

# Patient Assessment: Cardiovascular System

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## Cardiac History and Physical Examination

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## objectives

*Based on the content in this chapter, the reader should be able to:*

- Explain the components of the cardiovascular history.
- Describe the steps of the cardiovascular physical examination.
- Discuss the mechanisms responsible for the production of the first, second, third, and fourth heart sounds and their timing in the cardiac cycle.
- Explain each type of murmur, its timing in the cardiac cycle, and the area on the chest wall where it is most easily auscultated.
- Compare and contrast the usefulness of serum enzymes and myocardial proteins in diagnosing an acute myocardial infarction.
- Explain possible causes of serum creatine kinase elevations other than acute myocardial infarction and ischemia.
- Describe current techniques used for diagnostic purposes in cardiology.
- Discuss the nursing care before and after cardiac diagnostic studies.
- Outline the patient and family teaching appropriate to prepare the patient for cardiac diagnostic studies.
- Describe potential complications of cardiac diagnostic procedures.
- Explain the major features of an electrocardiogram (ECG) monitoring system.
- Compare and contrast a hard-wire monitoring system with a telemetry monitoring system.

- Explain correct electrode placement when monitoring the standard leads or the chest leads with a three-electrode and a five-electrode system.
- Discuss steps for troubleshooting ECG monitor problems.
- Describe the components of the ECG tracing and their meaning.
- Explain the steps used to interpret a rhythm strip.
- Describe the causes, clinical significance, and management for each of the arrhythmias discussed.
- Describe the parameters of a normal 12-lead ECG.
- Define electrical axis, and determine the direction of the axis for a 12-lead ECG.
- Explain the causes, clinical significance, and treatment for bundle branch blocks, atrial enlargement, and ventricular enlargement.
- Describe the ECG changes associated with serum potassium and calcium abnormalities.
- Analyze the characteristics of normal systemic arterial, right atrial, right ventricular, pulmonary artery, and pulmonary artery wedge pressure waveforms.
- Describe the system components required to monitor hemodynamic pressures.
- State nursing interventions that ensure accuracy of pressure readings.
- Discuss the major complications that can occur with an indwelling arterial line and pulmonary artery catheter.
- Describe the thermodilution method of measuring cardiac output.
- Identify the determinants of cardiac output.
- Evaluate the factors influencing oxygen delivery and consumption.
- Use  $\text{S}\bar{\text{v}}\text{O}_2$  monitoring to assess oxygen delivery and consumption.

The application of complex technology to the assessment and management of cardiovascular and cardiopulmonary conditions has increased greatly in the past several decades. Use of advanced and complex technologies is an integral part of the care of critically ill patients. Nevertheless, the value of a comprehensive cardiovascular history and physical examination should never be underestimated. The chapter begins with a discussion of the cardiac history and physical examination and then discusses technological assessment techniques.

## CARDIAC HISTORY AND PHYSICAL EXAMINATION

The cardiovascular history provides physiological and psychosocial information that guides the physical assessment, the selection of diagnostic tests, and the choice of treatment options. During the history, the nurse asks about the presenting symptoms, past health history, current health status, risk factors, family history, and social and personal history. The nurse also inquires about behaviors that promote or jeopardize cardiovascular health and uses this information in guiding health teaching. During the process of taking a thorough history and performing a physical examination, the nurse has an opportunity to establish rapport with the patient and to evaluate the patient's general emotional status.

### History

#### CHIEF COMPLAINT AND HISTORY OF PRESENT ILLNESS

The nurse begins the history by investigating the patient's chief complaint. The patient is asked to describe in his or her own words the problem or reason for seeking care.

The nurse then asks for more information about the present illness, using the questions in Box 17-1. Answers to these questions are essential to understanding the patient's perception of the problem. The nurse also asks the patient about any associated symptoms, including chest pain, dyspnea, edema of feet/ankles, palpitations and syncope, cough and hemoptysis, nocturia, cyanosis, and intermittent claudication.

#### Chest Pain

Chest pain is one of the most common symptoms of patients with cardiovascular disease. Therefore, it is an essential component of the assessment interview. Chest pain is often a disturbing or even frightening experience for a patient, so the patient may be hesitant to initiate a discussion of chest pain. The questions listed in Box 17-1 are particularly useful when assessing chest pain.

Because cardiac pain (angina pectoris) is the result of an imbalance between oxygen supply and oxygen demand, it usually develops over time. Typically, anginal pain does not start at maximal intensity. Not all chest pain is cardiac in origin, and careful reporting of the characteristics of the pain and the behaviors (or lack thereof) that precede the onset of pain is required. The nurse asks the patient about his or her *normal* baseline status before the symptoms developed. It is also important to ask about the *onset* of the symptoms to determine the date and time that the symptoms started and whether the onset was sudden or gradual.

Chest pain caused by coronary artery disease is often *precipitated* by physical or emotional exertion, a meal, or being out in the cold. *Palliative* measures to relieve anginal pain may include rest or sublingual nitrates; these measures usually do not relieve the pain of a myocardial infarction (MI). The *quality* of cardiac chest pain is often described as a heaviness, tightness, squeezing, or choking sensation. If

**box 17-1****Assessment Parameters: Questions to Ask in a Symptom Assessment**

- N Normal:** Describe your normal baseline. What was it like before this symptom developed?
- O Onset:** When did the symptom start? What day? What time? Did it start suddenly or gradually?
- P Precipitating and palliative factors:** What brought on the symptom? What seems to trigger it—factors such as stress, position change, or exertion? What were you doing when you first noticed the symptom? What makes the symptom worse? What measures have helped relieve the symptom? What have you tried so far? What measures did not relieve the symptom?
- Q Quality and quantity:** How does it feel? How would you describe it? How much are you experiencing now? Is it more or less than you experienced at any other time?
- R Region and radiation:** Where does the symptom occur? Can you show me? In the case of pain, does it travel anywhere such as down your arm or in your back?
- S Severity:** On a scale of 1 to 10, with 10 being the worst ever experienced, rate your symptom. How bad is the symptom at its worst? Does it force you to stop your activity and sit down, lie down, or slow down? Is the symptom getting better or worse, or staying about the same?
- T Time:** How long does the symptom last? How often do you get the symptom? Does it occur in association with anything, such as before, during, or after meals?

the pain is reported as superficial, knifelike, or throbbing, it is not likely to be anginal. Cardiac chest pain is usually located in the substernal *region* and often *radiates* to the neck, left arm, the back, or jaw. Although the pain is often referred to other areas, anginal pain is visceral in origin, and most complaints include a reference to a “deep, inside” pain. When the patient is asked to point to the painful area, the painful area is about the size of a hand or clenched fist. It is unusual for true anginal pain to be localized to an area smaller than a fingertip. Using a scale of 1 to 10, with 10 being the worst pain the patient has ever experienced, the patient is asked to rate the *severity* of the pain. When asked about *time*, the patient with cardiac chest pain reports the pain lasting anywhere from 30 seconds to hours.

Pain may be secondary to cardiovascular problems that are unrelated to a primary coronary insufficiency. Therefore, when obtaining the patient’s history, the nurse must consider other causes. For example, if the patient reports the pain is made worse by lying down, moving, or deep breathing, it may be caused by pericarditis. If the pain is retrosternal and accompanied by sudden shortness of breath and peripheral cyanosis, it may be caused by a pulmonary embolism.

**Dyspnea**

Dyspnea occurs in patients with both pulmonary and cardiac abnormalities. In patients with cardiac disease, it is

the result of inefficient pumping of the left ventricle, which causes a congestion of blood flow in the lungs. During history taking, dyspnea is differentiated from the usual breathlessness that follows a sudden burst of physical activity (e.g., running up four flights of stairs, sprinting across a parking lot). Dyspnea is a subjective complaint of true *difficulty* in breathing, not just shortness of breath. The nurse determines whether the breathing difficulty occurs only with exertion or also at rest. If dyspnea is present when the patient lies flat but is relieved by sitting or standing, it is *orthopnea*. If it is characterized by breathing difficulties starting after approximately 1 to 2 hours of sleep and relieved by sitting upright or getting out of bed, it is *paroxysmal nocturnal dyspnea*.

**Edema of the Feet and Ankles**

Although many other problems can leave a patient with swollen feet or ankles, heart failure may also be responsible because the heart is unable to mobilize fluid appropriately. Because gravity promotes the movement of fluids from intravascular to extravascular spaces, the edema becomes worse as the day progresses and usually improves at night after lying down to sleep. Patients or families may report that shoes do not fit anymore, socks that used to be loose are now too tight, and the indentations from sock bands take more time than usual to disappear. The nurse should inquire about the timing of edema development (e.g., immediately after lowering the extremities, only at the end of the day, only after a significant salt intake) and duration (e.g., relieved with temporary elevation of the legs or with constant elevation).

**Palpitations and Syncope**

Palpitations refer to the awareness of irregular or rapid heart beats. Patients may report the “skipping” of beats, a rushing of the heart, or a loud “thudding.” The nurse asks about onset and duration of the palpitations, associated symptoms, and any precipitating events that the patient or family can remember. Because a cardiac arrhythmia may compromise blood flow to the brain, the nurse asks about symptoms of dizziness, fainting, or syncope that accompany the palpitations.

**Cough and Hemoptysis**

Abnormalities such as heart failure, pulmonary embolus, or mitral stenosis may cause a cough or hemoptysis. The nurse asks the patient about the presence of a cough and inquires about the quality (wet or dry) and frequency of the cough (chronic or occasional, only when lying down or after exercise). If the cough produces expectorant, the nurse records its color, consistency, and amount perceived by the patient. If the patient reports spitting up blood (hemoptysis), the nurse asks if the substance spit up was streaked with blood, frothy bloody sputum, or frank blood (bright or dark).

**Nocturia**

Kidneys that are inadequately perfused by an unhealthy heart during the day may finally receive sufficient flow during rest at night to increase their output. The nurse asks about the number of times the patient urinates during the night. If the patient takes a diuretic, the nurse also

evaluates frequency of urination in relation to the time of day the diuretic is taken.

### Cyanosis

Cyanosis reflects the oxygenation and circulatory status of the patient. *Central* cyanosis is generally distributed and best found by examining the mucous membranes for discoloration and duskiess, and reflects reduced oxygen concentration. *Peripheral* cyanosis is localized in the extremities and protrusions (hands, feet, nose, ears, lips) and reflects impaired circulation.

### Intermittent Claudication

Claudication results when the blood supply to exercising muscles is inadequate. Usually the cause of claudication is significant atherosclerotic obstruction to the lower extremities. The limb is asymptomatic at rest unless the obstruction is severe. Blood supply to the legs is inadequate to meet metabolic demands during exercise, and ischemic pain results. The patient describes a cramping, “charley horse,” ache, or weakness in the foot, calf, thigh, or buttocks that improves with rest. The patient should be asked to describe the severity of the pain and how much exertion is required to produce the pain.

### PAST HEALTH HISTORY

When assessing the patient’s past health history, the nurse inquires about childhood illnesses such as rheumatic fever as well as previous illnesses such as pneumonia, tuberculosis, thrombophlebitis, pulmonary embolism, MI, diabetes

mellitus, thyroid disease, or chest injury. The nurse also asks about occupational exposures to cardiotoxic materials. Finally, the nurse seeks information about previous cardiac or vascular surgeries and any previous cardiac studies or interventions (Box 17-2).

### CURRENT HEALTH STATUS AND RISK FACTORS

As part of the health history, the nurse queries the patient about use of prescription and over-the-counter medications, vitamins, and herbs. It is essential to ask the patient about drug allergies, food allergies, or any previous allergic reactions to contrast agents. The nurse inquires about use of tobacco, drugs, and alcohol. The nurse also asks about dietary habits, including usual daily food intake, dietary restrictions or supplements, and intake of caffeine-containing foods or beverages. The patient’s sleep pattern and exercise and leisure activities also are noted (see Box 17-2).

Assessment of risk factors for cardiovascular disease is an important component of the history. Risk factors are categorized as major uncontrollable risk factors; major risk factors that can be modified, treated, or controlled; and contributing risk factors. Box 17-3 summarizes these risk factors.<sup>1,2</sup>

### FAMILY HISTORY

The nurse asks about the age and health, or age and cause of death, of immediate family members, including parents, grandparents, siblings, children, and grandchildren.

#### box 17-2

#### Cardiovascular Health History

##### Chief Complaint

- Patient’s description of the problem

##### History of the Present Illness

- Complete analysis of the symptoms (using the NOPQRST format; see Box 17-1)

##### Past Health History

- Childhood illnesses: rheumatic fever, murmurs, congenital anomalies
- Past medical problems: heart failure, hypertension, coronary artery disease, myocardial infarction, hyperlipidemia, valve disease, cardiac arrhythmias, peripheral vascular disease, diabetes
- Past surgeries: cardiovascular surgeries such as coronary artery bypass grafting, valve replacement, peripheral vascular procedures; surgeries for other health problems
- Past diagnostic tests and interventions: electrocardiogram, echocardiogram, cardiac catheterization, stress test, electrophysiological studies, percutaneous transluminal coronary angioplasty, stent placement, atherectomy, pacemaker implantation, implantable cardioverter-defibrillator placement, valvuloplasty

##### Current Health Status and Risk Factors

- Medications: prescription drugs, over-the-counter drugs, vitamins, herbs and supplements

- Allergies and reactions: medications, food, contrast agents
- Tobacco, alcohol, and substance use
- Diet
- Sleep patterns
- Exercise
- Leisure activities
- Risk factors: major risk factors that cannot be altered, major risk factors that can be altered, contributing risk factors (see Box 17-3)

##### Family History

- Hypertension, elevated cholesterol, coronary artery disease, myocardial infarction, stroke, peripheral vascular disease, cardiac arrhythmias

##### Social and Personal History

- Family composition
- Living environment
- Daily routine
- Sexual activity
- Occupation
- Coping patterns
- Cultural beliefs
- Spiritual/religious beliefs



**box 17-3****Risk Factors for Cardiovascular Disease****Major Uncontrollable Risk Factors**

- **Age:** There is an increased incidence of all types of atherosclerotic disease with aging. About 85% of people who die from coronary artery disease are age 65 years or older. Women at older ages who have a myocardial infarction are twice as likely as men to die from it within a few weeks.
- **Heredity:** The tendency for development of atherosclerosis seems to run in families. The risk is thought to be a combination of environmental and genetic influences. Even when other risk factors are controlled, the chance for development of coronary artery disease increases when there is a familial tendency.
- **Gender:** Men have a greater risk for development of coronary artery disease than women at earlier ages. After menopause, women's death rate from myocardial infarction increases but is not as great as men's.
- **Race:** Rates of cardiovascular disease are higher for African Americans, Mexican Americans, Native Americans, Native Hawaiians, and some Asian Americans.

