this is to differentiate between an ischemic area and an infarcted or scarred area of myocardium.

Exercise technetium perfusion test: Technetium 99m-laced compounds are administered and a scan is done to evaluate

cardiac perfusion, wall motion, and ejection fraction. This is probably the most useful noninvasive test to diagnose and monitor CAD.

RELATED NURSING CARE Assess medications; those that affect the blood pressure or heart rate should be discontinued for 24 to 36 hours prior to the test (unless the test is being done to monitor the effectiveness of the medications).

NAME OF TEST Nuclear persantine [dipyridamole] stress test

PURPOSE AND DESCRIPTION This test is used when the client is not physically able to walk on the treadmill. Persantine, given IV, dilates the coronary arteries and increases myocardial blood flow. Coronary arteries that are narrowed from CAD cannot dilate to increase myocardial perfusion.

RELATED NURSING CARE Client is NPO after midnight except for water. Food, fluids, and drugs that contain caffeine should be avoided for 24 hours prior to the test, as should decaffeinated fluids. Some drugs, such as theophylline preparations, are discontinued for 36 hours prior to the test.

NAME OF TEST Nuclear dobutamine stress test

PURPOSE AND DESCRIPTION Dobutamine is an adrenergic drug that increases myocardial contractility, heart rate, and systolic blood pressure, which increases coronary oxygen consumption and thus increases coronary blood flow.

RELATED NURSING CARE Client is NPO after midnight except for water. Discontinue beta-blockers, calcium channel blockers, and ACE inhibitors for 36 hours prior to the test. Do not administer nitrates for 6 hours prior to the test.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION An MRI may be used to identify and locate areas of myocardial infarction.

RELATED NURSING CARE Assess for any metallic implants (such as pacemaker, body piercing, or artificial joint), which would contraindicate the test.


DIAGNOSTIC TESTS of Cardiac Disorders (continued)

NAME OF TEST Computed tomography (CT) scan

PURPOSE AND DESCRIPTION A CT scan may be conducted to quantify calcium deposits in coronary arteries.

RELATED NURSING CARE Assess for allergy to iodine or seafood if contrast medium is to be administered.

NAME OF TEST Cardiolite scan

PURPOSE AND DESCRIPTION Used to evaluate blood flow in different parts of the heart. Cardiolite (technetium 99m sestamibi) is injected IV. In a dipyridamole cardiolite scan, dipyridamole (Persantine) is injected to increase blood flow to

coronary arteries. These scans may be done in conjunction with a treadmill test.

RELATED NURSING CARE See information in this table for treadmill test. Instruct the client to avoid intake of caffeine for 12 hours before having a test with dipyridamole cardiolite.

NAME OF TEST Positron emission tomography (PET)

PURPOSE AND DESCRIPTION Two scans are performed following injection of radionuclides, and the resulting images compared for myocardial perfusion and myocardial metabolic function. A stress test (treadmill) may be a part of the test. If the myocardium is ischemic or damaged, the images will be different. Normally, the images will be the same.

RELATED NURSING CARE Assess client's blood glucose: For accurate metabolic activity images, the blood glucose level must be between 60 and 140 mg/dL. If exercise is included in the test, the client will need to be NPO and avoid smoking and caffeine for 24 hours prior to the test.

NAME OF TEST Blood pool imaging

PURPOSE AND DESCRIPTION Following intravenous injection of technetium 99m pertechnetate, sequential evaluation of the heart can be performed for several hours.

Useful for evaluation of cardiac status following myocardial infarction and congestive heart failure and effectiveness of cardiac medications. Can be done at the client's bedside.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Echocardiogram

- M-mode
- Two-dimensional (2-D)
- Cardiac Doppler
- Color Doppler
- Stress echocardiogram

PURPOSE AND DESCRIPTION Echocardiograms use a transducer to record waves that are bounced off the heart, and to record the direction and flow of blood through the heart in audio and graphic data. An *M(motion)-mode echocardiogram*

records the motion, wall thickness, and chamber size of the heart. A *2-D echocardiogram* provides a cross-sectional view of the heart. *Color flow imaging* combines 2-D echocardiography and Doppler technology to evaluate the speed and direction of blood flow through the heart, which can identify pathology such as leaky valves. *Stress echocardiography* combines a treadmill test with ultrasound images to evaluate segmental function and wall motion. If the client is not physically able to exercise, IV dobutamine may be administered and ultrasound images taken.

RELATED NURSING CARE No special preparation is needed; see related nursing care for the client having a treadmill test for a stress echocardiogram.

NAME OF TEST Transesophageal echocardiography (TEE)

PURPOSE AND DESCRIPTION Allows visualization of adjacent cardiac and extracardiac structures to identify or monitor mitral and aortic valve pathology, left atrium intracardiac thrombus, acute dissection of the aorta, endocarditis,

perioperative left ventricular function, and intracardiac repairs during surgery. A transducer (probe) attached to an endoscope is inserted into the esophagus, and images are taken. Concurrent IV contrast medium, Doppler ultrasound, and color flow imaging may be used.

NAME OF TEST Cardiac catheterization (coronary angiography, coronary arteriography)

PURPOSE AND DESCRIPTION A cardiac catheterization may be performed to identify CAD or cardiac valvular disease, to determine pulmonary artery or heart chamber pressures, to obtain a myocardial biopsy, to evaluate artificial valves, or to perform angioplasty or stent an area of CAD. The test is performed by inserting a long catheter into a vein or artery (depending on whether the right side or the left side of the heart is being examined) in the arm or leg. Using fluoroscopy, the catheter is then threaded to the heart chambers or coronary arteries or both. Contrast dye is injected and heart structures are visualized and heart activity is filmed. The test is done for diagnosis and before heart surgery.

inferior vena cava into the right atrium to the pulmonary artery. Pressures are measured at each site and blood samples can be obtained for the right side of the heart. The functions of the tricuspid and pulmonary valves can be observed.

Left cardiac catheterization: The catheter is inserted into the brachial or femoral artery and advanced retrograde through the aorta to the coronary arteries and/or left ventricle. The patency of the coronary arteries and/or functions of the aortic and mitral valves and left ventricle can be observed.

NURSING CARE: CARDIAC CATHETERIZATION

Before the Procedure

- Explain the procedure to the client.
- No food or fluids are allowed for 6 to 8 hours before the test.

