PART IV

Activity and Exercise Patterns

UNIT 9
Responses to Altered Cardiac Function

UNIT 10
Responses to Altered Peripheral Tissue Perfusion

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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 30 Assessing Clients 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

MEDIALINK
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
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 heart is covered by the pericardium, a double layer of fibrosererous 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.

**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.
(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).
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 vena cavae, and is transported to the lungs via the pulmonary artery and its branches (Figure 30–5A). 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
nutrients to the myocardium. The left main coronary artery di-
vides 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
the left lateral wall of the left ventricle. The right coronary artery sup-
plies 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 dur-
ing ventricular relaxation that the coronary arteries fill with oxy-
gen-rich blood. After the blood perfuses the heart muscle, the
cardiac veins drain the blood into the coronary sinus, which em-
pies 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 fac-
tors 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). Ven-
tricular filling is followed by ventricular systole, a phase dur-
ing which the ventricles contract and eject blood into the
pulmonary and systemic circuits. Systole is followed by a re-
 laxation phase known as diastole, during which the ventricles
refill, the atria contract, and the myocardium is perfused. Nor-
ma

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 ventricular 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. Vasodilation 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 (m²), 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/m². 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 m². This client’s cardiac index is 4 ÷ 1.54, or 2.6 L/min/m². The second client is 6 feet, 2 inches (188 cm) tall and weighs 280 lb (81.7 kg), with a BSA of 2.52 m². This client’s cardiac index is 4 ÷ 2.52, or 1.6 L/min/m². 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
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 fluids 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.

**Repolariization**

Repolariization 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-
Noninvasive tests of cardiac structure and function include diagnostic tests to assess the structures and functions of the heart. These tests can be performed during 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 943–946 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 radionuclide tracers.
- Additional tests include electrocardiography (ECG), thallium myocardial perfusion imaging, and exercise thallium or technetium tests.
- Other tests include echocardiography, cardiac catheterization, pericardiocentesis, and stress tests.
- Stress tests are performed to evaluate the client’s response to exercise or pharmacological agents.

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, ask 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...
### Stress/exercise tests

**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 may be fatigued, develop symptoms, or reach their maximum predicted heart rate.

**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).
- 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.

### Thallium/technetium stress test (myocardial imaging perfusion test, cardiac blood pool imaging)

**PURPOSE AND DESCRIPTION** 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).
- 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.

### 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. 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.

### 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.

### Magnetic resonance imaging (MRI)

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

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

(Nota: 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.
### Diagnostic Tests of Cardiac Disorders (continued)

<table>
<thead>
<tr>
<th>NAME OF TEST</th>
<th>PURPOSE AND DESCRIPTION</th>
<th>RELATED NURSING CARE</th>
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<td><strong>RELATED NURSING CARE</strong></td>
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<tr>
<td><strong>Computed tomography (CT) scan</strong></td>
<td>A CT scan may be conducted to quantify calcium deposits in coronary arteries.</td>
<td><strong>Assess for allergy to iodine or seafood if contrast medium is to be administered.</strong></td>
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<td><strong>Cardioiite scan</strong></td>
<td>Used to evaluate blood flow in different parts of the heart. Cardioliite (technetium 99m sestamibi) is injected IV. In a dipyridamole cardioliite scan, dipyridamole (Persantine) is injected to increase blood flow to coronary arteries. These scans may be done in conjunction with a treadmill test.</td>
<td><strong>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.</strong></td>
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<td><strong>Positron emission tomography (PET)</strong></td>
<td>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.</td>
<td><strong>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.</strong></td>
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<td><strong>Blood pool imaging</strong></td>
<td>Following intravenous injection of technetium 99m pertechnetate, sequential evaluation of the heart can be performed for several hours.</td>
<td><strong>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.</strong></td>
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<td><strong>Echocardiogram</strong></td>
<td><strong>PURPOSE AND DESCRIPTION</strong> 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.</td>
<td><strong>No special preparation is needed.</strong></td>
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<tr>
<td><strong>Transesophageal echocardiography (TEE)</strong></td>
<td>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.</td>
<td><strong>No special preparation is needed; see related nursing care for the client having a treadmill test for a stress echocardiogram.</strong></td>
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<td><strong>Cardiac catheterization (coronary angiography, coronary arteriography)</strong></td>
<td>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.</td>
<td><strong>Explain the procedure to the client.</strong></td>
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**Right cardiac catheterization:** The catheter is inserted into the femoral vein or antecubital vein and then threaded through the 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.
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.

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 alternations. 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).

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.

- 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 the 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.

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.

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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.

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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.
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 (aVR, aVL, and aVF). 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. 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.
Have you noticed any changes in your energy level?

Describe the type of activity that brings on your chest pain.

What is the location of the chest pain you experienced? Did it move up to your jaw or into your left arm?