**Major Risk Factors That Can Be Modified, Treated, or Controlled**

- **Tobacco smoking:** A smoker's risk of a myocardial infarction is more than twice that of nonsmokers. Smokers who have a myocardial infarction are more likely to die and die within an hour than are nonsmokers. Cigarette smoking is the greatest risk factor for sudden death. Smokers have two to four times the risk of sudden death compared with nonsmokers. Chronic exposure to environmental tobacco smoke may increase the risk of heart disease.
- **High blood cholesterol:** The risk of coronary heart disease increases as the blood cholesterol level rises. When other risk factors are present, this risk increases even more.
- **Hypertension:** Known as the "silent killer," hypertension is a risk factor with no specific symptoms and no early warning signs. Men have a greater risk for hypertension than women until the age of 55 years. The risk for development of hypertension is about the same for men and women between the ages of 55 and 75 years. After the age of 75 years, hypertension is more likely to develop in women than in men. African Americans are more likely to have hypertension than whites. Hypertension increases the risk of stroke, myocardial infarction, kidney failure, and heart failure.
- **Physical inactivity:** A lack of physical exercise is a risk factor for coronary artery disease. Moderate to vigorous

regular exercise plays a significant role in preventing heart disease and blood vessel disease. Even moderate-intensity exercise is beneficial if performed regularly and long-term. Physical activity also plays a role in controlling cholesterol, diabetes, obesity, and hypertension.

- **Obesity:** There is an association between obesity and an increased mortality rate from coronary artery disease and stroke. Excess weight is also linked with an increased incidence of hypertension, insulin resistance, diabetes, and dyslipidemia.
- **Diabetes mellitus:** Diabetes significantly increases the risk for development of cardiovascular disease. The associated risk is greater for women than for men. Three fourths of individuals with diabetes die of some form of heart or blood vessel disease.

**Other Contributing Factors**

- **Stress:** A person's response to stress may be a contributing factor to heart disease. Stress in a person's life, his or her health behaviors, and socioeconomic status may all contribute to established risk factors. For example, individuals under stress may overeat, smoke, and not exercise.
- **Sex hormones:** Men are at higher risk of myocardial infarction than premenopausal women. After menopause, the risk for women increases because it is believed that the loss of natural estrogen as the woman ages may be a contributing factor.
- **Birth control pills:** Newer low-dose oral contraceptives carry a much lower risk of cardiovascular disease than the early forms of birth control pills. If a woman using oral contraceptives has other risk factors such as smoking or hypertension, her risk for development of blood clots and having a myocardial infarction increases.
- **Excessive alcohol intake:** Drinking too much alcohol can raise blood pressure, cause heart failure, lead to stroke, contribute to high triglycerides and obesity, and produce arrhythmias. The risk of heart disease in individuals who drink moderate amounts of alcohol (an average of one drink for women and two drinks for men per day) is lower than in those who do not drink alcohol.
- **Homocysteine levels:** An association has been shown between elevated levels of homocysteine and cardiovascular disease. Homocysteine causes increased platelet adhesiveness, enhances low-density lipoprotein deposition in the arterial wall, and activates the coagulation cascade.

The nurse inquires about cardiovascular problems such as hypertension, elevated cholesterol, coronary artery disease, MI, stroke, and peripheral vascular disease (see Box 17-2).

**SOCIAL AND PERSONAL HISTORY**

Although the physical symptoms provide many clues regarding the origin and extent of cardiac disease, social and personal history also contribute to the patient's health status. The nurse inquires about the patient's family, spouse or significant other, and children. Information about the

patient's living environment, daily routine, sexual activity, occupation, coping patterns, and cultural and spiritual beliefs contributes to the nurse's understanding of the patient as a person and guides interaction with the patient and family (see Box 17-2).

**Physical Examination**

Cardiac assessment requires examination of all aspects of the individual, using the standard steps of inspection, palpation, percussion, and auscultation. A thorough and careful

examination helps the nurse detect subtle abnormalities as well as obvious ones.

## INSPECTION

### General Appearance

Inspection begins as soon as the patient and nurse interact. General appearance and presentation of the patient are key elements of the initial inspection. Critical examination reveals a first impression of age, nutritional status, self-care ability, alertness, and overall physical health.

It is necessary to note the ability of the patient to move and speak with or without distress. Consider the patient's posture, gait, and musculoskeletal coordination.

### Jugular Venous Distension

Pressure in the jugular veins reflects right atrial pressure and provides the nurse with an indication of heart hemodynamics and cardiac function. The height of the level of blood in the right internal jugular vein is an indication of right atrial pressure because there are no valves or obstructions between the vein and the right atrium.

The internal jugular veins are not directly visible, because they lie deep to the sternomastoid muscles in the neck (Fig. 17-1). The goals of the examination are to determine the highest point of visible pulsation in the internal jugular veins, to note the level of head elevation, and to measure this point of visible pulsation as the vertical distance above the sternal angle. The patient is placed in the bed supine with the head of the bed elevated 30, 45, 60, and 90 degrees. The patient is examined at each elevation with the head slightly turned away from the examiner. The nurse uses tangential light to observe for the highest point of visible pulsation.<sup>3,4</sup>

Next, the angle of Louis is located by palpating where the clavicle joins the sternum (suprasternal notch). The examining finger is slid down the sternum until a bony prominence is felt. This prominence is known as the *angle of Louis*. A vertical ruler is placed on the angle of Louis. Another ruler is placed horizontally at the level of the pul-

sation. The intersection of the horizontal ruler with the vertical ruler is noted, and the intersection point on the vertical ruler is read.

Normal jugular venous pulsation should not exceed 3 cm above the angle of Louis. See Figure 17-2 for an illustration of the procedure for assessment of jugular venous pressure. A level more than 3 cm above the angle of Louis indicates an abnormally high volume in the venous system. Possible causes include right-sided heart failure, obstruction of the superior vena cava, pericardial effusion, and other cardiac or thoracic diseases. An increase in the jugular venous pressure of more than 1 cm while pressure is applied to the abdomen for 60 seconds (hepatojugular or abdominjugular test) indicates the inability of the heart to accommodate the increased venous return.

### Chest

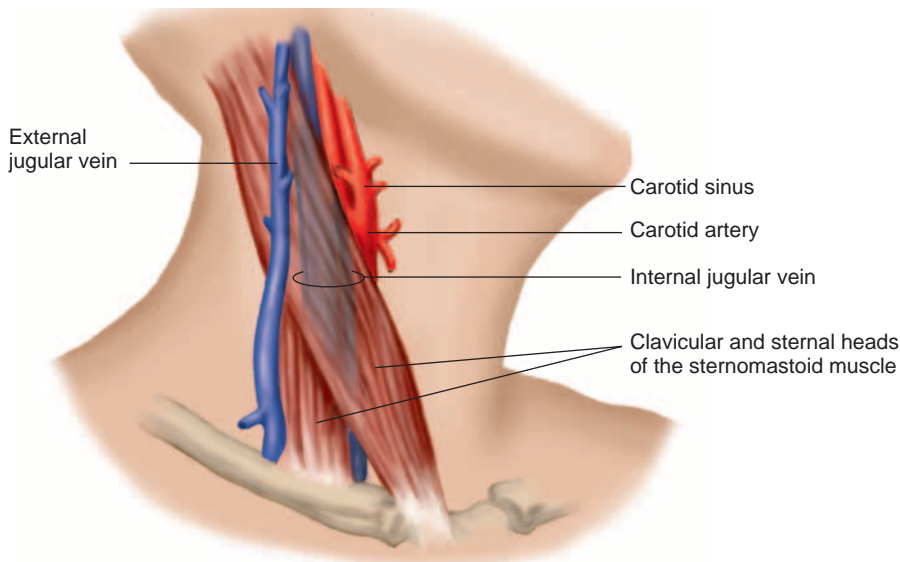
The chest is inspected for signs of trauma or injury, symmetry, chest contour, and any visible pulsations. Thrusts (abnormally strong precordial pulsations) are noted. Any depression (sternum excavatum) or bulging of the precordium is recorded.

### Extremities

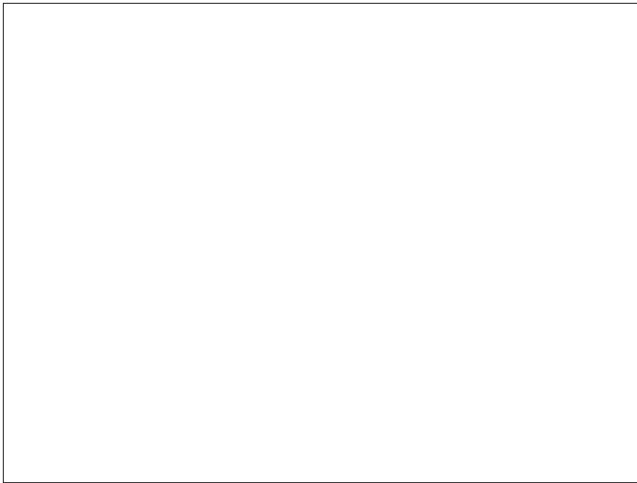
A close inspection of the patient's extremities can also provide clues about cardiovascular health. The extremities are examined for lesions, ulcerations, unhealed sores, and varicose veins. Distribution of hair on the extremities also is noted. A lack of normal hair distribution on the extremities may indicate diminished arterial blood flow to the area.

### Skin

Skin is evaluated for moistness or dryness, color, elasticity, edema, thickness, lesions, ulcerations, and vascular changes. Nail beds are examined for cyanosis and clubbing, which may indicate chronic cardiac abnormalities. General differences in color and temperature between body parts may provide perfusion clues.



**figure 17-1** Internal jugular veins. (From Bickley L: Bates' Guide to Physical Examination and Health History [8th Ed], p 32. Philadelphia, Lippincott Williams & Wilkins, 2003.)



**figure 17-2** Assessment of jugular venous pressure. Place the patient supine in bed and gradually raise the head of the bed to 30, 45, 60, and 90 degrees. Using tangential lighting, note the highest level of venous pulsation. Measure the vertical distance between this point and the sternal angle. Record this distance in centimeters and the angle of the head of the bed.

## PALPATION

### Pulses

Cardiovascular assessment continues with palpation and involves the use of the pads of the finger and balls of the hand. Using the pads of the fingers, the carotid, brachial, radial, femoral, popliteal, posterior tibial, and dorsalis pedis pulses are palpated. The peripheral pulses are compared bilaterally to determine rate, rhythm, strength, and symmetry. The 0-to-3 scale described in Box 17-4 is used to rate the strength of the pulse. The carotid pulses should never be assessed simultaneously because this can obstruct flow to the brain.

Pulses can also be described according to their characteristics. For example, *pulsus alternans* is a pulse that alternates in strength with every other beat; it is often found in patients with left ventricular failure. *Pulsus paradoxus* is a pulse that disappears during inspiration but returns during expiration. To determine if the condition is pathological, the sphygmomanometer is deflated until the pulse is heard only during expiration and the corresponding pressure noted. As the cuff continues to deflate, the point at which the pressure is heard *throughout* the inspiratory and expiratory cycle is noted. The second systolic pressure

reading is subtracted from the first; if the difference is greater than 10 mm Hg during normal respirations, it is considered pathological. During the assessment of pulses, the nurse compares the warmth and size of the palpated areas to monitor perfusion.

### Precordium

The chest wall is palpated to assess for the point of maximal impulse (PMI), thrills, and abnormal pulsations. The nurse first uses the pads of the fingers and then places a hand flat against the patient's chest, using light pressure.

A systematic palpation sequence is used with the patient in a supine position and includes the precordial areas shown in Figure 17-3. Palpation starts with locating the PMI. In most patients, the PMI represents the point where the apical pulse is most readily felt. The PMI is palpated, noting its location, diameter, amplitude, and duration. Usually, the PMI is located in the midclavicular line at about the fourth or fifth intercostal space. If the pulse is difficult to palpate, it may be necessary to ask the patient to turn on the left side (left lateral decubitus position).

Next, the nurse palpates the lower left sternal border area, the upper left sternal border area, the sternoclavicular area, the right upper sternal border area, the lower right sternal border area, and finally the epigastric area. During palpation of these areas, the nurse feels for a thrill, which is a palpable vibration. A thrill usually represents a disruption in blood flow related to a defect in one of the semilunar valves.

## PERCUSSION

With the advent of radiological means of evaluating cardiac size, percussion is not a significant contribution to cardiac assessment. However, a gross determination of heart size can be made by percussing for the dullness that reflects the cardiac borders.

## AUSCULTATION

Data obtained by careful and thorough auscultation of the heart are essential in planning and evaluating care of the critically ill patient. In this section, the basic principles underlying cardiac auscultation; the factors responsible for the production of normal heart sounds; and the pathological conditions responsible for the production of extra sounds, murmurs, and friction rubs are discussed.

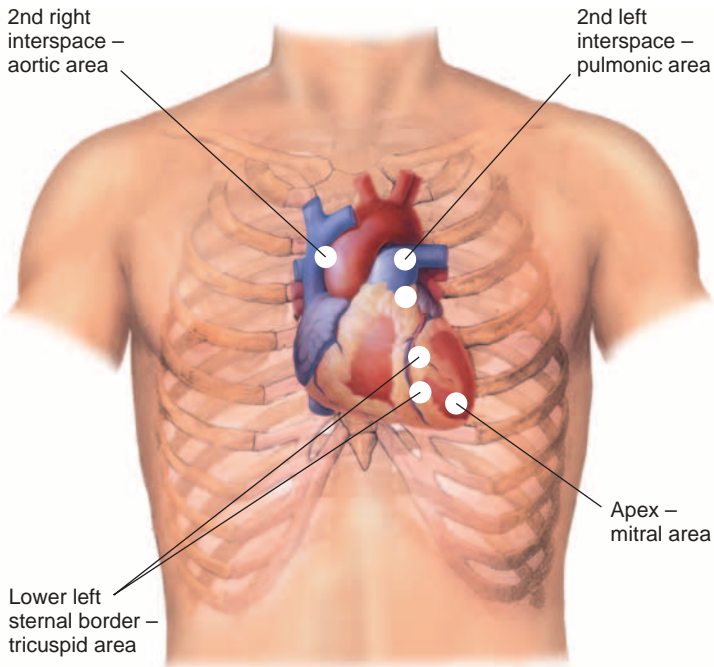
To facilitate accurate auscultation, the patient should be relaxed and comfortable in a quiet, warm environment with adequate lighting. The patient should be in a recumbent position with the trunk elevated 30 to 45 degrees. To help hear abnormal sounds, the patient may be asked to roll partly onto the left side (left lateral decubitus position). This position helps bring the left ventricle closer to the chest wall.