Right cardiac catheterization: The catheter is inserted into the femoral vein or antecubital vein and then threaded through the

(continued)

DIAGNOSTIC TESTS of Cardiac Disorders (continued)

- Assess for allergies to seafood, iodine, or iodine contrast dyes (if previous tests have been done). If an allergic response to the dye is possible, antihistamines (such as Benadryl) or steroids may be administered the evening before and the morning of the test.
- Assess for use of aspirin or NSAIDs (risk of bleeding), Viagra (risk of heart problems), or history of kidney disease (dye used may be toxic to the kidneys).
- Discontinue oral anticoagulant medications. Heparin may be ordered to prevent thrombi.
- An IV of 5% D₅W is started at a keep-vein-open rate (to be available if emergency drugs have to be administered).
- Establish baseline of peripheral pulses.
- Take and record baseline vital signs.

Procedure

- Client is positioned on a padded table that tilts. A local anesthetic is used at the site of catheter insertion. ECG leads are applied and vital signs are monitored during the procedure.

NAME OF TEST Pericardiocentesis

PURPOSE AND DESCRIPTION This procedure is performed to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. It may also be done as an emergency procedure for the client with cardiac tamponade (which may result in death). A large-gauge (16 to 18) needle is inserted to the left of the xiphoid process into the pericardial sac and excess fluid is withdrawn (see Figure 30–9 ■). The needle is attached to an ECG lead to help determine if the needle is touching the epicardial surface, thus preventing piercing of the myocardium.

NURSING CARE: PERICARDIOCENTESIS

Before the Procedure

- Gather all supplies:
 - a. Pericardiocentesis tray
 - b. ECG machine and electrode patches
 - c. Emergency cart with defibrillator
 - d. Dressing
 - e. Culture bottles (if indicated)
- Reinforce teaching and answer questions about the procedure or associated care. Provide emotional support.
- Ensure that informed consent has been obtained.
- Provide for privacy.
- Obtain and document baseline vital signs.
- Connect the client to a cardiac monitor; obtain a baseline rhythm strip for comparison during and after the procedure.
- Connect the precordial ECG lead of the hub of the aspiration needle using an alligator clamp.

During the Procedure

- Follow standard precautions.
- Position seated at a 45- to 60-degree angle. Place a dry towel under the rib cage to catch blood or fluid leakage.
- Observe the ST segment for elevation and the ECG monitor for signs of myocardial irritability (PVCs) during the procedure. These indicate that the needle is touching the myocardium and should be withdrawn slightly.

The client lies supine and is asked to cough and deep breathe frequently. The procedure takes 1/2 to 3 hours.

- Tell the client that a hot, flushing sensation may be felt for a minute or two when the dye is injected.

After the Procedure

- Monitor vital signs every 15 minutes for the first hour and then every 30 minutes until stable. Assess cardiac rhythm and rate for alterations. Assess peripheral pulses distal to the insertion site.
- Assess client for complaints of chest heaviness, shortness of breath, and abdominal or groin pain.
- Monitor catheter insertion site for bleeding or hematoma.
- Administer pain medications as prescribed.
- Instruct client to remain on bed rest for 6 to 12 hours (or as ordered). If a collagen-like plug was inserted after removal of the catheter, only a 2- to 3-hour bed rest is necessary.
- Encourage oral fluids unless contraindicated (i.e., if the client has congestive heart failure).

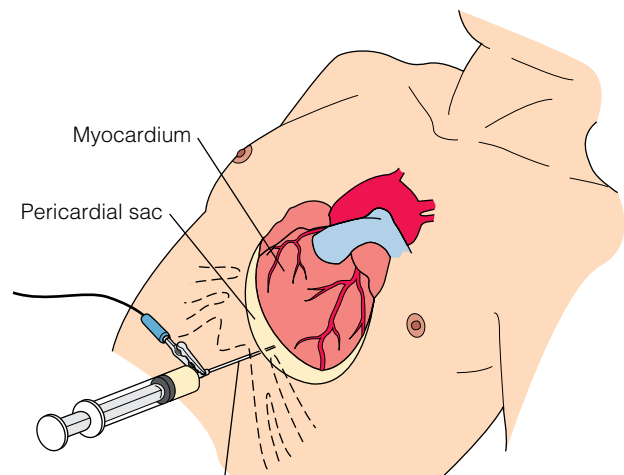


Figure 30–9 ■ Pericardiocentesis.

- Notify the physician of changes in cardiac rhythm, blood pressure, heart rate, level of consciousness, and urine output. These may indicate cardiac complications.
- Monitor central venous pressure (CVP) and blood pressure closely. As the effusion is relieved, CVP will decrease, and BP will increase.

After the Procedure

- Document the procedure and the client's response to and tolerance of the procedure.
- Continue to monitor vital signs and cardiac rhythm every 15 min during the first hour, every 30 min during the next hour, every hour for the next 24 hours.
- Record the amount of fluid removed as output on the intake and output record.
- If indicated, send a sample of aspirated fluid for culture and sensitivity and laboratory analysis.
- Assess heart and breath sounds.

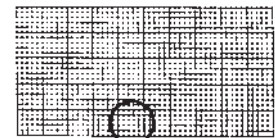
BOX 30–1 Electrocardiogram

The electrocardiogram (ECG) is a graphic record of the heart’s activity. Electrodes applied to the body surface are used to obtain a graphic representation of cardiac electrical activity. These electrodes detect the magnitude and direction of electrical currents produced in the heart. They attach to the electrocardiograph by an insulated wire called a lead. The electrocardiograph converts the electrical impulses it receives into a series of waveforms that represent cardiac depolarization and repolarization. Placement of electrodes on different parts of the body allows different views of this electrical activity, much like turning the head while holding a camera provides different views of the scenery. ECG waveforms and patterns are examined to detect dysrhythmias as well as myocardial damage, the effects of drugs, and electrolyte imbalances.

ECG waveforms reflect the direction of electrical flow in relation to a positive electrode. Current flowing toward the positive electrode produces an upward (positive) waveform; current flowing away from the positive electrode produces a downward (negative) waveform. Current flowing perpendicular to the positive pole produces a biphasic (both positive and negative) waveform. Absence of electrical activity is represented by a straight line called the isoelectric line.

ECG waveforms are recorded by a heated stylus on heat-sensitive paper. The paper is marked at standard intervals that represent time and voltage or amplitude (see Figure 1). Each small box is 1 mm². The recording speed of the standard ECG is 25 mm/second, so each small box represents 0.04 second. Five small boxes horizontally and vertically make one large box, equivalent to 0.20 second. Five large boxes represent 1 full second. Measured vertically, each small box represents 0.1 mV.