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...
Table 30-1 Assessing Chest Pain

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

...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 murmurs, 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 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

<table>
<thead>
<tr>
<th>Functional Health Pattern</th>
<th>Interview Questions and Leading Statements</th>
</tr>
</thead>
</table>
| **Health Perception-Health Management** | ■ 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** | ■ 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** | ■ Has a heart problem interfered with your usual bowel and bladder elimination? Explain. |
| **Activity-Exercise** | ■ 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** | ■ 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** | ■ 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** | ■ How does having this condition make you feel about yourself? |
| **Role-Relationships** | ■ How does this condition affect your relationships with others?  
■ Has having this condition interfered with your ability to work? Explain. |
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

<table>
<thead>
<tr>
<th>AGE-RELATED CHANGE</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardium: ↓ efficiency and contractibility.</td>
<td>Decreased cardiac output when under physiologic stress with resulting tachycardia that lasts longer. The person may require rest time between physical activities.</td>
</tr>
<tr>
<td>Sinus node: † in thickness of shell surrounding the node, and a ↓ in the number of pacemaker cells</td>
<td>Stroke volume may increase to compensate for tachycardia, leading to increased blood pressure.</td>
</tr>
<tr>
<td>Left ventricle: Slight hypertrophy, prolonged isometric contraction phase and relaxation time; † time for diastolic filling and systolic emptying cycle.</td>
<td>Blood pressure increases to compensate for increased peripheral resistance and decreased cardiac output.</td>
</tr>
<tr>
<td>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.</td>
<td></td>
</tr>
</tbody>
</table>

### CARDIAC ASSESSMENTS

#### Apical Impulse Assessment

<table>
<thead>
<tr>
<th>Technique/Normal Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>First using the palmar surface and then repeating with finger pads, palpate the precordium for symmetry of movement</td>
<td>- 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).</td>
</tr>
<tr>
<td></td>
<td>- 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</td>
</tr>
</tbody>
</table>
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.

Increasing 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 pulmonic stenosis, aortic stenosis, pulmonic HTN, or atrial septal defect.

Increased pulsation at the aortic area may suggest aortic aneurysm. A palpable second heart sound (S2) may be noted with systemic HTN.

### 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.

- 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.
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: S1, S2, murmurs, pericardial friction rubs.
   b. Next, auscultate each area with the bell of the stethoscope to listen for lower pitched sounds: S3, S4, 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 S1 (first heart sound) and note its intensity. At each auscultatory area, listen for several cardiac cycles. See Figure 30–11 for auscultation areas. S1 is loudest at the apex of the heart.

- An accentuated S1 occurs with tachycardia, states in which cardiac output is high (fever, anxiety, exercise, anemia, hyperthyroidism), complete heart block, and mitral stenosis.
- A diminished S1 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 S1 occurs with complete heart block and grossly irregular rhythms.

Listen for splitting of S1.
**S1 splitting may occur during inspiration.**

Identify S2 (second heart sound) and note its intensity. S2 immediately follows S1 and is loudest at the base of the heart.

- An accentuated S2 may be heard with HTN, exercise, excitement, and conditions of pulmonary HTN such as CHF and cor pulmonale.
- A diminished S2 occurs with aortic stenosis, a fall in systolic blood pressure (shock), and increased anteroposterior chest diameter.
- Wide splitting of S2 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).

Listen for splitting of S2. No splitting of S2 should be heard.

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

- An opening snap results from the opening sound of a stenotic mitral valve.
- A pathologic \( S_3 \) (a third heart sound that immediately follows \( S_2 \), called a ventricular gallop) results from myocardial failure and ventricular volume overload (e.g., CHF, mitral or tricuspid regurgitation).
- An \( S_4 \) (a fourth heart sound that immediately precedes \( S_1 \), called an atrial gallop) results from increased resistance to ventricular filling after atrial contraction (e.g., HTN, CAD, aortic stenosis, and cardiomyopathy).
- A combined \( S_3 \) and \( S_4 \) is called a summation gallop and occurs with severe CHF.

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

An opening snap results from the opening sound of a stenotic mitral valve.

- A pathologic \( S_3 \) (a third heart sound that immediately follows \( S_2 \), called a ventricular gallop) results from myocardial failure and ventricular volume overload (e.g., CHF, mitral or tricuspid regurgitation).
- An \( S_4 \) (a fourth heart sound that immediately precedes \( S_1 \), called an atrial gallop) results from increased resistance to ventricular filling after atrial contraction (e.g., HTN, CAD, aortic stenosis, and cardiomyopathy).
- A combined \( S_3 \) and \( S_4 \) is called a summation gallop and occurs with severe CHF.

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.

- 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.

EXPLORE Medialink

Prentice Hall Nursing MediaLink DVD-ROM
Audio Glossary
NCLEX-RN® Review

Animation/Videos
Cardiac A&P
Dysrhythmias
Heart Sounds
Hemodynamics
Oxygen Transport

COMPANION WEBSITE www.prenhall.com/lemone
Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Cardiac Catheterization
Case Study: Chest Pain
MediaLink Application: Heart Sounds
Links to Resources
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 catherization

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.

BIBLIOGRAPHY