A good-quality stethoscope is essential. The earpieces should fit the ears snugly and comfortably and follow the natural angle of the ear canals. Sound waves that travel a shorter distance are more intense and less distorted; therefore, the tubing of the stethoscope should be approximately 12 inches in length and somewhat rigid. It is best to have two tubes leading from the head of the stethoscope, one to each ear. The head of the stethoscope should be equipped

### box 17-4

#### Rating Scale Used for Assessing Strength of Pulses

0	Absent
1	Palpable but thready, weak, easily obliterated
2	Normal, not easily obliterated
3	Full, bounding, easily palpable, cannot obliterate



**figure 17-3** Areas of auscultation. I. Aortic area (second intercostal space to the right of the sternum). II. Pulmonic area (second intercostal space to the left of the sternum). III. Tricuspid area (fifth intercostal space to the left of the sternum). IV. Mitral or apical area (fifth intercostal space midclavicular line). (From Bickley L: *Bates' Guide to Physical Examination and Health History* [8th Ed], p 278. Philadelphia, Lippincott Williams & Wilkins, 2003.)

with both a diaphragm and a bell on a valve system that allows the clinician to switch easily between the two components. The diaphragm is used to hear high-frequency sounds, such as the first and second heart sounds ( $S_1$ ,  $S_2$ ), friction rubs, systolic murmurs, and diastolic insufficiency murmurs. The diaphragm should be placed firmly on the chest wall to create a tight seal. Low-frequency sounds, such as the third and fourth heart sounds ( $S_3$ ,  $S_4$ ) and the diastolic murmurs of mitral and tricuspid stenosis, are best heard with the stethoscope bell, which should be placed lightly on the chest wall only to seal the edges.

The precordium should be auscultated systematically (see Fig. 17-3). Some authorities suggest the use of anatomical names for the auscultation areas (e.g., aortic and pulmonic), whereas others discourage the use of such labels because murmurs of more than one origin can be heard in a given area.<sup>3</sup> The nurse begins the examination by listening with the stethoscope diaphragm in the right second intercostal space along the sternum. This area is sometimes called the aortic area and is the place where  $S_2$  is loudest. Next, the nurse places the stethoscope in the left second intercostal space along the sternum, which is known as the pulmonic listening area, and from there moves the stethoscope down the left sternal border between the second and fifth spaces, one intercostal space at a time. The lower left sternal border area is sometimes referred to as the tricuspid area. Finally, the nurse moves the stethoscope to the mitral area or apex of the heart, where  $S_1$  is the loudest. This pattern is then repeated with the stethoscope bell.

In each area auscultated, the nurse should identify  $S_1$ , noting the intensity of the sound, respiratory variation, and splitting.  $S_2$  should then be identified and the same characteristics assessed. After  $S_1$  and  $S_2$  are identified, the presence of extra sounds is noted—first in systole, then in diastole. Finally, each area is auscultated for the presence of murmurs and friction rubs.

### First Heart Sound

$S_1$  is timed with the closure of the mitral and tricuspid valves at the beginning of ventricular systole (Fig. 17-4). Because mitral valve closure is responsible for most of the sound produced,  $S_1$  is heard best in the mitral or apical area. The upstroke of the carotid pulse correlates with  $S_1$  and can be used to help distinguish  $S_1$  from  $S_2$ .

The intensity (loudness) of  $S_1$  varies with the position of the atrioventricular (AV) valve leaflets at the beginning of ventricular systole and the structure of the leaflets (thickened or normal). A loud  $S_1$  is produced when the valve leaflets are wide open at the onset of ventricular systole and corresponds to a short PR interval on the surface electrocardiogram (ECG) tracing. A lengthening of the PR interval produces a soft  $S_1$  because the leaflets have had time to float partially closed before ventricular systole. Mitral stenosis also increases the intensity of  $S_1$  due to a thickening of the valvular structures.

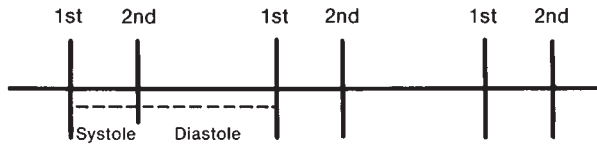
In general,  $S_1$  is heard as a single sound. If right ventricular systole is delayed, however,  $S_1$  may be split into its two component sounds. The most common cause of this splitting is delay in the conduction of impulses through the right bundle branch; the splitting correlates with a right bundle branch block (RBBB) pattern on the ECG. Splitting of  $S_1$  is heard best over the tricuspid area.

### Second Heart Sound

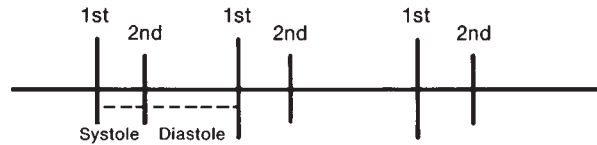
$S_2$  is produced by the vibrations initiated by the closure of the aortic and pulmonic semilunar valves and is heard best at the base of the heart (Fig. 17-5). This sound represents the beginning of ventricular diastole.

Like  $S_1$ ,  $S_2$  consists of two separate components. The first component of  $S_2$  is aortic valve closure; the second component is pulmonic valve closure. With inspiration, systole of the right ventricle is slightly prolonged because of increased filling of the right ventricle. This causes the pulmonic valve to close later than the aortic valve and  $S_2$  to

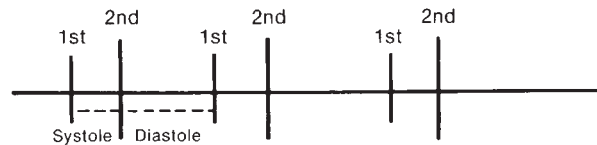




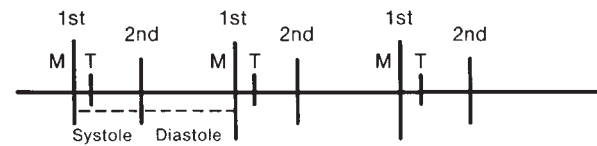
**Normal:**  $S_1$  is produced by the closure of the AV valves and correlates with the beginning of ventricular systole. It is heard best in the apical or mitral area.



**Loud First Sound:** The intensity of the first heart sound may be increased when the PR interval is shortened, as in tachycardia, or when the valve leaflets are thickened, as in mitral stenosis.



**Soft First Sound:** A soft  $S_1$  is heard when the PR interval is prolonged.



**Split First Sound:** A split  $S_1$  is heard when right ventricular emptying is delayed. The mitral valve closes before the tricuspid valve and “splits” the sound into its two components.

**figure 17-4** First heart sound.

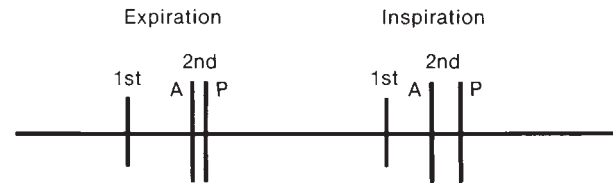
become “split” into its two components. This normal finding is termed *physiological splitting* and is heard best on inspiration with the stethoscope placed in the second intercostal space to the left of the sternum.

The intensity of  $S_2$  may be increased in the presence of aortic or pulmonic valvular stenosis or with an increase in the diastolic pressure forcing the semilunar valves to close, as occurs in pulmonary or systemic hypertension.

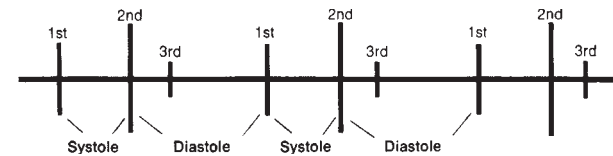
### Third Heart Sound

An  $S_3$  may be physiological or pathological (Fig. 17-6). A physiological  $S_3$  is a normal finding in children and healthy young adults; it usually disappears after 25 to 35 years of age. An  $S_3$  in an older adult with heart disease signifies ventricular failure.

An  $S_3$  is a low-frequency sound that occurs during the early, rapid-filling phase of ventricular diastole. A non-compliant or failing ventricle cannot distend to accept this rapid inflow of blood. This causes turbulent flow, result-



**figure 17-5** Second heart sound. The second heart sound is produced by the closure of the semilunar valves (aortic and pulmonary). During inspiration, there is an increase in venous return to the right side of the heart, which causes a delay in the emptying of the right ventricle and the closure of the pulmonic valve. This allows the two components of the second heart sound to separate or split during inspiration.

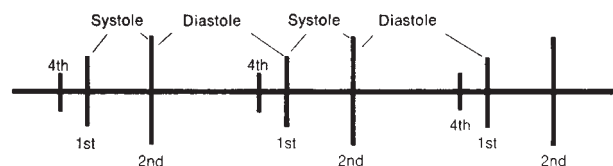


**figure 17-6** Third heart sound. An  $S_3$  or ventricular gallop is heard in early diastole, shortly after the second heart sound. The presence of a pathological  $S_3$  may be indicative of heart failure.

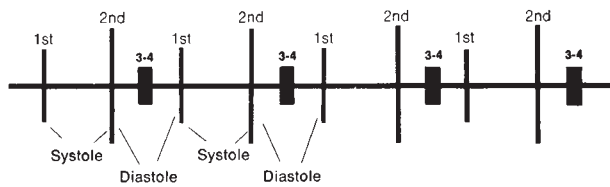
ing in the vibration of the AV valvular structures or the ventricles themselves, producing a low-frequency sound. An  $S_3$  associated with left ventricular failure is heard best at the apex with the stethoscope bell. The sound may be accentuated by turning the patient slightly to the left side. A right ventricular  $S_3$  is heard best at the xiphoid or lower left sternal border and varies in intensity with respiration, becoming louder on inspiration.

### Fourth Heart Sound

An  $S_4$  or atrial gallop is a low-frequency sound heard late in diastole just before  $S_1$ . It is rarely heard in healthy patients (Fig. 17-7). The sound is produced by atrial contraction forcing blood into a noncompliant ventricle that, by virtue of its noncompliance, has an increased resistance to filling. Systemic hypertension, MI, angina, cardiomyopathy, and aortic stenosis all may produce a decrease in left ventricular compliance and an  $S_4$ . A left ventricular  $S_4$  is auscultated at the apex with the bell of the stethoscope. Conditions affecting right ventricular compliance, such as pulmonary hypertension or pulmonic stenosis, may produce a right ventricular  $S_4$  heard best at the lower left sternal border; it increases in intensity during inspiration.



**figure 17-7** Fourth heart sound. An  $S_4$  is a late diastolic sound that occurs just prior to  $S_1$ . It is a low-frequency sound heard best with the bell of the stethoscope.



**figure 17-8** Summation gallop. With rapid heart rates,  $S_3$  and  $S_4$  may become audible as a single, very loud sound that occurs in mid-diastole. This sound is a summation gallop.

### Summation Gallop

With rapid heart rates, as ventricular diastole shortens, if  $S_3$  and  $S_4$  are both present, they may fuse together and become audible as a single diastolic sound. This is called a *summation gallop* (Fig. 17-8). This sound is loudest at the apex and is heard best with the stethoscope bell while the patient lies turned slightly to the left side.

### Heart Murmurs

Murmurs are sounds produced either by the forward flow of blood through a narrowed or constricted valve into a dilated vessel or chamber or by the backward flow of blood through an incompetent valve or septal defect. Murmur classification is based on timing in the cardiac cycle. Systolic murmurs occur between  $S_1$  and  $S_2$ . Diastolic murmurs occur after  $S_2$  and before the onset of the following  $S_1$ . Murmurs are described further according to the anatomical location on the anterior chest where the sound is heard the loudest. Any radiation of the sound also should be noted. The quality of the sound produced is described as blowing, harsh, rumbling, or musical (Box 17-5). The intensity or loudness of a murmur is described using a grading system. Grade I is faint and barely audible; grade II is soft; grade III

#### box 17-5

##### Attributes of Heart Murmurs

- **Timing:** A systolic murmur is heard between  $S_1$  and  $S_2$ . A diastolic murmur is heard between  $S_2$  and  $S_1$ .
- **Location of maximal intensity:** The nurse describes the anatomical location where the murmur is heard best. The location is identified based on intercostal space and its relation to the sternum, the apex, the mid-clavicular line, or one of the axillary lines.
- **Radiation or transmission from the point of maximal intensity:** The nurse notes the site farthest from the location of greatest intensity at which the sound is still heard. The farthest site is identified using anatomical landmarks as described above.
- **Pitch:** The terms *high*, *medium*, or *low* are used to describe the pitch of the murmur.
- **Quality:** Terms such as *harsh*, *raspy*, *vibratory*, *blowing*, or *musical* are used to describe the quality of the sound.
- **Intensity:** A grading system is used to describe the intensity of the murmur. See Box 17-6 for a summary of the grading system.

#### box 17-6

##### Gradation of Heart Murmurs

- Grade 1 Barely audible in a quiet room; very faint; may not be heard in all positions
- Grade 2 Quiet, but clearly audible
- Grade 3 Moderately loud
- Grade 4 Loud with a palpable thrill
- Grade 5 Very loud with an easily palpable thrill
- Grade 6 Very loud; may be heard with stethoscope entirely off of the chest; thrill palpable and visible

is audible but not palpable; grade IV and V murmurs are associated with a palpable thrill; and a grade VI murmur is audible without a stethoscope (Box 17-6).

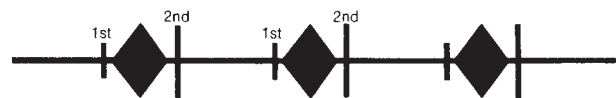
**Systolic Murmurs.** As previously described,  $S_1$  is produced by mitral and tricuspid valve closure and signifies the onset of ventricular systole. Murmurs occurring after  $S_1$  and before  $S_2$  are therefore classified as systolic murmurs.

During ventricular systole, the aortic and pulmonic valves are open. If either of these valves is stenotic or narrowed, a sound classified as a mid-systolic ejection murmur is heard. Because the AV valves close before blood is ejected through the aortic and pulmonic valves, there is a delay between  $S_1$  and the beginning of the murmur. The murmurs associated with aortic stenosis and pulmonic stenosis are described as crescendo–decrescendo or diamond shaped (Fig. 17-9), meaning that the sound increases and then decreases in intensity. The quality of these murmurs is harsh, and they are of medium pitch. The murmur caused by aortic stenosis is heard best in the aortic area and may radiate into the neck. The murmur of pulmonic stenosis is heard best over the pulmonic area.

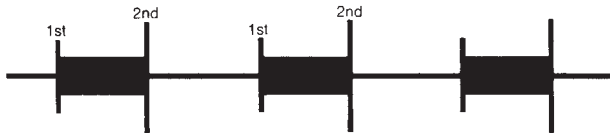
Systolic regurgitant murmurs are caused by the backward flow of blood from an area of higher pressure to an area of lower pressure. Mitral or tricuspid valvular insufficiency or a defect in the ventricular septum produces systolic regurgitant murmurs, which are harsh and blowing in quality. The sound is described as holosystolic, meaning that the murmur begins immediately after  $S_1$  and continues throughout systole up to  $S_2$  (Fig. 17-10).

Mitral insufficiency produces this type of murmur, heard most easily in the apical area with radiation to the left axilla. This type of murmur associated with tricuspid insufficiency is heard loudest at the left sternal border and increases in intensity during inspiration. This murmur may radiate to the cardiac apex.

A ventricular septal defect also produces a harsh, blowing holosystolic sound caused by blood flowing from the



**figure 17-9** Murmur associated with aortic or pulmonic stenosis. Blood flow through a stenotic aortic or pulmonic valve produces a crescendo–decrescendo midsystolic ejection murmur.