Both bipolar and unipolar leads are used in recording the ECG. A bipolar lead uses two electrodes of opposite polarity (negative and positive). In a *unipolar* lead, one positive electrode and a negative reference point at the center of the heart are used. The electrical potential between the two monitoring points is graphically recorded as the ECG waveform.



1 large box or 5 mm = 0.5 mV

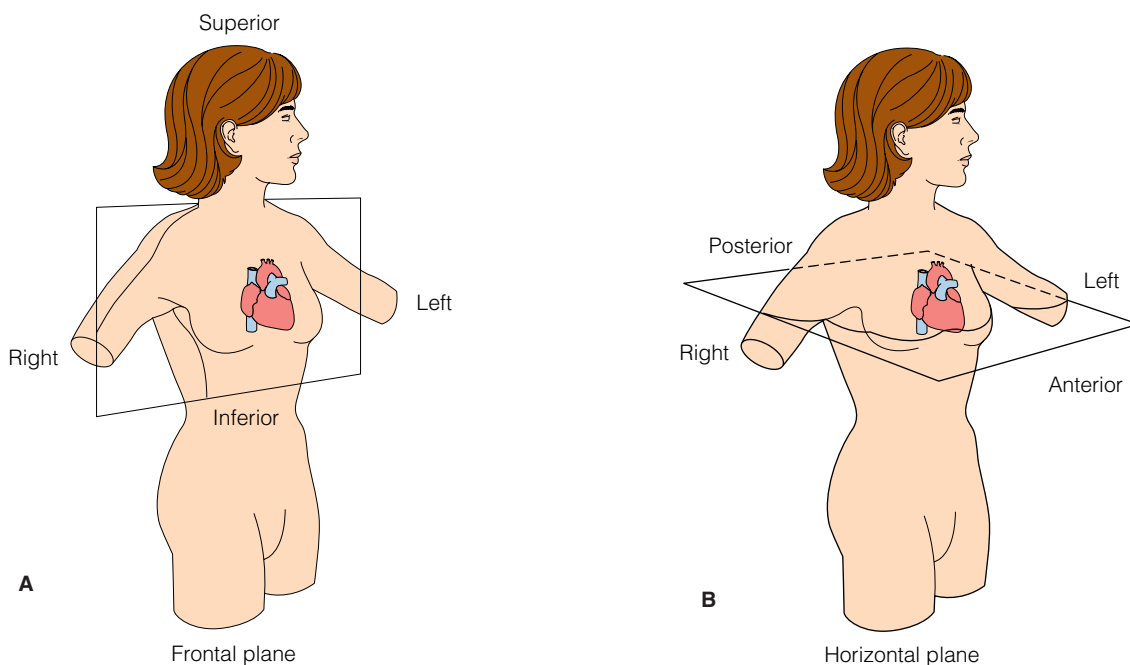
1 large box or 5 mm = 0.20 Second

1 small box or 1 mm = 0.04 Second

1 mm = 0.1 mV

1) Time and voltage measurements on ECG paper at a recording speed of 25 mm/second.

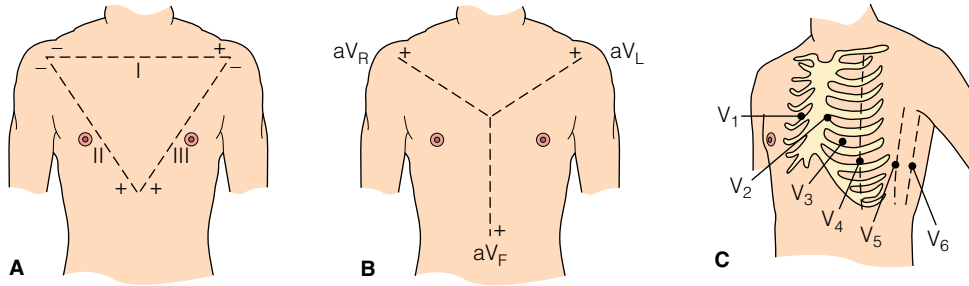
The heart can be viewed from both the frontal plane and the horizontal plane (see Figure 2). Each plane provides a unique perspective of the heart muscle. The frontal plane is an imaginary cut through the body that views the heart from top to bottom (superior–inferior) and side to side (right–left). This perspective of the heart is analogous to a paper doll cutout. It provides information about the inferior and lateral walls of the heart. The horizontal plane is a cross-sectional view of the heart from front to back (anterior–posterior) and side to side (right–left). Information regarding the anterior, septal, and lateral walls of the heart, as well as the posterior wall, are obtained from this view.



2) Planes of the heart. A, Frontal plane, B, horizontal plane.

(continued)

BOX 30–1 Electrocardiogram (continued)



3) Leads of the 12-lead ECG. A, Bipolar limb leads I, II, III; B, Unipolar limb leads aV_R, aV_L, aV_F; C, Unipolar precordial leads V₁ to V₆.

A standard 12-lead ECG provides a simultaneous recording of six limb leads and six precordial leads (see Figure 3). The limb leads provide information about the heart in the frontal plane and include three bipolar leads (I, II, III) and three unipolar leads (aV_R, aV_L, and aV_F). The bipolar limb leads measure electrical activity between a negative lead on one extremity and a positive lead on another. The unipolar limb leads (called augmented leads) measure the electrical activity between a single positive electrode on a limb (right arm [R], left arm [L], or left leg [F for foot]), and the center of the heart.

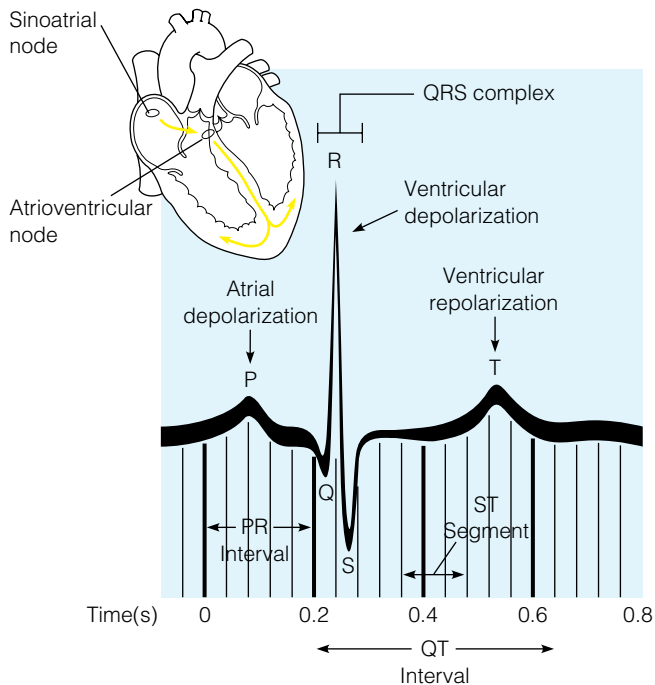
The precordial leads, also known as chest leads or V leads, view the heart in the horizontal plane. They include six unipolar leads (V₁, V₂, V₃, V₄, V₅, and V₆), which measure electrical activity between the center of the heart and a positive electrode on the chest wall.