**figure 17-10** Murmur associated with tricuspid or mitral regurgitation. A holosystolic murmur is caused by the regurgitant flow of blood through an incompetent mitral or tricuspid valve. Flow of blood from the left ventricle to the right ventricle through a ventricular septal defect also produces this type of murmur.

left to the right ventricle through a defect in the septal wall during systole. The associated murmur is heard best from the fourth to sixth intercostal spaces on both sides of the sternum and is accompanied by a palpable thrill.

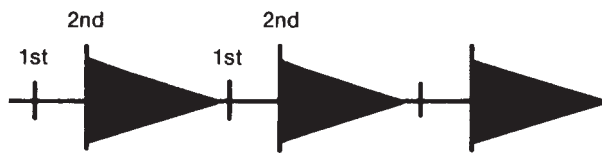
**Diastolic Murmurs.** Diastolic murmurs occur after  $S_2$  and before the next  $S_1$ . During diastole, the aortic and pulmonic valves are closed while the mitral and tricuspid valves are open to allow filling of the ventricles.

Aortic or pulmonic valvular insufficiency produces a blowing diastolic murmur that begins immediately after  $S_2$  and decreases in intensity as regurgitant flow decreases through diastole. These murmurs are described as early diastolic decrescendo murmurs (Fig. 17-11).

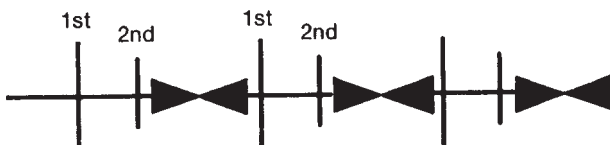
The murmur associated with aortic insufficiency is heard best in the aortic area and may radiate along the right sternal border to the apex. Pulmonic valve insufficiency produces a murmur that is loudest in the pulmonic area.

Stenosis or narrowing of the mitral or tricuspid valve also produces a diastolic murmur. The AV valves open in mid-diastole shortly after the aortic and pulmonic valves close, causing a delay between  $S_2$  and the start of the murmur of mitral and tricuspid stenosis. This murmur decreases in intensity from its onset and then increases again as ventricular filling increases because of atrial contraction; this is termed *decrescendo-crescendo* (Fig. 17-12).

The murmur associated with mitral stenosis is heard best at the apex with the patient turned slightly to the left side. Tricuspid stenosis produces a murmur that increases



**figure 17-11** Murmur associated with aortic or pulmonic insufficiency. Regurgitant flow through an incompetent aortic or pulmonic valve produces a diastolic decrescendo murmur.



**figure 17-12** Murmur associated with tricuspid or mitral stenosis. This low-frequency murmur is heard best with the bell of the stethoscope. It occurs after  $S_1$  and has a decrescendo-crescendo configuration.

in intensity with inspiration and is loudest in the fifth intercostal space along the left sternal border.

### Friction Rubs

A pericardial friction rub can be heard when the pericardial surfaces are inflamed. This high-pitched, scratchy sound is produced by these inflamed layers rubbing together. A rub may be heard anywhere over the pericardium with the diaphragm of the stethoscope. The rub may be accentuated by having the patient lean forward and exhale. A pericardial friction rub, unlike a pleural friction rub, does not vary in intensity with respiration.

## CARDIAC LABORATORY STUDIES

Knowledge of the purpose and significance of laboratory values in relation to the diagnosis and prognosis of acute MI can enhance the quality of nursing care available to patients. Laboratory studies include both routine serum analysis and special studies, such as serum and cardiac enzymes. Nurses who have a basic understanding of laboratory studies can exercise judgment in interpreting results relative to other information about the patient. The ability to use this kind of judgment may well affect the patient's clinical course or prognosis.

### Routine Laboratory Studies

Appropriate assessment of normal and compromised cardiac function is essential to ensure accurate evaluation and correct diagnosis of the patient experiencing symptoms consistent with a cardiovascular disorder or coronary artery disease. Nurses can more appropriately plan the care of the patient and initiate interventions if they have an understanding of these laboratory tests and recognize their implications. Valuable information may be obtained by assessing levels of hematological components, coagulation factors, electrolytes, and phospholipids. Determination of these laboratory studies may vary with institutional techniques and equipment used. Normal and abnormal assay ranges have been universally established, and a brief listing of frequently ordered laboratory studies with their normal values can be found in Table 17-1. A more extensive explanation of the effects of abnormal laboratory determinations is provided in other parts of this text and is not addressed here.

## HEMATOLOGICAL STUDIES

Accurate assessment of the patient with a possible cardiac disruption merits review of hematological function. It is important for the critical care nurse to understand the role of blood cells in cardiac function and their contribution to the maintenance of healthy tissue. Blood is the transport medium for nutrients, such as oxygen and glucose, as well as electrolytes, plasma proteins, hormones, and medications. It is also the vehicle for removal of the products of metabolism. Changes in blood cell integrity and total cell count may reflect specific disorders of the cardiac system and should be considered an integral part of the laboratory assessment.

**table 17-1** ■ Normal Reference Ranges for Laboratory Blood Tests

Blood Test	Reference Range	Blood Test	Reference Range
<b>Hematological Studies</b>		<b>Blood Chemistries—(cont.)</b>	
Red blood cell count		Bilirubin	
Men	4.6–6.2 × 10 <sup>6</sup>	Total	0.2–1.3 mg/dL
Women	4.2–5.4 × 10 <sup>6</sup>	Direct	0–20 mg/dL
Hematocrit		Calcium	
Men	40%–50%	Total	8.9–10.3 mg/dL
Women	38%–47%	Free (ionized)	4.6–5.1 mg/dL
Hemoglobin		Creatinine	
Men	13.5–18.0 g/100 mL	Men	0.9–1.4 mg/dL
Women	12.0–16.0 g/100 mL	Women	0.8–1.3 mg/dL
Corpuscle indices		Glucose (fasting)	65–110 mg/dL
Mean corpuscular volume	82–98 fL	Magnesium	1.3–2.2 mEq/L
Mean corpuscular hemoglobin	27–31 pg	Phosphorus	2.5–4.5 mg/dL
Mean corpuscular hemoglobin concentration	32%–36%	Phosphatase, alkaline	35–148 U
White blood cell count		Protein (total)	6.5–8.5 g/dL
Total	4,500–11,000/mm <sup>3</sup>	Urea nitrogen	8–26 mg/dL
Differential (in number of cells/mm <sup>3</sup> blood)		Uric acid	65–110 mg/dL
Total leukocytes	5,000–10,000 (100%)	Men	4.0–8.5 mg/dL
Total neutrophils	3,000–7,000 (60%–70%)	Women	2.8–7.5 mg/dL
Lymphocytes	1,500–3,000 (20%–30%)		
Monocytes	375–500 (2%–6%)	<b>Serum Enzymes*</b>	
Eosinophils	50–400 (1%–4%)	CK-MM	95%–100%
Basophils	0–50 (0.1%)	CK-MB	0%–5%
Sedimentation rate	0–30 mm/h	CK-BB	0%
		LDH-1	Dependent on assay technique ratio
		LDH-1: LDH-2 ratio	<1.0
<b>Coagulation Studies*</b>		Aspartate aminotransferase	<50 U/L
Platelet count	250,000–500,000/mm <sup>3</sup>		
Prothrombin time	12–15 s	<b>Myocardial Proteins</b>	
Partial thromboplastin time	60–70 s	Troponin-I	0–2 ng/mL
Activated partial thromboplastin time	35–45 s	Troponin-T	0–3.1 ng/mL
Activated clotting time	75–105 s	Myoglobin	
Fibrinogen level	160–300 mg/dL	Men	20–90 ng/mL
Thrombin time	11.3–18.5 s	Women	10–75 ng/mL
<b>Blood Chemistries</b>		<b>Cholesterol</b>	
Serum electrolytes		Total blood cholesterol	
Sodium	135–145 mEq/L	Desirable	<200 mg/dL
Potassium	3.3–4.9 mEq/L	Borderline high	200–239 mg/dL
Chloride	97–110 mEq/L	High	≥240 mg/dL
Carbon dioxide	22–31 mEq/L	LDL cholesterol	
Blood gases		Desirable	<130 mg/dL
pH	7.35–7.45	Borderline high	130–159 mg/dL
PaO <sub>2</sub>	80–105 mm Hg	High	≥160 mg/dL
Paco <sub>2</sub>	35–45 mm Hg	HDL cholesterol	>35 mg/dL
Bicarbonate	22–29 mEq/L	Apolipoprotein A	120 mg/dL
Base excess, deficit	0±2.3 mEq/L	Apolipoprotein B	134 mg/dL
Sao <sub>2</sub>	98%		
Sv̄CO <sub>2</sub>	75%		

\*Examples: regional laboratory techniques and methods may result in variations.

Knowledge of normal blood values is vital to understand deviations from normal that can be seen with various cardiac disruptions. It is necessary to review both the red blood cell count, which assesses cellular nutrition, and the white cell count, which assesses defensive capability against infections, when diagnosing specific insults. Table 17-1 lists the components of these helpful hematological studies.

## COAGULATION STUDIES

Coagulation studies are also warranted in the laboratory assessment of patients with cardiac disease (see Table 17-1). Establishment of a baseline for coagulation function provides important information about the patient's ability to form, maintain, and dissolve blood clots. Such information may prove instrumental in patient care decisions. This is



especially true in relation to the administration of anti-coagulation agents, whether for long-term management, such as warfarin for the management of atrial fibrillation, or for emergency interventions, such as the use of fibrinolytic therapy during an acute MI.

## BLOOD CHEMISTRIES

Mechanisms that ensure homeostatic function at the cellular and tissue level depend on the appropriate production and modulation of intracellular and extracellular electrolytes. It is important that the nurse understand normal electrolyte functions and the unique, perhaps life-threatening, situations that may occur when they are significantly abnormal. A thorough analysis of basic electrolyte chemistries is always appropriate in screening of the patient with cardiac disease, whether in the inpatient or outpatient setting. These studies are almost universally obtained during the initial clinical examination. The blood chemistries most commonly assessed are sodium, potassium, chloride, carbon dioxide, calcium, glucose, magnesium, and phosphorus. Table 17-1 provides the normal assay values for common electrolytes.

### Common Electrolytes

Sodium is the most abundant cation in the body. It is essential in the maintenance of acid–base balance and osmolality of extracellular fluids as well as in the transmission of nerve impulses. It plays a pivotal role in fluid balance, and its concentration is primarily regulated by the kidneys. Significant alterations of cellular function are evident when sodium levels are lower than normal (hyponatremia) or greater than normal (hypernatremia).

Potassium is the major intracellular cation. Its role in the evaluation of cardiac patients is important because it is released when cells are damaged. It is essential for maintenance of oncotic pressure, intracellular osmolality, and acid–base balance, as well as for its role in cellular reactions. In addition, potassium is vital to the normal functioning of skeletal, smooth, and cardiac muscle. It is particularly important in the regulation of cardiac rate and force of contraction.

Chloride is another major extracellular cation. Like sodium and potassium, it plays a role in acid–base balance and is an important component in the evaluation of acid–base balance.

The carbon dioxide electrolyte is a reflection of carbon dioxide *content* (mainly bicarbonate), not carbon dioxide *gas*. In some settings, carbon dioxide is reported as bicarbonate ( $\text{HCO}_3^-$ ).

### Other Blood Chemistries

Calcium, like potassium, is important for cardiac function. It plays a significant role in the initiation and propagation of electrical impulses and in myocardial contractility. It is also important for blood clotting, teeth and bone formation, and intracellular energy production. Ionized calcium (free calcium) is responsible for cardiac and neuromuscular excitability. Calcium is reported as total and free (ionized) values.

Glucose levels are important to monitor with baseline laboratory studies because they reflect the nutritive status of the cell. Alterations in glucose, such as in diabetes mel-

litus, can provide the clinician with both diagnostic as well as prognostic information.

Magnesium is the second major intracellular cation after potassium. It is important in many metabolic processes and is necessary for the normal functioning of the neuromuscular system. It facilitates enzyme activities, which help maintain protein synthesis and metabolism, carbohydrate and lipid metabolism, and nucleic acid synthesis. Alterations in normal magnesium levels are reflected in disruptions in neuromuscular activity, such as in the patient with arrhythmia.

Phosphorus reflects levels of serum phosphate. It is controlled by the parathyroid gland and regulated in the kidneys. Phosphate is important for normal cellular function and for oxygen delivery. It is reciprocal to calcium. Abnormalities can be seen with alterations in heart rate, alterations in neuromuscular function, and reciprocal changes in serum calcium.

## SERUM LIPID STUDIES

A review of serum lipid levels can provide the nurse with a perspective on cardiovascular risk for the patient presenting with a cardiac event. Measurement of cholesterol, low-density and high-density lipoprotein, and triglycerides can aid in the evaluation for the presence of atherosclerosis and coronary artery disease. (See Table 17-1 for a list of normal lipid values.) Cholesterol, a precursor of bile acids and steroid hormones, can accumulate in arterial walls, where it can be atherogenic. Cholesterol levels vary with age, diet, level of activity, and stress. Low-density lipoproteins (LDLs), which represent 60% to 70% of the total serum cholesterol found in the serum, carry plasma cholesterol. Higher levels of LDL are associated with a higher risk for the development of cardiovascular disease. High-density lipoproteins (HDLs) are responsible for carrying 20% to 30% of the total serum cholesterol. HDL has been implicated in a protective role against atherogenesis and appears to have an inverse relationship with the development of coronary artery disease. Higher levels of HDL are associated with decreased risk of coronary heart disease. Factors that influence the serum levels of LDL and HDL can be found in Table 17-2. Triglycerides represent stored fat in tissues. The normal range for triglycerides is 40 to 200 mg/dL. Levels greater than 200 mg/dL can contribute to the development of atherosclerosis and coronary artery disease.

## Enzyme Studies

Enzymes are found in all living cells and act as catalysts in biochemical reactions. They are present in low amounts in the serum of healthy individuals. However, when cells are injured, enzymes leak from damaged cells, resulting in serum enzyme concentrations greater than the usual low levels. No single enzyme is specific to the cells of a single organ. Each organ contains a variety of enzymes, and there is considerable overlap among organs in the enzymes they contain. However, the distribution of enzymes in the cells of organs is relatively organ specific. When organ damage occurs, the presence of abnormally high levels of enzymes in the serum, their distribution, and the timing of their appearance and disappearance make the clinical use of serum enzyme studies relevant.

**table 17-2** ■ Factors That Influence Low-Density Lipoprotein (LDL) and High-Density Lipoprotein (HDL) Levels

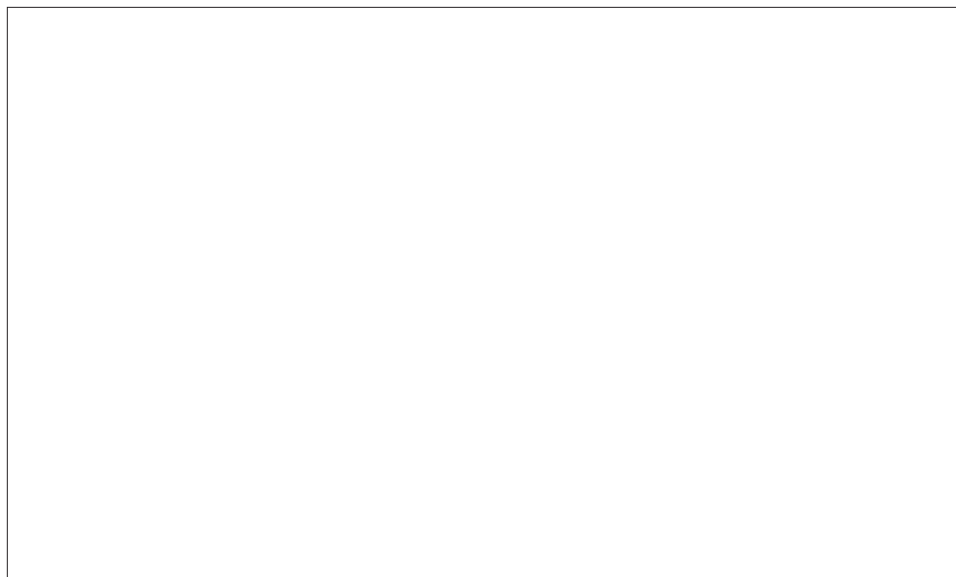
LDL Levels	HDL Levels
Increased with	Increased with
Diets high in cholesterol	Not smoking
Diets high in saturated fat	Lean body mass
Alcohol	Estrogen
Strict vegetarian diet	Vigorous exercise
Hypothyroidism	Diet low in sucrose and starch
Obesity	Increased clearance of very-low-density lipoprotein (triglyceride)
Obstructive liver disease	Alcohol
Nephrosis	Decreased with
Thiazide diuretics	Cigarette smoking
Beta-adrenergic blocking agents	Obesity
Progestin and anabolic steroids	Progesterone
Decreased with	Male sex
Low-cholesterol diet	Sedentary lifestyle
Low-fat diet	Hypertriglyceridemia
Alcohol restriction	Type 2 diabetes mellitus
Regular strenuous exercise	Strict vegetarian diet
	Hypertriglyceridemia
	Anabolic steroids
	Starvation
	Beta-adrenergic blocking agents
	Infectious illness

From Baker M: Laboratory tests using blood. In Woods S, Froelicker E, Motzer S (eds): *Cardiac Nursing* (4th Ed). Philadelphia, Lippincott Williams & Wilkins, 2000.

Cardiac enzymes are enzymes found in cardiac tissue. When cardiac injury occurs, as in acute MI, these enzymes are released into the serum, and their concentrations can be measured (Fig. 17-13). Cardiac tissue enzymes are present in other organs as well, so elevation of one or more of these enzymes is not a specific indicator of cardiac injury. Because cardiac damage does result in above-normal serum concentrations of these enzymes, however, the quantifica-

tion of cardiac enzyme levels, along with other diagnostic tests and the clinical presentation of the patient, is routinely used for diagnosing cardiac disease, particularly acute MI.

Only three of the many enzymes present in cardiac tissue have widespread use in the diagnosis of acute MI: creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate aminotransferase (AST; previously termed serum glutamic oxaloacetic transaminase [SGOT]). LDH and



**figure 17-13** Peak elevation and duration of serum enzymes after acute myocardial infarction. (Data from Antman EM: *Acute myocardial infarction*. In Braunwald E [ed]: *Heart Disease: A Textbook in Cardiovascular Medicine* [5th Ed], pp 1184–1228. Philadelphia: W. B. Saunders, 1997. Used with permission.)

AST were first used in the 1950s. Since the mid-1960s, CK has become the most important addition to the enzyme diagnosis of acute MI. None of the three enzymes is specific to cardiac tissue. However, CK and LDH can be divided further into components called *isoenzymes*. In each case, at least one of the isoenzymes is more specific for cardiac disease. The increase in these more specific components of LDH or CK relative to their other isoenzymes has resulted in their common use in diagnosis of acute MI. Because of the nonspecificity of AST and the widespread availability of CK and LDH isoenzymes, the routine sampling of serum for AST for the diagnosis of acute MI is no longer recommended. The value of drawing blood samples for measuring CK, LDH, and their associated isoenzymes for the diagnosis of acute MI is discussed in the following text.

### CREATINE KINASE

The level of total CK in plasma usually becomes abnormal 6 to 8 hours after onset of MI and peaks in 24 and 28 hours. When patients appear at the hospital soon after the onset of symptoms, the initial CK frequently is within normal limits, and at the time of hospital admission, it is not possible to discriminate on the basis of CK those who are having an acute MI from those who are not. For this reason, CK is sampled every 4 to 6 hours for the first 24 hours after the onset of symptoms. Within 2 to 4 days after MI, the serum concentration of total CK usually has returned to normal. Therefore, abnormal total CK levels may be missed in patients who present more than 24 hours after the onset of infarction. The normal level of total CK typically is higher in men than in women and in African Americans than in whites. The upper limit of normal may vary among laboratories, and nurses must be aware of the normal value used in their laboratory. In general, the amount of total CK correlates with the amount of myocardial damage and is of prognostic importance. With a small infarction, total CK may increase to two to three times the initial level, but never reach the upper limit of normal, and CK isoenzymes may be valuable.

Skeletal muscle contains more CK than the heart, whereas the cerebral cortex has slightly less CK. Conditions that result in damage or injury to the brain or skeletal muscle also may result in abnormal levels of CK in plasma. Skeletal muscle diseases, such as polymyositis and muscular dystrophy, and the effects of alcohol, strenuous exercise, convulsions, trauma, surgery, and intramuscular injections in skeletal muscle may give rise to abnormal CK levels. Cerebrovascular disease also may result in abnormal CK levels. In cerebrovascular disease, the increase occurs later, lasts longer, and is not as high as the abnormal CK levels caused by acute MI. Although the clinical presentation of the patient, the ECG, and the amount and time course of abnormal CK levels are useful in determining whether the diagnosis of acute MI is appropriate, it is not always possible to distinguish MI from other clinical conditions. For this reason, CK isoenzymes, in addition to total CK, usually are obtained serially.

#### *Creatine Kinase Isoenzymes*

Electrophoresis, glass bead, and radioimmunoassay are techniques used to measure CK isoenzymes. The three CK isoenzymes routinely reported are CK-MM, CK-BB, and

CK-MB, which are found to the greatest extent in skeletal muscle, brain, and heart muscle, respectively. Total CK usually consists entirely of CK-MM, and neither CK-BB nor CK-MB is present. In other words, skeletal muscle accounts for the normal levels of CK found in healthy individuals. Normal skeletal muscle may contain up to 2% CK-MB, and values of CK-MB of as much as 5% are not necessarily considered diagnostic. The amount of CK-MB in cardiac muscle is 15% to 22%, with the remainder being CK-MM. When cardiac damage occurs, as in acute MI, total CK increases, and the percentage of CK-MB is greater than 5%. Although other organs, such as the tongue, small intestine, uterus, and prostate, contain CK-MB, the presence of CK-MB in amounts greater than 5% generally is considered diagnostic for myocardial damage in the presence of chest pain or other symptoms believed to represent myocardial ischemia.

Within 6 to 12 hours after the onset of infarction, CK-MB usually begins to appear in serum, and it peaks at approximately 24 hours. However, the appearance and peak may be significantly earlier in patients who have a non-Q-wave infarction or who have undergone successful recanalization of the infarct-related coronary vessel by angioplasty or thrombolytic therapy. Patients who present more than 24 hours after the onset of symptoms may not benefit from measurement of CK isoenzymes because the levels already may have returned to normal. As with total CK, serial sampling should be performed every 4 to 6 hours for the first 24 hours after the onset of symptoms. Patients who continue to have signs or symptoms of myocardial ischemia after hospital admission should continue to undergo serial CK isoenzyme sampling.

Most laboratories report the absolute amount of each CK isoenzyme present in the serum, although some also report the percentage. Normal values for absolute amounts of each of the CK isoenzymes vary by laboratory and by the measuring technique used. The amount of CK-MB released into the serum after an acute MI offers a better correlation with infarction size than total CK because of its specificity to cardiac muscle.

Because total CK and CK isoenzymes are the cardiac enzymes whose levels become abnormal earliest after the onset of infarction, routine serial sampling for other cardiac enzymes is unnecessary. Serial analysis of CK isoenzymes is the most specific, sensitive, and cost-effective means of diagnosing acute MI. Perhaps more important, CK isoenzymes also have made it possible to “rule out” an acute MI more quickly and reliably. It no longer requires 2 to 3 days of intensive care unit (ICU) hospitalization to determine that AST or LDH enzyme levels remain normal; rather, an acute MI can be ruled out in less than 24 hours if a patient’s CK isoenzyme levels do not become abnormal. Nurses and physicians not only are able to provide earlier reassurance to patients who are found not to have acute infarction, but patients can be discharged sooner to a less costly environment than the ICU.

Clinical presentation of the patient and the ECG usually are helpful in distinguishing patients with acute MI. Cardiac disorders other than acute MI, including pericarditis, myocarditis, and trauma, also may be associated with abnormal total CK and CK-MB levels. In addition, CK-MB levels have been reported to be abnormal after cardiac surgery and cardioversion.

### *Creatine Kinase Isoforms*

The isoenzymes CK-MB and CK-MM may be divided further into isoform or subform components using electrophoretic or immunoassay techniques. Laboratory performance of these tests is time-consuming and labor intensive. However, they are used clinically in some hospitals because isoforms may offer confirmation or exclusion of MI earlier than CK isoenzymes. Efforts are underway to develop faster and less labor-intensive CK isoform measurements that will result in their widespread clinical use.

Two subforms of CK-MB and three subforms of CK-MM have been identified. Because CK-MB is more specific to cardiac muscle than CK-MM, CK-MB isoforms are appropriate for patients with suspected MI. CK-MB<sub>2</sub> (tissue CK-MB) is released into the serum and converted to CK-MB<sub>1</sub> (plasma CK-MB) by carboxypeptidase N, another enzyme present in serum. In patients without MI, the amounts of CK-MB<sub>2</sub> and CK-MB<sub>1</sub> present in serum are small, and the ratio of CK-MB<sub>2</sub> to CK-MB<sub>1</sub> is approximately one. In patients with acute MI, CK-MB<sub>2</sub> is released into the serum in larger quantities than normal and the amount of CK-MB<sub>2</sub> and the ratio of CK-MB<sub>2</sub> to CK-MB<sub>1</sub> in the serum of these patients becomes elevated.

Abnormal elevations of CK-MB<sub>2</sub> have been reported as early as 2 hours after onset of symptoms of MI. In this study, in patients with acute MI, 59% had diagnostic CK-MB isoforms on serum samples obtained within 2 to 4 hours of onset of symptoms, whereas only 23% were diagnosed by the conventional CK isoenzyme assay. In addition, in patients in whom blood samples were obtained within 8 hours of onset of symptoms, CK-MB isoforms were positive in 100% of cases, compared with 71% for CK isoenzymes.

Because the ratio of CK-MB<sub>2</sub> to CK-MB<sub>1</sub> may remain elevated only for up to 12 hours after onset of infarction, CK-MB isoforms likely will not be as useful as CK isoenzymes in patients presenting to the hospital more than 12 hours after the onset of symptoms. Nevertheless, the importance of a reliable early laboratory marker of infarction cannot be underestimated. The period of early sensitivity of CK-MB isoforms is similar to the therapeutic window for the administration of thrombolytic therapy and may be useful in identifying additional patients who could benefit. Also, acute MI can be excluded as a diagnosis within 8 hours of symptom onset using CK-MB isoform assay, compared with the 18 to 24 hours required for the conventional CK isoenzyme assay (Table 17-3).

### **LACTATE DEHYDROGENASE**

LDH can be found in many organs besides the heart, including the liver, skeletal muscle, kidney, lung, fat, and red blood cells. Because LDH is found in several other tissues in addition to the heart, it is no longer considered a specific test for MI and is not ordered as frequently today. However, LDH is used to help diagnose myocarditis, liver and renal dysfunction, and skeletal muscle disorders or trauma. It may be abnormally elevated in various conditions, including hemolytic anemia; pulmonary infarction; renal infarction; hepatic disorders, such as hepatitis and hepatic congestion; and skeletal muscle disorders.

### *Lactate Dehydrogenase in Cardiac Disease*

Total LDH is less specific than CK for cardiac disease. It usually begins to appear in the serum within 24 hours after the onset of acute MI and does not peak until 2 to 3 days; it may remain elevated for 7 to 10 days before returning to normal levels. The use of LDH in the diagnosis of acute MI is unnecessary if the diagnosis can be confirmed by CK and CK-MB. Patients who present more than 24 hours after the onset of symptoms (CK and CK-MB levels already may have returned to normal) or those who have been having symptoms of myocardial ischemia for several days may be appropriate for sampling for total LDH and LDH isoenzymes. LDH may help confirm an MI that occurred several days earlier. Although LDH increases more slowly and remains elevated longer than CK, the time course of abnormal levels for both enzymes overlaps. A single sample for LDH may be obtained in patients who present more than 24 hours after symptom onset. If the LDH value is abnormally elevated, the sample may be further analyzed for LDH isoenzymes. Routine serial sampling of LDH or LDH isoenzymes in these patients is not recommended because in the face of nondiagnostic CK isoenzymes, there is no evidence that serial LDH or LDH isoenzyme sampling improves the diagnostic yield.<sup>1</sup>

Care must be taken when obtaining blood samples for LDH because hemolysis may result in LDH being released from red blood cells, causing falsely elevated levels. The upper limit of normal for LDH, like CK, is higher in men than women and varies by laboratory. Nurses must know the normal values for their laboratory.

### *Lactate Dehydrogenase Isoenzymes*

Although LDH isoenzymes are not as specific as CK isoenzymes in the diagnosis of acute MI, they nevertheless are helpful in patients whose CK isoenzyme levels may have returned to normal. Electrophoretic techniques are used to measure LDH (see Table 17-3). The isoenzyme that moves most quickly toward the positive pole of the electrical field, LDH<sub>1</sub>, is found most abundantly in heart muscle. Somewhat lower amounts of LDH<sub>1</sub> are present in kidney, brain, and red blood cells. LDH<sub>5</sub>, the LDH isoenzyme that moves most slowly toward the positive electrode, is found most abundantly in liver and skeletal muscle. LDH<sub>2</sub>, LDH<sub>3</sub>, and LDH<sub>4</sub> are present in intermediate amounts in these organs between the extremes of LDH<sub>1</sub> and LDH<sub>5</sub>. In healthy individuals, LDH<sub>1</sub> makes up between 17% and 27% of total LDH, whereas LDH<sub>2</sub> comprises 28% to 38%; LDH<sub>1</sub> is always present in a lesser percentage than LDH<sub>2</sub>. Because the heart contains relatively more LDH<sub>1</sub> than LDH<sub>2</sub>, the ratio of the percentage of LDH<sub>1</sub> to LDH<sub>2</sub> usually becomes one or more whenever cardiac injury occurs. This “flip” in the ratio of the percentage of LDH<sub>1</sub> to LDH<sub>2</sub> occurs 1 to 3 days after the onset of MI.