The cardiac cycle is depicted as a series of waveforms, the P, Q, R, S, and T waves (see Figure 4).

- The *P wave* represents atrial depolarization and contraction. The impulse is from the sinus node. The P wave precedes the QRS

complex and is normally smooth, round, and upright. P waves may be absent when the SA node is not acting as the pacemaker. Atrial repolarization occurs during ventricular depolarization and usually is not seen on the ECG.

- The *PR interval* represents the time required for the sinus impulse to travel to the AV node and into the Purkinje fibers. This interval is measured from beginning of P wave to beginning of QRS complex. If no Q wave is seen, the beginning of the R wave is used. The PR interval is normally 0.12 to 0.20 second (up to 0.24 second is considered normal in clients over age 65). PR intervals greater than 0.20 second indicate a delay in conduction from the SA node to the ventricles.
- The *QRS complex* represents ventricular depolarization and contraction. The QRS complex includes three separate waves: The Q wave is the first negative deflection, the R wave is the positive or upright deflection, and the S wave is the first negative deflection after the R wave. Not all QRS complexes have all three waves; nonetheless, the complex is called a QRS complex. The normal duration of a QRS complex is from 0.06 to 0.10 second. QRS complexes greater than 0.10 second indicate delays in transmitting the impulse through the ventricular conduction system.
- The *ST segment* signifies the beginning of ventricular repolarization. The ST segment, the period from the end of the QRS complex to the beginning of the T wave, should be isoelectric. An abnormal ST segment is displaced (elevated or depressed) from the isoelectric line.
- The *T wave* represents ventricular repolarization. It normally has a smooth, rounded shape that is usually less than 10 mm tall. It usually points in the same direction as the QRS complex. Abnormalities of the T wave may indicate myocardial ischemia or injury, or electrolyte imbalances.
- The *QT interval* is measured from the beginning of the QRS complex to the end of the T wave. It represents the total time of ventricular depolarization and repolarization. Its duration varies with gender, age, and heart rate; usually, it is 0.32 to 0.44 second long. Prolonged QT intervals indicate a prolonged relative refractory period and a greater risk of dysrhythmias. Shortened QT intervals may result from medications or electrolyte imbalances.
- The *U wave* is not normally seen. It is thought to signify repolarization of the terminal Purkinje fibers. If present, the U wave follows the same direction as the T wave. It is most commonly seen in hypokalemia.

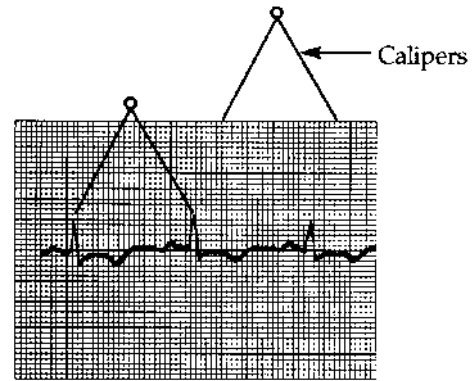


4) Normal ECG waveform and intervals.

BOX 30–2 Interpreting an ECG

Interpreting an ECG strip to determine the cardiac rhythm is a skill that takes practice to learn and master. Many methods are used to analyze ECGs. It is important to use a consistent method for ECG analysis. Identifying and interpreting complex dysrhythmias requires advanced skills and knowledge obtained through further training. One method follows:

- **Step 1: Determine rate.** Assess heart rate. Use P waves to determine the atrial rate and R waves for the ventricular rate. Several approaches can determine the heart rate.
 - Count the number of complexes in a 6-second rhythm strip (the top margin of ECG paper is marked at 3-second intervals), and multiply by 10. This provides an estimate of the rate and is particularly valuable if rhythms are irregular.
 - Count the number of large boxes between two consecutive complexes, and divide 300 (the number of large boxes in 1 minute) by this number. For example, there are 6 large boxes between two R waves; 300 divided by 6 equals a ventricular rate of 50 bpm. Memorize the following sequence for rapid rate determination: 300, 150, 100, 75, 60, 50, 43. One large box between complexes equals a rate of 300; two, a rate of 150; three, a rate of 100; and so on.
 - Count the number of small boxes between two consecutive complexes, and divide 1500 (the number of small boxes in 1 minute) by this number. For example, there are 19 small boxes between two R waves; 1500 divided by 19 equals a ventricular rate of 79 bpm. This is the most precise measurement of heart rate.
- **Step 2: Determine regularity.** Regularity is the consistency with which the P waves or QRS complexes occur. In a regular rhythm, all waves occur at a consistent rate. Rhythm regularity is determined by measuring the interval between consecutive waves. Place one point of an ECG caliper (a measuring device) on the peak of the P wave (for atrial rhythm) or the R wave (for ventricular rhythm). Adjust the other point to the peak of the next wave, P to P or R to R (see the figure in this box). Keeping the calipers set at this distance, evaluate intervals between consecutive waves. The rhythm is *regular* if all caliper points fall on succeeding wave peaks. Alternately, use a strip of blank paper on top of the ECG strip, marking the peaks of two or three consecutive waves. Then move the paper along the strip to consecutive waves. Wave peaks that vary by more than one to three small boxes (depending on the rate) are *irregular*. Irregular rhythms may be *irregularly irregular* (if the intervals have no pattern) or *regularly irregular* (if a consistent pattern to the irregularity can be identified).



- **Step 3: Assess P wave.** The presence or absence of P waves helps determine origin of the rhythm. All the P waves should be alike in size and shape (*morphology*). If P waves are not seen or they differ in shape, the rhythm may not originate in the sinus node.
- **Step 4: Assess P to QRS relationship.** Determine the relationship between P waves and QRS complexes. There should be one and only one P wave for every QRS complex, because the normal stimulus for ventricular contraction originates in the sinus node.
- **Step 5: Determine interval durations.** To evaluate impulse transmission through the cardiac conduction system, measure the PR interval, QRS duration, and QT interval. To measure, count the number of small boxes from the beginning of the interval to the end, and multiply by 0.04 second. Then determine whether the interval duration is within its normal limits. For example, the PR interval is 3.5 small boxes wide, or 0.14 second. This is within the normal limits of 0.12 to 0.20 second. This interval should be consistent, not varying from beat to beat. A PR interval greater than 0.20 second or one that varies from beat to beat is abnormal.