### **DIAGNOSTIC LIMITATIONS TO ENZYME STUDIES**

Enzyme determinations can serve only as adjuncts to ECG and clinical diagnosis. To be of most value, they should be used with discretion. Consideration must be given to the length of time since onset of symptoms occurred because each enzyme rises and returns to normal at different intervals.



**table 17-3 ■ Biochemical Markers for Diagnosing Acute Myocardial Infarction**

Test and Description	Time to Run Test	First Detectable in Serum (After Onset of Symptoms)	Peak Levels and Normal Values	Time Required for Reliable Diagnosis*
CK-MB	Electrophoresis: 1–2 h	Electrophoresis: 4–8 h	Electrophoresis: 10–24 h nl = 24–195 IU/L	8–12 h
MB isoenzyme of CK (more abundant in myocardial tissue)	Monoclonal antibody test: 10–40 min	Monoclonal antibody test: 2–3 h	Monoclonal antibody test: 10–18 h nl = 0–9 ng/mL	2–3 h
MB2/MB1 ratio Ratio of subforms of CK-MB isoenzymes: MB2 in the tissue and MB1 in the plasma	25 min	1–6 h; may be detected as early as <1 h	4–8 h nl = 1 : 1	1 h
LDH	Varies <sup>†</sup>	Within 24 h	2–4 days; nl = 80–120 <sup>†</sup> nl = 71–IU/L <sup>†</sup>	2–3 days
Isoforms of LDH LDH <sub>1</sub> LDH <sub>2</sub> LDH <sub>3</sub> LDH <sub>4</sub> LDH <sub>5</sub>			normal values <sup>†</sup> nl = 17–27% nl = 28–28% nl = 16–20% nl = 2–3% nl = 0.5–1.5%	
Myoglobin Heme protein (found in myocardial and skeletal muscle)	10 min	2–3 h; may be detected as early as <1 h	2–4 h (cyclic rise and fall) nl = 30–90 ng/mL	2h
Troponin I, troponin T Contractile protein (found in myofibrils)	30 min	4–6 h	10–24 h; elevated 5–14 days 2–3 h Troponin I elevated for 5–7 days; troponin T elevated for 10–14 days nl = 0–0.4 ng/mL	2–3 h

\* Refers to the minimal time after onset of symptoms.

<sup>†</sup> Values vary according to laboratory performing the test

From Apple S: Advanced strategies for diagnosing acute myocardial infarction. Heartbeat 6:1–12, 1995; adapted and updated from various resources.

Enzyme determinations have been of greatest value to patients whose ECG and clinical picture are equivocal for diagnosis of MI. Enzyme elevation may well confirm a suspected diagnosis. Sometimes it is difficult or impossible to interpret infarction on ECG because of previous infarction changes; effects of certain drugs or electrolyte imbalances; conduction defects, such as bundle branch block or Wolff-Parkinson-White syndrome; arrhythmias; or a functioning pacemaker. Enzyme determination may be a distinct advantage in such a setting. If a definite diagnosis can be made by ECG, enzyme tests may not be needed, except for academic and prognostic interest.

Because serum enzyme elevations are nonspecific in the diagnosis of MI, they must be considered in view of the total clinical picture. We are in a highly technical age of nursing and must not forget to look at and listen to the patient before making judgments and decisions.

### Biochemical Markers: Myocardial Proteins

Two newer diagnostic markers are being used in the laboratory for evaluating myocardial damage. Myoglobin and troponin levels have shown high specificity for detecting myocardial damage. Both proteins are evaluated using the same monoclonal antibody technique used for CK-MB subform quantification. Table 17-1 presents the normal values for these proteins.

Myoglobin detection can be helpful in the determination of ischemic muscle damage. Found in skeletal and cardiac muscle, it appears in the serum earlier than CK and may be detectable in the serum less than 1 hour after the onset of symptoms. Myoglobin usually peaks in 2 to 4 hours. It is detected for *any* muscle damage and is not specific for cardiac muscle damage alone. This characteristic facilitates

its value in the *exclusion* of an MI. Myoglobin levels may be very sensitive to reperfusion events after thrombolytic therapy and thereby prove to be of significant clinical value in this intervention.

Troponin is highly specific for cardiac muscle damage and is detectable in two subforms, cardiac troponin I (cTnI) and troponin T (cTnT). An advantage of troponin assays is that, unlike myoglobin, troponin is unaffected by skeletal injury. Troponin I can be detected within 4 hours after an acute MI and can remain elevated for 7 to 10 days. In a healthy person, this contractile protein is not detectable, and its high sensitivity and specificity can provide rapid diagnostic information in the patient with a suspected acute MI. Troponin values may be greater than 10 ng/mL in the presence of an acute MI. Lower levels (0.4 ng/dL) are often seen with lesser insults, such as angina pectoris. Troponin I may play a role in predicting an increase in mortality in non-Q-wave infarctions and unstable angina. Troponin T has been shown to be highly sensitive in detecting minor myocardial injury and may provide valuable prognostic information in patients experiencing angina pectoris. It has been reported that troponin T may detect smaller amounts of myocardial damage than even high-quality serial echocardiograms and may remain elevated in the plasma for 10 to 14 days after myocardial insult.

### Neurohumoral Hormones: Brain-type Natriuretic Peptide

When the cardiac muscle decompensates, hormones are released from extracardiac and cardiac origins. Norepinephrine and endothelin are hormones released as a peripheral response to cardiac impairment. Natriuretic peptides are neurohumoral hormones released by the heart. Atrial natriuretic peptides are secreted as a result of atrial myocardial distension, but only minute amounts are released in response to ventricular distension. Another neurohumoral hormone, brain-type natriuretic peptide (BNP), was first isolated in the porcine brain—hence the “B.” Although the human brain does secrete BNP, the primary site of release is in the cardiac ventricles, with only very small amounts released in the atria. BNP is released in response to ventricular dilation and increased intraventricular pressures.<sup>2</sup>

BNP levels are helpful in the diagnosis of ventricular dysfunction caused by heart failure. Results of the blood test that measures endogenous levels of BNP may be available in about 30 minutes, making this test especially useful for diagnosing heart failure in the emergency department. Endogenous BNP levels diagnose decompensated heart failure with 95% accuracy.<sup>3</sup> For patients with an acute MI, BNP levels increase rapidly during the first 24 hours and then plateau.<sup>4</sup> Elevated levels of BNP provide important prognostic information for the patient with acute coronary syndromes. In acute MI and unstable angina, elevated BNP levels are predictive of a greater risk of death, postinfarction heart failure, or reinfarction.<sup>5</sup>

### Newer Diagnostic Markers

C-reactive protein and P-selectin are newer markers of inflammation and necrosis that have been implicated as factors that can cause disruption of fibrous cap lesions under-

lying acute coronary events. C-reactive protein, an acute-phase protein and marker of systemic inflammation, has been shown to be elevated in patients with acute coronary syndromes.<sup>6,7</sup> Normal values are 0 to 2 mg/dL. Serum values greater than 3 mg/dL in patients with acute coronary syndrome or greater than 5 mg/dL in patients who are post-coronary interventional procedure may indicate a higher risk and merit closer monitoring or more thorough evaluation.<sup>6</sup>

D dimer is another newer physiological marker that may be useful in predicting the risk of cardiac events. D dimer represents the end product of thrombus formation and dissolution that occurs at the site of active plaques in acute coronary syndromes; this process precedes myocardial cell damage and release of protein contents.<sup>8,9</sup> It is thought that D dimer, which is detected early and remains elevated for days, can identify unstable plaque in high-risk patients when troponin and CK-MB have not yet been released.<sup>9</sup> Universal normal serum values for D dimer are not yet established, although a threshold of 500 µg/L indicates increased sensitivity for acute MI.<sup>9</sup> Studies are underway to determine the effectiveness of D dimer in identifying patients who may benefit from the use of anticoagulant or antiplatelet therapy.

### case study ■ ACUTE ANTEROSEPTAL MYOCARDIAL INFARCTION

Mrs. James, a 74-year-old widow, is being admitted to the coronary care unit from the emergency department with a diagnosis of acute anteroseptal MI. Her history is significant for the onset of chest pain more than 4 hours before she arrived in the emergency department.

Symptom assessment in the emergency department, using the “NOPQRST” parameters described in Box 17-1, reveals the following:

- **Normal:** No history of chest pain
- **Onset:** 1 PM.
- **Precipitating and palliative factors:** No pain relief with rest
- **Quality and quantity:** Pain described as heaviness in the chest
- **Region and radiation:** Mid-sternal pain that radiates to the back
- **Severity:** Pain described as 7/10
- **Time:** Patient in pain for 4 hours before she called an ambulance

The patient has a history of hypertension and hypercholesterolemia. She currently takes atenolol and atorvastatin (Lipitor). Previous surgeries include a hysterectomy and a right hip replacement. Mrs. James lives alone and has two daughters who live nearby.

Important findings on physical examination are:

- **Vital signs:** temperature 99°F (37.2°C); respirations 24 breaths/minute; pulse rate 98 beats/minute and regular; blood pressure 164/92 mm Hg
- **Neck:** jugular venous distension

- Cardiac: S<sub>1</sub>, S<sub>2</sub>, regular rhythm, and an audible S<sub>3</sub>
- Respiratory: fine crackles in the bases
- Extremities: +1 pedal edema

Laboratory studies reveal an elevated CK with positive MB, elevated myoglobin (100 ng/mL), and elevated troponin (0.8 ng/mL).

The nurse in the emergency department tells you to expect to receive Mrs. James in about 30 minutes. She has O<sub>2</sub> at 2-L nasal cannula with an intravenous (IV) line of 5% dextrose in water (D<sub>5</sub>W) at 90 mL/hour. ■

## CARDIAC DIAGNOSTIC STUDIES

Cardiovascular diagnostic techniques have expanded dramatically in the past few years, especially in the area of non-invasive testing. This permits a more careful screening of the population for high-risk procedures and low-risk methods for monitoring disease progression and response to treatment. In addition, many technologies are combined for a functional assessment of the patient's cardiac status so the best treatment option can be chosen.

The critical care nurse often cares for patients who undergo one or more of these procedures. Understanding the principles on which the procedures are based enables the nurse to answer questions, incorporate diagnostic findings into the patient's plan of care, and provide high-level nursing care. The critical care nurse also can decrease the anxiety of patients and their families by providing an explanation of the procedure.

### Noninvasive Techniques

#### STANDARD 12-LEAD ELECTROCARDIOGRAM

##### *Purpose*

The standard ECG records electrical impulses as they travel through the heart. In patients with normal conduction, the first electrical impulse for each cardiac cycle originates in the sinus node and is spread to the rest of the heart through the specialized conduction system—the intra-atrial tracts, AV node, bundle of His, and right and left bundles. As the impulse traverses the conduction system, it penetrates the surrounding myocardium and provides the electrical stimuli for atrial and ventricular contraction. The change in electrical potential in cells of the specialized conduction system as the impulse proceeds is very small and cannot be measured from electrodes outside the body. However, the change in electrical potential of myocardial cells produces an electrical signal that can be recorded from the surface of the body, as is done with an ECG.

Impulses that originate in sites other than the sinus node or impulses that are prevented from traversing the conduction system because of disease or drugs interrupt the normal order of electrical sequences in the myocardium. An ECG may be used to record these abnormal patterns of impulse formation or conduction. A clinician then has a visual record of the abnormal pattern from which to identify the arrhythmia.

In addition, an abnormal ECG tracing may result from diseased myocardial cells. For example, in patients with left ventricular hypertrophy (LVH), impulses traversing the enlarged muscle mass of the left ventricle produce a

larger electrical signal than normal. In contrast, impulses are unable to traverse myocardial cells that are irreversibly damaged, such as in MI, and no electrical signal is present in the infarcted cells of the left ventricle.

##### *Procedure*

The standard 12-lead ECG is so named because the usual electrode placement and recording device permit the electrical signal to be registered from 12 different views. The four limb and six precordial leads are attached to the patient as shown in Figure 17-14. For the limb leads, the recording device alternates the combination of electrodes that are active during recording of electrical signals from the heart (Fig. 17-15). This results in six standard views or leads (I, II, III, augmented voltage of the right arm [aVR], augmented voltage of the left arm [aVL], and augmented voltage of the left foot [aVF]) that are recorded in the heart's frontal plane. The six precordial leads (V<sub>1</sub>, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>) are arranged across the chest to record electrical activity in the heart's horizontal plane (see Fig. 17-14). Abnormal localized areas of myocardial conduction, such as occur with ischemia or infarction, may be identified in the leads that are nearest to that part of the heart. For example, an inferior MI produces changes in the leads that view the inferior aspect of the heart, or leads II, III, and aVF.

Used routinely in ICU patients, ECGs assess arrhythmias and myocardial ischemia or MI. An ECG is performed easily at the bedside with the patient ideally placed in the supine position and the electrodes arranged as previously described. In some patients, chest bandages may preclude placement of the precordial leads. It is important that the patient remain still during the ECG recording so that skeletal muscle movement does not result in extraneous noise or artifact in the electrical signal. Additional horizontal plane leads may be recorded by placing electrodes on the right side of the chest to view right ventricular activity or the back of the chest to view left ventricular posterior wall activity (see Fig. 17-14).

##### *Nursing Assessment and Management*

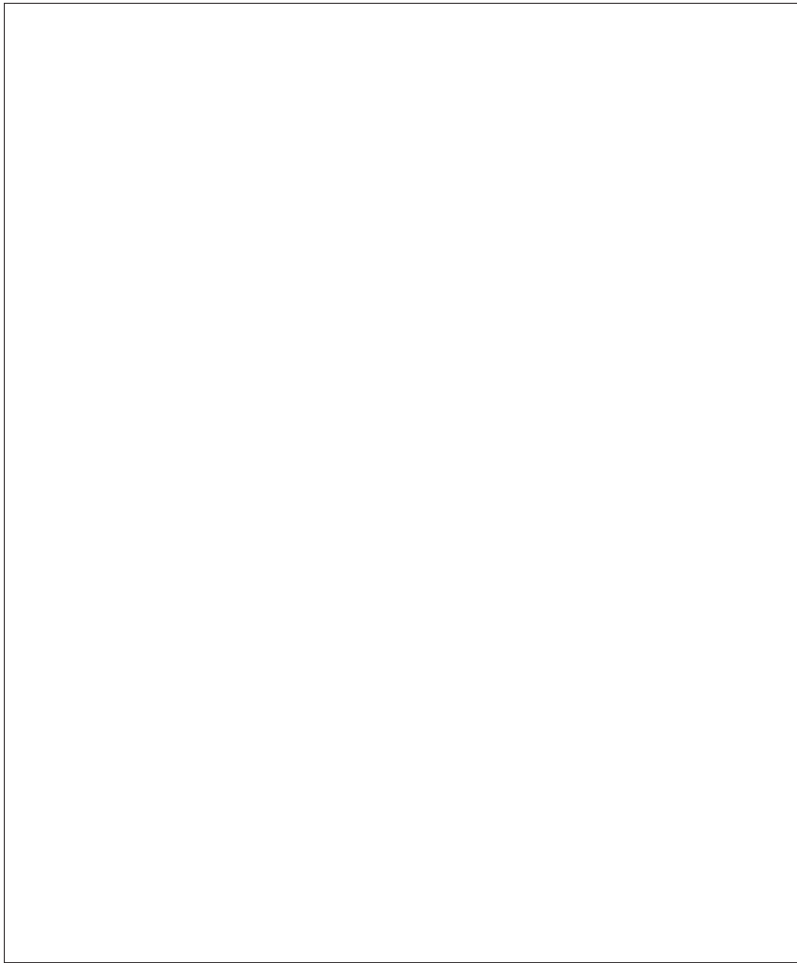
Critical care nurses often record an ECG in the event of a change in patient status. This change in status includes the development of arrhythmias. Evaluation of a rhythm strip in relationship to arrhythmias is discussed later in this chapter. Often, an ECG is obtained during episodes of chest pain before the administration of sublingual nitroglycerin. The ECG provides documentation of ST segment changes associated with the pain.