The QRS complex duration is normally between 0.06 and 0.10 second. A QRS complex greater than 0.12 second indicates delayed ventricular conduction.

The QT interval is normally 0.32 to 0.44 second. It varies inversely with the heart rate: The faster the heart rate, the shorter the QT interval. As a general rule, the QT interval should be no more than half the previous R–R interval. A prolonged QT interval indicates a prolonged relative refractory period of the heart.
- **Step 6: Identify abnormalities.** Note the presence and frequency of *ectopic* (extra) beats, deviation of the ST segment above or below the baseline, and abnormalities in waveform shape and duration.

chest pain), or may be part of a total health assessment. If the client has a problem with cardiac function, analyze its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the client:

- What is the location of the chest pain you experienced? Did it move up to your jaw or into your left arm?
- Describe the type of activity that brings on your chest pain.
- Have you noticed any changes in your energy level?

- Have you felt light-headed during the times your heart is racing?

The interview begins by exploring the client's chief complaint (e.g., chest pain, palpitations, or shortness of breath). Describe the client's chest pain in terms of location, quality or character, timing, setting or precipitating factors, severity, aggravating and relieving factors, and associated symptoms (Table 30–1).

Explore the client's history for heart disorders such as angina, heart attack, congestive heart failure (CHF), hypertension



GENETIC CONSIDERATIONS

Cardiac Disorders

- Familial hypercholesterolemia is a single gene disorder that results in atherosclerosis and CAD, which may occur at an earlier age than in the general population (i.e., before age 55 in men and age 65 in women). However, increased cholesterol levels may also be inherited and are a risk factor for CAD in both men and women.
- Marfan's syndrome is an autosomal-dominant inherited disorder that affects the skeleton, the eyes, and the cardiovascular system. The cardiovascular effects are a dilatation of the proximal aorta and aortic dissection associated with degeneration of the elastic fibers in the tunica media of the aorta. There may also be thoracic aortic aneurysms.
- Supraventricular aortic stenosis (SVAS) is a genetic vascular disorder resulting in an hourglass-shaped stenosis of the ascending aorta. It may also affect other major arteries, including the pulmonary, carotid, cerebral, renal, and coronary arteries.
- Hypertrophic cardiomyopathy, a disease of sarcomere proteins, has a genetic transmission.
- Williams syndrome is a rare genetic disorder characterized by characteristic "elfin-like" features and heart and blood vessel problems (as well as other physical problems).
- Long QT syndrome (LQTS) is an inherited genetic disorder that results from structural abnormalities of the potassium channels in the heart, leading to dysrhythmias. This can result in unconsciousness, and may cause sudden cardiac death in teenagers and young adults when exposed to stressors ranging from exercise to loud sounds.

(HTN), and valvular disease. Ask the client about previous heart surgery or illnesses, such as rheumatic fever, scarlet fever, or recurrent streptococcal throat infections. Also ask about the presence and treatment of other chronic illnesses such as diabetes mellitus, bleeding disorders, or endocrine disorders. Review the client's family history for CAD, HTN, stroke, hyperlipidemia, diabetes, congenital heart disease, or sudden death.

Ask the client about past or present occurrence of various cardiac symptoms, such as chest pain, shortness of breath, difficulty breathing, cough, palpitations, fatigue, light-headedness or dizziness, fainting, heart **murmur**, blood clots, or swelling. Because cardiac function affects all other body systems, a full history may need to explore other related systems, such as respiratory function and/or peripheral vascular function.

Review the client's personal habits and nutritional history, including body weight; eating patterns; dietary intake of fats, salt, fluids; dietary restrictions; hypersensitivities or intolerances to food or medication; and the use of caffeine and alcohol. If the client uses tobacco products, ask about type (cigarettes, pipe, cigars, snuff), duration, amount, and efforts to quit. If the client uses street drugs, ask about type, method of intake (e.g., inhaled or injected), duration of use, and efforts to quit. Include questions about the client's activity level and tolerance, recreational activities, and relaxation

TABLE 30–1 Assessing Chest Pain

CHARACTERISTIC	EXAMPLES
Location	Substernal, precordial, jaw, back Localized or diffuse Radiation to neck, jaw, shoulder, arm
Character/quality	Pressure; tightness; crushing, burning, or aching quality; heaviness; dullness; "heartburn" or indigestion
Timing: onset, duration, and frequency	Onset: Sudden or gradual? Duration: How many minutes does the pain last? Frequency: Is the pain continuous or periodic?
Setting/precipitating factors	Awake, at rest, sleep interrupted? With activity? With eating, exertion, exercise, elimination, emotional upset?
Intensity/severity	Can range from 0 (no pain) to 10 (worst pain ever felt)
Aggravating factors	Activity, breathing, temperature
Relieving factors	Medication (nitroglycerin, antacid), rest; there may be no relieving factors
Associated symptoms	Fatigue, shortness of breath, palpitations, nausea and vomiting, sweating, anxiety, light-headedness or dizziness

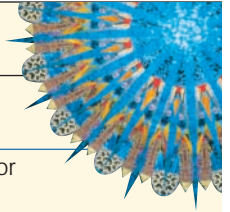
habits. Assess the client's sleep patterns for interruptions in sleep due to dyspnea, cough, discomfort, urination, or stress. Ask how many pillows the client uses when sleeping. Also consider psychosocial factors that may affect the client's stress level: What is the client's marital status, family composition, and role within the family? Have there been any changes? What is the client's occupation, level of education, and socioeconomic level? Are resources for support available? What is the client's emotional disposition and personality type? How does the client perceive his or her state of health or illness, and how able is the client to comply with treatment?

Interview questions categorized by functional health patterns are listed in the table on the next page.

Physical Assessment

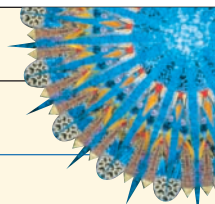
Physical assessment of cardiac function may be performed either as part of a total assessment or alone for clients with suspected or known problems with cardiac function. Assess the heart through inspection, palpation, and auscultation over the precordium (the area of the chest wall overlying the heart). Normal age-related findings for the older adult are summarized in Table 30–2. Before beginning the assessment, collect all required equipment and explain the techniques to the client to decrease anxiety. A quiet environment is essential to hear and assess heart sounds accurately.