Some patients fear being shocked by the ECG recorder. Preparatory instruction for patients should include an explanation of the manner in which the electrical impulses of the heart are recorded.

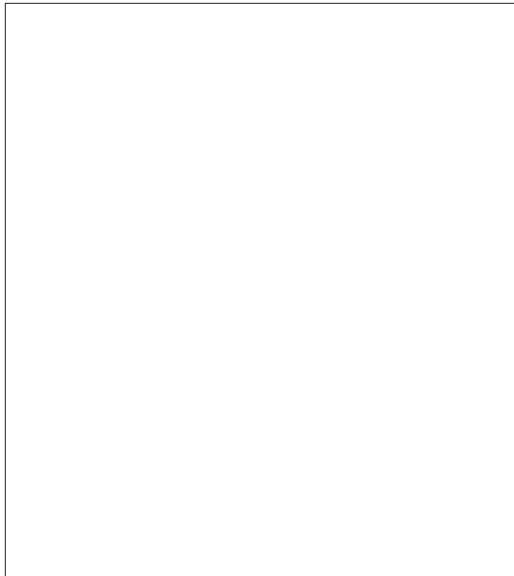
#### HOLTER MONITORING

##### *Purpose*

Ambulatory monitoring of coronary care or telemetry patients provides a noninvasive method of assessing for arrhythmias, response to arrhythmia treatment, pacemaker failure, and ECG signs of myocardial ischemia. Patients who present to the hospital with syncope, near syncope, or palpitations may not have recurrence of symptoms while at rest. Holter monitoring permits these patients to ambulate



**figure 17-14** Electrocardiogram electrode placement. The standard left precordial leads are V<sub>1</sub>, fourth intercostal space, right sternal border; V<sub>2</sub>, fourth intercostal space, left sternal border; V<sub>3</sub>, diagonally between V<sub>2</sub> and V<sub>4</sub>; V<sub>4</sub>, fifth intercostal space, left mid-clavicular line; V<sub>5</sub>, same horizontal line as V<sub>4</sub>, anterior axillary line; V<sub>6</sub>, same horizontal line as V<sub>4</sub> and V<sub>5</sub>, midaxillary line. The right precordial leads, placed across the right side of the chest, are the mirror opposite of the left leads.



**figure 17-15** Frontal plane leads = standard limb leads, I, II, III, plus augmented leads aVR, aVL, and aVF. This allows an examination of electrical conduction across a variety of planes (e.g., left arm to leg, right arm to left arm).

while their heart rhythm is recorded continuously to ascertain whether the etiology of the symptoms is caused by arrhythmia. Many patients with unstable angina have transient episodes of ST segment depression or elevation without associated angina pectoris; Holter monitoring enables the documentation and quantification of these episodes of “silent ischemia.”

#### *Procedure*

The Holter monitor is a battery-powered tape-recording device that may be worn on a belt around the patient’s waist or carried on a shoulder strap. Commonly, two leads are recorded continuously on tape through four or five electrodes placed on the patient’s anterior chest; the electrodes are arranged so that one lead reflects the inferior wall of the heart, and the other lead reflects the anterior wall. Continuous recording of ECG leads usually is performed for 24 to 48 hours. The Holter monitor contains a clock so that time also is recorded on the tape. After completion of the test, the tape is removed and played back for identification and quantification of ST segment changes or arrhythmias.

#### *Nursing Assessment and Management*

Patients who are scheduled to undergo Holter monitoring should be instructed to bathe before the test because the



electrodes cannot be removed during the 24- to 48-hour recording. Skin preparation and electrode placement are crucial to obtaining high-quality ECG recordings. It may be necessary to wrap material or fishnet over the electrodes and cables on the patient's torso to reduce movement artifact. Often, the skin under and around the electrodes becomes irritated, and the patient must be cautioned to avoid pulling at the electrodes because loss of electrical contact can mimic sinus pauses or heart block, making the diagnostic interpretation of the test difficult.

Most Holter monitors have an "event" button that can be pushed whenever symptoms occur; this button sends a signal to mark the tape. Patients are asked to maintain a diary of symptoms and activities and the time they occurred and should be instructed to record the time from the Holter monitor clock. Patients should be encouraged to maintain normal activities while wearing the monitor and told that it is desirable to record an entry in the diary at least every 2 hours. Hospitalized patients may need the assistance of nursing staff in maintaining their diaries.

### IMPLANTABLE LOOP MONITOR

The insertable loop recorder (ILR) is a newer, implantable device designed to capture and record the patient's ECG during a syncopal episode. The evaluation of syncope has long been problematic; isolated measurements such as Holter monitoring and random 12-lead ECG tracings seldom provide clinicians with an accurate confirmation of the causes leading to syncope.<sup>1</sup> The ILR is now used to provide longer and more inclusive surveillance for up to 1 year or longer. The device may be programmed so that a correlation can be established between the patient's intrinsic cardiac rhythm and syncopal events, and it continually monitors the patient's cardiac electrical activity, recording it in a memory loop. The patient or family member is instructed to activate the loop recorder, using a hand-held activator that works in concert with a computer programmer with a telemetry head that communicates with the device. When the activator is triggered, the ILR stores the ECG before, during, and after the syncopal event. When the ECG is retrieved by the clinician, a determination regarding the cause of the patient's syncope can be made and appropriate treatment can then be initiated, thereby reducing the risk of morbidity and mortality.<sup>1</sup>

### SIGNAL-AVERAGED ELECTROCARDIOGRAPHY

#### *Purpose*

Signal-averaged ECG is a noninvasive method for assessing patients who are at high risk for sudden cardiac death due to ventricular arrhythmias. In recent years, it has been used primarily in patients who are recovering from or have a history of MI to define the risk of ventricular tachycardia. In addition, patients who are admitted with unexplained syncope may benefit from signal-averaged ECG after other noncardiac causes have been excluded.

The major mechanism of ventricular tachycardia in patients with a history of MI relates to an area of slow conduction in the left ventricle. This area of slow conduction depolarizes late after most of the ventricle has depolarized, producing small-amplitude and late electrical poten-

tials not visible on the normal 12-lead ECG. The signal-averaged ECG allows these late potentials to be identified by repetitively mapping the patient's QRS complexes onto each other; filtering out noise, such as movement or electrical interference; averaging the repetitively mapped QRS complexes; and amplifying the averaged signal. There is some variation between laboratories in the definition of a positive signal-averaged ECG; however, in general, late potentials are considered to be present if the QRS complex duration is prolonged, the terminal low-amplitude portion of the QRS complex is prolonged, or the root mean square voltage of the terminal portion of the QRS complex is less than 20 mV.

#### *Procedure*

In addition to a ground electrode, six other electrodes are placed on the patient's chest during the 20 minutes required for performing the signal-averaged ECG. The six electrodes constitute three paired leads that are at right angles to each other; one set of leads is placed horizontally on the mid-right and left anterior chest, a second set is placed vertically at the top and bottom of the sternum, and the third set is placed anteroposteriorly just to the left of the sternum and on the posterior thorax. The patient must rest quietly in a supine position for the duration of the study. Extraneous noise, such as muscle movement, interferes with interpretation of the test, and patients who are restless or agitated or have difficulty lying supine are not good subjects for signal-averaged ECG.

#### *Nursing Assessment and Management*

The critical care nurse may be responsible for explaining the general format of the test and for emphasizing the need to remain as motionless as possible during the study to achieve accurate data.

### CHEST RADIOGRAPHY

#### *Purpose*

Chest radiography is a routine diagnostic test used to assess critically ill patients with cardiac disease. The test can be performed easily at the bedside in patients too ill to be transported to the radiology department. The image obtained on a radiograph that allows visualization of vascular and cardiac shapes is based on the premise that thoracic structures vary in density and permit different amounts of radiation to reach the film.

Chest radiography may be used for the evaluation of cardiac size, pulmonary congestion, pleural or pericardial effusions, and position of intracardiac lines, such as transvenous pacemaker electrodes or pulmonary artery (PA) catheters.

#### *Procedure*

Cardiac size is evaluated best in the radiology department, where the procedure can be standardized with the patient standing and the radiograph taken from a posterior view at a distance of 6 feet. Portable bedside chest radiographs usually are taken from an anterior view with the patient lying supine or sitting erect and are not standardized.

Patients undergoing radiography of the chest should be instructed not to move while the radiograph is being taken. Proper positioning of the radiographic plate behind the patient is important to ensure that thoracic structures

are aligned on the film. Care should be taken to remove all metal objects, including fasteners on clothing, from the field of view because metal blocks the x-ray beam. Patients usually are asked to take a deep breath and hold it when the radiograph is taken to displace the diaphragm downward; this may be uncomfortable for patients who have undergone recent thoracic surgery.

### Nursing Assessment and Management

The critical care nurse's role in obtaining diagnostic thoracic radiographic films often is limited to the ICU, where portable radiographs are made. With unstable patients, the nurse must decide when the film can be taken. It is important that IV lines not become tangled or loosened while one is trying to place the radiographic plate in the proper position.

Female patients of childbearing potential should have a lead drape placed over the abdomen to protect the ovaries from any radiation scatter. For the same reason, caregivers and family members should leave the patient's room when the radiograph is taken. When caregivers cannot leave the patient's bedside, a lead apron should be worn.

## ECHOCARDIOGRAPHY

### Purpose

The use of echocardiography in diagnosing and monitoring heart disease has increased dramatically since its introduction in the 1960s. For many patients, echocardiography has been an invaluable substitute for more invasive procedures in the management of heart disease. Echocardiography now refers to a group of tests that use ultrasound either alone or in combination with other technologies. Its growth as a clinical tool is likely to continue. Because of miniaturization of the equipment, research concerning intravascular ultrasound (IVUS) devices that would permit the identification of intraluminal defects is being conducted.

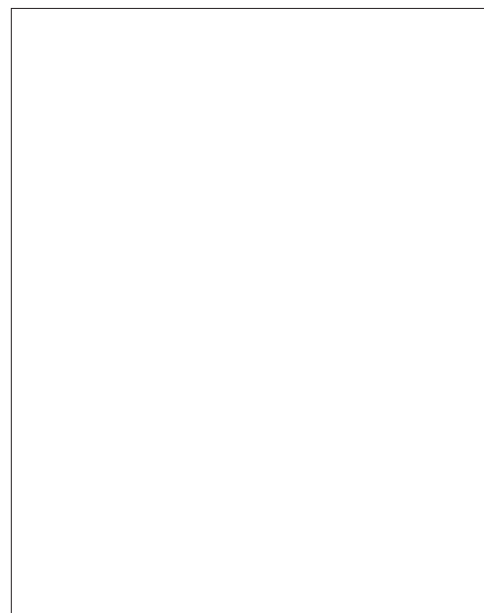
Echocardiography is used clinically in many cardiac conditions. The type of echocardiogram performed depends on the condition being evaluated. In critical care patients, echocardiography is used most often to assess ejection fraction, segmental wall motion, systolic and diastolic ventricular volumes, and mitral valve regurgitation due to papillary muscle dysfunction, and to detect the presence of mural thrombi, valve vegetations, or pericardial fluid. Echocardiography is a helpful diagnostic tool in the presence of sudden clinical deterioration in acute MI, in which significant complications may be observed or suspected. It also may be used in the evaluation of function of all four cardiac valves, including calculation of gradients and orifice size, intracardiac tumors, and aortic dissection. In some centers, echocardiography has made it possible for young patients unlikely to have coronary artery disease to undergo valve replacement without requiring a preoperative cardiac catheterization. M-mode and two-dimensional echocardiography can be performed easily at the bedside, but the reduced noise and light levels of the laboratory usually result in better recordings.

### Procedure

*M-mode echocardiography* is the first and simplest use of ultrasonography in patients with cardiac disease. This technique provides a rapid assessment of valvular motion

and chamber wall thickness. It requires a transducer that acts both as a sound transmitter and a sound receiver. The transducer is placed on the anterior chest in an intercostal space or subcostal position to avoid bony structures. A single ultrasound beam is sent from the transducer and directed toward the heart. As the sound waves reach various structures in the path of the beam, some pass through and around the structures, and some are reflected back to the transducer by the interface between two structures of differing densities. The more distant the interface, the longer it takes for the reflected sound waves to reach the transducer. A recording device is connected to the transducer so that as the reflected sound waves are received, they are converted to an electrical signal. If only one ultrasound wave beam is emitted from the transducer, the recording contains echoes from structures in the beam's path. For example, if transducer position #1 in Figure 17-16 is used, the recording would contain sound waves reflected from the chest wall, the free right ventricular wall, a space representing the right ventricular cavity, the intraventricular septum, a space representing the left ventricular cavity, and the posterior wall of the left ventricle.

In M-mode echocardiography, ultrasound waves are transmitted intermittently; the remainder of the time, the transducer is receiving the reflected sound waves. Typically, an M-mode recording is made with the reflected sound waves on the vertical axis and time on the horizontal axis. As the heart moves during the cardiac cycle, the recording displays this movement. Because M-mode echocardiography is based on a single beam, the so-called "ice-pick" view, only a small region of the heart can be visualized at any



**figure 17-16** Echocardiographic views of the heart. A cross-section of the heart shows the structures through which the ultrasonic beam passes as it is directed from the apex (1) toward the base (4) of the heart. CW, chest wall; T, transducer; S, sternum; ARV, anterior right ventricular wall; RV, right ventricular cavity; IVS, intraventricular septum; LV, left ventricle; PPM, posterior papillary muscle; PLV, posterior left ventricular wall; AMV, anterior mitral valve; PMV, posterior mitral valve; AO, aorta; LA, left atrium.

one time. The four positions of the transducer depicted in Figure 17-16 are the typical views used during an M-mode echocardiogram.

*Two-dimensional (2D) echocardiography* is performed in a similar manner except for two major differences: The ultrasound waves are transmitted in a pie-shaped beam, resulting in a “plane” of reflected echoes, and the recording device is a video camera so that the two dimensions of the plane and movement over time are recorded. In addition to parasternal and subcostal transducer positions, apical, and suprasternal positions also may be used in 2D echocardiography.