The client may sit or lie in the supine position. Movements over the precordium may be more easily seen with tangential

FUNCTIONAL HEALTH PATTERN INTERVIEW The Cardiac System

Functional Health Pattern
Interview Questions and Leading Statements

Health Perception-Health Management	<ul style="list-style-type: none"> ■ Have you ever had any problems with your heart, such as angina (pain), heart attack, or disease of the valves? If so, describe. What was used to treat these problems? ■ Have you been diagnosed with high blood pressure? If so, how is it treated? ■ Do you have a history of rheumatic fever, scarlet fever, or strep throat infections? If so, describe them and their treatment. ■ Have you had your cholesterol checked recently? What is it? If you have high cholesterol, how is it treated? ■ Have you ever had tests to check the function of your heart? Describe them if so. ■ Do you take any medications to make your heart function more effectively, such as aspirin, those to control your heart rate, anticoagulants, or diuretics? How often do you take them? ■ Do you have a pacemaker? At what age and for what problem? How do you check the batteries? ■ Do you smoke, chew tobacco, or use snuff? If so, how often and how much? ■ Do you drink alcohol? If so, what type, how much, and for how long? ■ Are you able to manage your activities of daily living and work independently? Explain.
Nutritional-Metabolic	<ul style="list-style-type: none"> ■ Describe your food and liquid intake in a 24-hour period. How often do you eat fried foods, fast foods, or meat? ■ How much salt do you use on food? ■ Do you eat high-fiber foods? If so, what are they and how often? ■ Have you had a recent weight gain or loss? Explain. ■ Have you noticed any change in color of your skin; for example, pale or dusky or flushed? If so, do you know what causes this? ■ Have you had any swelling in your feet or legs? Where and how much? What do you do to relieve it? ■ Do you feel tired during the day? What do you do when you are tired?
Elimination	<ul style="list-style-type: none"> ■ Has a heart problem interfered with your usual bowel and bladder elimination? Explain.
Activity-Exercise	<ul style="list-style-type: none"> ■ Describe your usual activity in a 24-hour period. ■ Has there been any change in your ability, energy, or strength to perform your usual activities (such as bathing, cleaning house, yard work, shopping)? If so, explain. ■ Do you ever have to stop and rest while doing daily activities? Explain. ■ Do you notice shortness of breath with certain activities? If so, what are they? How long does this last? What do you do to breathe better? ■ Describe any cough you have had. Was it dry or wet? Do you cough up mucus? If so, what color is it? How long have you had the cough? ■ Have you experienced any numbness or tingling, dizziness or light-headedness, or palpitations? Describe if so. ■ Have you ever used oxygen?
Sleep-Rest	<ul style="list-style-type: none"> ■ How long do you sleep each night? Do you feel rested after you sleep? ■ Does your heart problem interfere with your ability to sleep and rest? Explain. ■ How many pillows do you use at night? ■ Where do you sleep at night (e.g., in a recliner to breathe more easily)? ■ Do you ever feel short of breath while you are resting or sleeping? Does this wake you up if so? Explain.
Cognitive-Perceptual	<ul style="list-style-type: none"> ■ Describe any chest pain you have experienced. When did it occur? Where was it located? On a scale of 0 to 10, with 10 being the worst pain you have ever had, rate the pain and describe it (for example, burning, crushing, stabbing, squeezing, heavy, tight). ■ What were you doing when the pain began, for example, were you working or resting? Did it begin suddenly or gradually? How long did it last? ■ Did you have any other symptoms with the pain, such as nausea or vomiting, sweating, racing heart, pale skin, palpitations? ■ What made the pain worse? What did you do to try to relieve the pain? Did that work?
Self-Perception-Self-Concept	<ul style="list-style-type: none"> ■ How does having this condition make you feel about yourself?
Role-Relationships	<ul style="list-style-type: none"> ■ How does this condition affect your relationships with others? ■ Has having this condition interfered with your ability to work? Explain.

(continued)

FUNCTIONAL HEALTH PATTERN INTERVIEW **The Cardiac System (continued)****Functional Health Pattern****Interview Questions and Leading Statements**

Sexuality-Reproductive	<ul style="list-style-type: none"> ■ Has this condition interfered with your usual sexual activity? ■ Have you ever had chest pain during sexual activity? What do you do for it? ■ Do you use a slower pace or different positions that are less stressful for you during sexual activities? Does this help?
Coping-Stress-Tolerance	<ul style="list-style-type: none"> ■ Has having this condition created stress for you? ■ Have you experienced any kind of stress that makes this condition worse? Explain. ■ Describe what you do when you feel stressed.
Value-Belief	<ul style="list-style-type: none"> ■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

lighting (in which the light is directed at a right angle to the area being observed, producing shadows). Assess the following types of movements:

- The **apical impulse** is a normal, visible pulsation (**thrust**) in the area of the midclavicular line in the left fifth intercostal space. It can be seen on inspection in about half of the adult population. (The apical impulse was previously called the point of maximal impulse [PMI] but this is no longer used because a maximal impulse may occur

in other areas of the precordium as a result of abnormal conditions.)

- **Retraction** is a pulling in of the tissue of the precordium; a slight retraction just medial to the midclavicular line at the area of the apical impulse is normal and is more likely to be visible in thin clients.
- **Pulsations** (other than the normal apical pulsations), which may be called **heaves** or **lifts**, are considered abnormal. They may occur as the result of an enlarged ventricle.

TABLE 30–2 Age-Related Cardiac Changes

AGE-RELATED CHANGE	SIGNIFICANCE
Myocardium: ↓ efficiency and contractibility. Sinus node: ↑ in thickness of shell surrounding the node, and a ↓ in the number of pacemaker cells Left ventricle: Slight hypertrophy, prolonged isometric contraction phase and relaxation time; ↑ time for diastolic filling and systolic emptying cycle. Valves and blood vessels: Aorta is elongated and dilated, valves are thicker and more rigid, and resistance to peripheral blood flow increases by 1% per year.	<ul style="list-style-type: none"> ■ Decreased cardiac output when under physiologic stress with resulting tachycardia that lasts longer. The person may require rest time between physical activities. ■ Stroke volume may increase to compensate for tachycardia, leading to increased blood pressure. ■ Blood pressure increases to compensate for increased peripheral resistance and decreased cardiac output.

CARDIAC ASSESSMENTS**Technique/Normal Findings****Abnormal Findings****Apical Impulse Assessment**

First using the palmar surface and then repeating with finger pads, palpate the precordium for symmetry of movement

- An enlarged or displaced heart is associated with an apical impulse lateral to the midclavicular line (MCL) or below the fifth left intercostal space (ICS).
- Increased size, amplitude, and duration of the apical impulse are associated with left ventricular volume overload (increased afterload) in conditions such as HTN and aortic

Technique/Normal Findings

and the apical impulse for location, size, amplitude, and duration. The sequence for palpation is shown in Figure 30–10 ■. To locate the apical impulse, ask the client to assume a left lateral recumbent position. Simultaneous palpation of the carotid pulse may also be helpful. *The apical impulse is not palpable in all clients. The apical impulse may be palpated in the mitral area, and has only a brief small amplitude.*

Palpate the subxiphoid area with the index and middle finger. *No pulsations or vibrations should be palpated.*

Cardiac Rate and Rhythm Assessment

Auscultate heart rate. *The heart rate should be 60 to 100 beats per minute with regular rhythm.*

Simultaneously palpate the radial pulse while listening to the apical pulse. *The radial and apical pulses should be equal.*

Auscultate heart rhythm. *The heart rhythm should be regular.*

Abnormal Findings

stenosis, and with pressure overload (increased preload) in conditions such as aortic or mitral regurgitation.

- Increased amplitude alone may occur with hyperkinetic states, such as anxiety, hyperthyroidism, and anemia.
- Decreased amplitude is associated with a dilated heart in cardiomyopathy.
- Displacement alone may also occur with dextrocardia, diaphragmatic hernia, gastric distention, or chronic lung disease.
- A **thrill** (a palpable vibration over the precordium or an artery) may accompany severe valve stenosis.
- A marked increase in amplitude of the apical impulse at the right ventricular area occurs with right ventricular volume overload in atrial septal defect.
- An increase in amplitude and duration occurs with right ventricular pressure overload in pulmonic stenosis and pulmonary hypertension. A lift or heave may also be seen in these conditions (and in chronic lung disease).
- A palpable thrill in this area occurs with ventricular septal defect.
- Right ventricular enlargement may produce a downward pulsation against the fingertips.
- An accentuated pulsation at the pulmonary area may be present in hyperkinetic states.
- A prominent pulsation reflects increased flow or dilation of the pulmonary artery.
- A thrill may be associated with aortic or pulmonary stenosis, aortic stenosis, pulmonary HTN, or atrial septal defect.
- Increased pulsation at the aortic area may suggest aortic aneurysm.
- A palpable second heart sound (S_2) may be noted with systemic HTN.

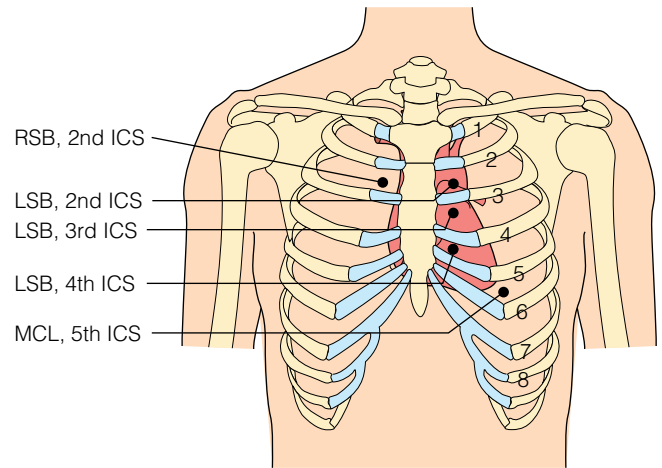


Figure 30–10 ■ Areas for inspection and palpation of the precordium, indicating the sequence for palpation.

- A heart rate exceeding 100 beats per minute (beats/min) is tachycardia. A heart rate less than 60 beats/min is bradycardia.

- If the radial pulse falls behind the apical rate, the client has a pulse deficit, indicating weak, ineffective contractions of the left ventricle.

- **Dysrhythmias** (abnormal heart rate or rhythm) may be regular or irregular in rhythm; their rates may be slow or fast. Irregular rhythms may occur in a pattern (e.g., an early beat every second beat, called bigeminy), sporadically, or with frequency and disorganization (e.g., atrial fibrillation). A pattern of gradual increase and decrease in heart rate that is within normal heart rate and that correlates with inspiration and expiration is called sinus arrhythmia.



Technique/Normal Findings Abnormal Findings

Heart Sounds Assessment

See guidelines for cardiac auscultation in Box 30–3.

BOX 30–3 Guidelines for Cardiac Auscultation

1. Locate the major auscultatory areas on the precordium (see Figure 30–11).
2. Choose a sequence of listening. Either begin from the apex and move upward along the sternal border to the base, or begin at the base and move downward to the apex. One suggested sequence is shown in Figure 30–11.
3. Listen first with the client in the sitting or supine position. Then ask the client to lie on the left side, and focus on the apex. Lastly, ask the client to sit up and lean forward. These position changes bring the heart closer to the chest wall and enhance auscultation. Carry out the following steps when the client assumes each of these positions:
 - a. First, auscultate each area with the diaphragm of the stethoscope to listen for high-pitched sounds: S_1 , S_2 , murmurs, pericardial friction rubs.
 - b. Next, auscultate each area with the bell of the stethoscope to listen for lower pitched sounds: S_3 , S_4 , murmurs.
 - c. Listen for the effect of respirations on each sound; while the client is sitting up and leaning forward, ask the client to exhale and hold the breath while you listen to heart sounds.

Identify S_1 (first heart sound) and note its intensity. At each auscultatory area, listen for several cardiac cycles. See Figure 30–11 ■ for auscultation areas. S_1 is loudest at the apex of the heart.

- An accentuated S_1 occurs with tachycardia, states in which cardiac output is high (fever, anxiety, exercise, anemia, hyperthyroidism), complete heart block, and mitral stenosis.
- A diminished S_1 occurs with first-degree heart block, mitral regurgitation, CHF, CAD, and pulmonary or systemic HTN. The intensity is also decreased with obesity, emphysema, and pericardial effusion. Varying intensity of S_1 occurs with complete heart block and grossly irregular rhythms.

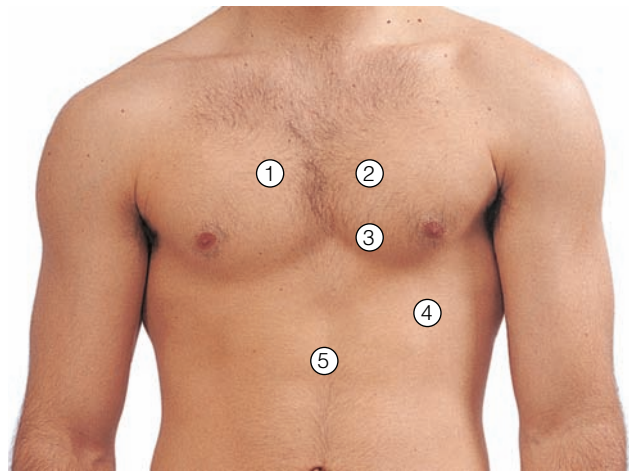


Figure 30–11 ■ Areas for auscultation of the heart.

Listen for splitting of S_1 .
Splitting of S_1 may occur during inspiration.

- Abnormal splitting of S_1 may be heard with right bundle branch block and premature ventricular contractions.

Identify S_2 (second heart sound) and note its intensity.
 S_2 immediately follows S_1 and is loudest at the base of the heart.

An accentuated S_2 may be heard with HTN, exercise, excitement, and conditions of pulmonary HTN such as CHF and cor pulmonale.

Listen for splitting of S_2 .
No splitting of S_2 should be heard.

- A diminished S_2 occurs with aortic stenosis, a fall in systolic blood pressure (shock), and increased anteroposterior chest diameter.
- Wide splitting of S_2 is associated with delayed emptying of the right ventricle, resulting in delayed pulmonary valve closure (e.g., mitral regurgitation, pulmonary stenosis, and right bundle branch block).
- Fixed splitting occurs when right ventricular output is greater than left ventricular output and pulmonary valve closure is delayed (e.g., with atrial septal defect and right ventricular failure).
- Paradoxical splitting occurs when closure of the aortic valve is delayed (e.g., left bundle branch block).
- Ejection sounds (or clicks) result from the opening of deformed semilunar valves (e.g., aortic and pulmonary stenosis).
- A midsystolic click is heard with mitral valve prolapse (MVP).

Identify extra heart sounds in systole.
Extra heart sounds are not present.



Technique/Normal Findings

Identify the presence of extra heart sounds in diastole. *Extra heart sounds are not present in diastole.*

Identify extra heart sounds in both systole and diastole. *No extra heart sounds should be heard during systole and diastole.*

Murmur Assessment

Identify any murmurs. Note location, timing, presence during systole or diastole, and intensity. Use the following scale to grade murmurs:
 I = Barely heard
 II = Quietly heard
 III = Clearly heard
 IV = Loud
 V = Very loud
 VI = Loudest; may be heard with stethoscope off the chest. A thrill may accompany murmurs of grade IV to grade VI. Note pitch (low, medium, high), and quality (harsh, blowing, or musical). Note pattern/shape, crescendo, decrescendo, and radiation/transmission (to axilla, neck). *No murmurs should be heard.*

Abnormal Findings

- An opening snap results from the opening sound of a stenotic mitral valve.
- A pathologic S₃ (a third heart sound that immediately follows S₂, called a ventricular gallop) results from myocardial failure and ventricular volume overload (e.g., CHF, mitral or tricuspid regurgitation).
- An S₄ (a fourth heart sound that immediately precedes S₁, called an atrial gallop) results from increased resistance to ventricular filling after atrial contraction (e.g., HTN, CAD, aortic stenosis, and cardiomyopathy).
- A combined S₃ and S₄ is called a summation gallop and occurs with severe CHF.
- A pericardial friction rub results from inflammation of the pericardial sac, as with pericarditis.
- Midsystolic murmurs are heard with semilunar valve disease (e.g., aortic and pulmonary stenosis) and with hypertrophic cardiomyopathy.
- Pansystolic (holosystolic) murmurs are heard with AV valve disease (e.g., mitral and tricuspid regurgitation, ventricular septal defect).
- A late systolic murmur is heard with MVP.
- Early diastolic murmurs occur with regurgitant flow across incompetent semilunar valves (e.g., aortic regurgitation).
- Middiastolic and presystolic murmurs, such as with mitral stenosis, occur with turbulent flow across the AV valves.
- Continuous murmurs throughout systole and all or part of diastole occur with patent ductus arteriosus.

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TEST YOURSELF NCLEX-RN® REVIEW

- 1 Which circulatory process supplies the heart with blood?
 1. the systemic circulation
 2. the pulmonary circulation
 3. the coronary circulation
 4. the hepatic circulation
- 2 The amount of blood pumped by the ventricles in 1 minute is known as:
 1. heart rate.
 2. ventricular contraction.
 3. stroke volume.
 4. cardiac output.
- 3 During what part of the cardiac cycle is the myocardium perfused?
 1. prior to atrial filling
 2. prior to ventricular relaxation
 3. during diastole
 4. during pulmonary perfusion
- 4 A client who is hemorrhaging has decreased preload. What physiologic event will follow?
 1. increased afterload
 2. increased ejection fraction
 3. decreased cardiac output
 4. decreased action potential
- 5 What physiologic process is responsible for the electrical impulse that stimulates myocardial contraction?
 1. action potential
 2. cardiac reserve
 3. cardiac potential
 4. ventricular contraction
- 6 The intensity of chest pain may be assessed by asking which question?
 1. "Did the pain move into your left arm?"
 2. "Was your pain relieved by resting or worse when you were busy?"
 3. "On a scale of 0 (no pain) to 10 (worst pain), what number was your pain?"
 4. "Was the pain a pressure, a burning, or a tightness?"
- 7 Which of the following is the most basic exercise stress test?
 1. treadmill test
 2. lipid profile
 3. echocardiogram
 4. cardiac catheterization
- 8 At what anatomic location would you assess the apical impulse?
 1. left midclavicular, fifth intercostal space
 2. left substernal, sixth intercostal space
 3. right midaxillary, second intercostal space
 4. right nipple line, any intercostal space
- 9 Your client's pulse rate is 50. You would document this as:
 1. tachycardia.
 2. bradycardia.
 3. hypertension.
 4. hypotension.
- 10 When auscultating heart sounds, where would S₁ be heard most loudly?
 1. over the clavicles
 2. at the apex of the heart
 3. at the carotid pulse
 4. at the base of the heart

See *Test Yourself answers in Appendix C.*

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