Recently, exercise or pharmacological stress testing has been used in conjunction with 2D echocardiography. A combination of physical exercise and 2D echocardiography has long been recognized as a valuable method to confirm or evaluate the presence of coronary artery disease and the extent of its involvement. An image taken at rest is compared with an image taken immediately after exercise. In patients with significant coronary artery disease, ventricular wall motion abnormalities develop after exercise in the segments supplied by diseased arteries. In patients who are unwilling or unable to exercise because of physical or psychological constraints, pharmacological agents have been used appropriately and effectively to stress the heart physiologically. Vasodilators, such as adenosine and dipyridamole, have been used in the past to induce myocardial ischemia in several imaging modalities (e.g., thallium scan testing) and echocardiography. Observation of areas of reduced blood flow can unmask coronary artery disease and quantify myocardium at risk.

In recent years, dobutamine hydrochloride has been proven effective in pharmacological stress testing in combination with echocardiography. Dobutamine is a synthetic catecholamine with beta<sub>1</sub>-receptor and alpha-receptor properties, which give the drug both inotropic and chronotropic effects that mimic physical exercise. With dobutamine stress echocardiography (DSE), global or regional wall motion abnormalities in compromised myocardial muscle may be observed before, during, and after titrated dobutamine infusion. If motion abnormalities are noted, the test is deemed positive and the patient can then be considered for surgical revascularization to improve cardiac performance. Distinct advantages of DSE include the fact that the test is noninvasive, can be safely performed a few weeks after an acute MI, and can illuminate wall motion abnormalities that cannot be assessed with electrographic monitoring.

*Doppler echocardiography* superimposes Doppler techniques on either M-mode or 2D images. The direction of blood flow can be assessed by measuring echoes reflected from red blood cells as they move away or toward the transducer. This type of study is particularly useful in patients with valvular stenosis or regurgitation; blood flow is quite turbulent through a stenotic valve, and in the opposite direction with regurgitation. When the direction of flow is color encoded, the study is known as a color Doppler echocardiogram. Audio signals usually are recorded during Doppler studies. Contrast material also may be used in conjunction with M-mode or 2D echocardiography. Although many agents have been used as contrast material, almost any liquid injected intravenously contains microbubbles. As the

microbubbles travel through the heart, they produce multiple echoes. This technique is especially useful in identifying right-to-left intracardiac shunts because of the early appearance of the microbubble echoes in the left atrium or ventricle.

A newer use of Doppler ultrasound technology makes use of an esophageal Doppler monitor. This device can provide valuable information on heart function, patient fluid status, and the impact of therapeutic interventions.<sup>2,3</sup> This noninvasive, harmless technology uses a thin silicone probe that is inserted into the esophagus of an intubated, sedated patient. The probe is positioned close to the descending aorta. Data collected can provide the clinician with hemodynamic information, such as heart rate, stroke volume, preload, afterload, cardiac output, systemic vascular resistance, and cardiac index, that has been demonstrated to be as accurate as information obtained from an invasive PA catheter.<sup>4,5</sup> Because this technique is safe and more readily available for a wider range of patients, it has been implicated in reducing postoperative complications and lengths of hospital stays for critically ill patients.<sup>6</sup>

*Transesophageal echocardiography (TEE)* is another method of ultrasonographic study that is made possible by placing a 2D transducer on the end of a flexible endoscope and positioning it at various locations in the esophagus (Fig. 17-17). Doppler and color Doppler also can be added. Because the transducer is closer to cardiac structures, the images are usually superior to those obtained with transthoracic techniques. TEE is useful in situations where it is technically impossible to image structures of interest from the usual transthoracic position—in particular, the aorta, atria, and valves. Newer techniques such as transgastric and transthoracic echocardiography are beginning to appear and may demonstrate value in the detection of structural anomalies.

### *Nursing Assessment and Management*

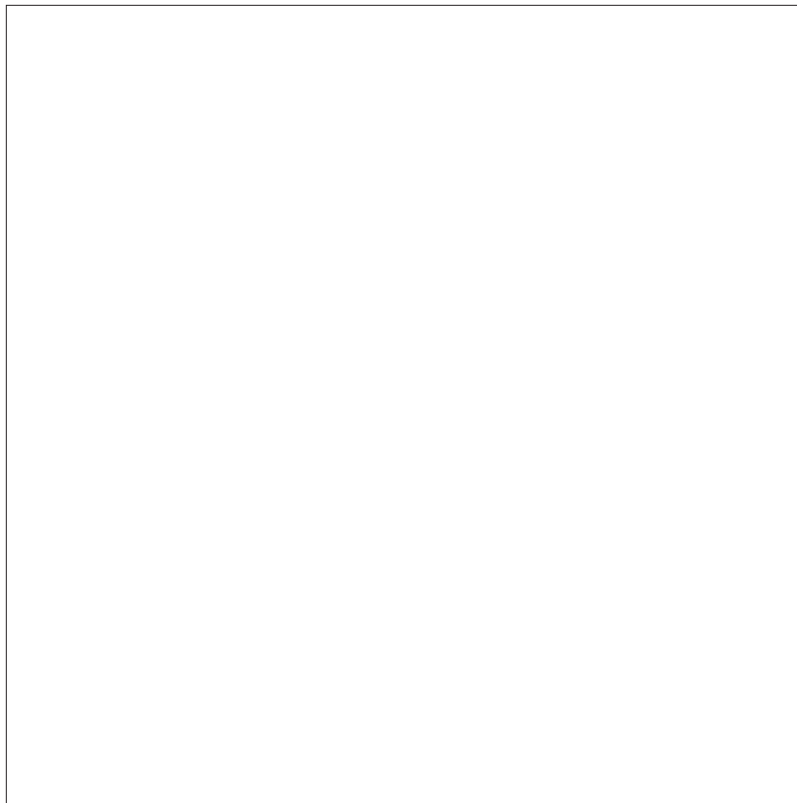
There are no specific prestudy restrictions for patients undergoing transthoracic echocardiography. During the study, the patient usually is placed in the supine position or turned slightly to the left side. Noise and light should be kept to a minimum. There is no discomfort associated with transthoracic echocardiography; however, the patient may experience chest wall discomfort due to the positioning of the transducer after the study. Suboptimal imaging may occur in patients who are obese or have obstructive lung disease.

Patients who are scheduled to undergo transesophageal echocardiography should take nothing by mouth (NPO) for 6 or more hours before the study. Mild to moderate sedation may be administered intravenously both before and during the test. Emergency equipment should be readily available in case of oversedation. A local anesthetic spray usually is applied to the posterior oropharynx to block the gag reflex before the endoscope is inserted orally. After the procedure, the patient should remain NPO until the gag reflex has returned. Table 17-4 summarizes nursing considerations for caring for the patient undergoing TEE.

## PHONOCARDIOGRAPHY

### *Purpose*

In phonocardiography, heart sounds are recorded by a microphone and converted to electrical activity that is



**figure 17-17** Views of the heart from a transesophageal echocardiogram. **(A)** Horizontal scan plane of aortic arch and distal portion of aorta. **(B)** Basal short-axis (transverse), long-axis (sagittal) views, and short-axis views of both atria. **(C)** Four-chamber and left atrioventricular long-axis views. Sagittal scan plane can image a cross-section of the left ventricle. **(D)** Transgastric short-axis view of left and right ventricles. **(E)** Transverse and sagittal scan sections of descending aorta.

recorded. This procedure may be used to obtain precise measurements of the timing of cardiac cycle events, to determine the characteristics and timing of murmurs and abnormal heart sounds, to measure systolic time intervals, and to teach cardiac auscultation. There are no contraindications or risks associated with this procedure.

#### *Procedure*

For a phonocardiogram, the patient is brought to a quiet room and asked to lie on a comfortable table or bed. Microphones are applied to the chest wall over areas where the heart sounds and murmurs are auscultated best. The microphones pick up the sound of the heart beat and convert it to an electrical impulse that is then amplified, filtered, and recorded. Some of the microphones are allowed to lie free on the chest; others are attached by straps or Ace bandages. A recording of sound waves is obtained, usually in conjunction with an ECG and a carotid pulse wave recording. These accessory recordings provide a reference point for the timing of cardiac events.

Special maneuvers or the use of pharmacological agents may accentuate certain heart sounds and murmurs. These include the inhalation of amyl nitrate, injection of IV isoproterenol or vasopressors, changes in position (sitting or squatting), variations in breathing (deep inspiration and expiration), and the performance of a Valsalva maneuver.

#### *Nursing Assessment and Management*

Phonocardiography usually takes 1 to 2 hours. Patients should be told beforehand that they may be asked to perform certain maneuvers or may be given certain agents to facilitate the diagnostic value of the test.

## EXERCISE ELECTROCARDIOGRAPHY

### *Purpose*

Exercise ECG is used primarily as an outpatient procedure to assess patients at risk for the presence of coronary artery disease. However, its use in coronary care and telemetry units is becoming more widespread. Patients who have presented to the hospital with chest pain but without associated ECG changes, have had the diagnosis of infarction excluded, and have remained symptom free may undergo exercise ECG to evaluate the etiology of their presenting symptoms and whether continued hospitalization is warranted. In addition, exercise ECG may be used to evaluate patients with arrhythmias whose symptoms are exacerbated by exercise. Low-level exercise ECG, a modification of the standard exercise test, is commonly performed before discharge in patients hospitalized with acute MI.

Patients with significant coronary artery disease may have normal ECGs at rest when myocardial oxygen supply is sufficient to meet oxygen demands. However, with increased oxygen demands during exercise, coronary blood flow cannot increase adequately because of coronary artery stenoses, and ECG changes may occur.

Exercise ECG should not be performed in patients who have left bundle branch block (LBBB) or pre-excitation at rest because the baseline QRS complex abnormalities preclude interpreting the ST segment response to exercise. The test also is less specific in women, especially those who are young or middle-aged, than in men. It is common practice to perform a low-level exercise test before hospital discharge after acute MI to identify patients at risk for ischemic events and to determine exercise prescription.



**table 17-4 ■ Nursing Considerations for the Patient Undergoing Transesophageal Echocardiography (TEE)**

Nursing Action	Rationale
<b>Preprocedure</b>	
1. Evaluate patient for contraindications.	Patients with history of dysphagia or esophageal disease are not candidates for TEE.
2. Instruct patient and family about procedure.	Some discomfort may occur; patient will receive moderate sedation but will be closely monitored.
3. Ensure that documented patient history is adequate and informed consent has been signed.	Medication allergies should be noted; procedure requires consent signature.
4. Ensure that patient has been NPO for 6 hours before procedure.	Aspiration precaution is vital.
5. Prepare patient for procedure.	Remove oral prosthetics, as indicated; have patient void.
6. Insert peripheral IV catheter.	IV access is required for routine medication administration; emergency vascular access line should be available.
7. Place patient on cardiac monitor with blood pressure and pulse oximetry.	Patient must be continually monitored during procedure.
8. Ensure that emergency resuscitation equipment is nearby, including medications, defibrillator, and suction apparatus.	Cardiac arrest precaution.
<b>During Procedure</b>	
1. Monitor cardiac rhythm, blood pressure, pulse oximetry, and airway patency per institutional policy.	Continuous observation is required after moderate sedation.
2. Assist physician with patient positioning and endoscope placement.	Allaying fears enhances patient cooperation.
3. Monitor for complications.	Vagal stimulation may occur with a resultant vasovagal reaction; transient tachycardia/bradycardia and blood pressure alterations may appear; patient may experience hypoxia or laryngospasm.
4. Reassure patient throughout procedure.	Allaying fears enhances patient cooperation.
5. Document patient response to procedures.	Per institutional protocol.
<b>Postprocedure</b>	
1. Assess vital signs at conclusion of procedure; document per institutional policy.	Comparison with baseline is necessary to monitor sedation recovery.
2. Assist patient to position of comfort or on one side.	Position provides comfort and patent airway support.
3. Keep patient NPO until gag reflex is assessed.	Prudent aspiration risk.
4. If gag reflex is present, encourage patient to cough; offer lozenges or ice to sooth sore throat; keep NPO per physician order.	Interventions provide patient opportunity to clear residual secretions and obtain comfort.
5. If outpatient, instruct patient not to drive for at least 12 h.	If patient was sedated during procedure, it is best if family member or another drives patient home.
6. Instruct patient to seek care or contact physician in event of dyspnea, hemoptysis, or severe pain.	If symptoms of complications occur, patient should be reevaluated.

The low-level test exercise target is approximately 70% to 80% of the predicted age-adjusted maximum. In patients with uncomplicated MI, the low-level exercise test has been performed safely as early as 3 days after infarction.

### Procedure

Although there are various exercise protocols, most are based on either walking on a treadmill or riding a stationary bicycle. The test usually begins at a low level of exercise and increases every 2 to 3 minutes until the patient reaches

a target level of oxygen consumption, manifests signs or symptoms of coronary artery disease, or reaches a predicted heart rate level calculation. Oxygen consumption, the amount of oxygen used in milliliters per minute per kilogram, usually is expressed in metabolic equivalents that take into account the age of the patient. If a treadmill is used to perform exercise, the speed or the uphill slope of the treadmill is increased at the beginning of each 2- to 3-minute stage; in cycling, the resistance of the pedals or braking mechanism is increased at the beginning of each stage.

Patients who have not previously undergone exercise testing should be allowed briefly to practice walking on the treadmill or riding the bicycle. Before starting the test, a resting ECG and blood pressure are obtained with the patient in sitting and standing positions. During the test, an ECG and blood pressure are obtained at the end of each stage of the protocol and immediately before termination. The test usually is terminated when signs or symptoms of myocardial ischemia develop or when the patient manifests other symptoms, such as fatigue or dyspnea, and cannot continue. In the absence of myocardial ischemia or serious arrhythmias, every effort should be made to reach the patient's predicted level of exercise to avoid a non-diagnostic test. During the recovery period, monitoring of the ECG and blood pressure continues until the patient has reached baseline values. It is mandatory that emergency resuscitation equipment and trained personnel be available in areas where exercise testing is performed.

Indications of myocardial ischemia during exercise testing are the development of (1) ST segment depression of 1 mm or more, (2) angina pectoris, or (3) failure to increase systolic blood pressure to 120 mm Hg or more or a sustained decrease of 10 mm Hg or more with progressive stages of exercise. The ECG leads in which ST segment depression occurs during exercise are not specific to the coronary artery involved. The greater the number of leads with ST segment depression, however, the more likely it is that the patient has multivessel coronary artery disease. The development of ST segment elevation or T-wave changes during exercise testing is not specific for myocardial ischemia, and its significance requires further assessment. Often, exercise ECG is performed in conjunction with echocardiography, radionuclide perfusion imaging, or radionuclide ventriculography to assess better the extent of coronary artery disease and its effect on ventricular function.

### *Nursing Assessment and Management*

Patients who are scheduled to undergo exercise ECG should abstain from eating or drinking caffeine-containing beverages several hours before testing to prevent abdominal cramps or nausea from developing at maximal exercise and to minimize blood diversion to the gastrointestinal tract, which decreases available coronary blood supply. They also should wear comfortable shoes for walking on a treadmill or riding a bicycle. The lead system is the same as used for the standard 12-lead ECG. However, the limb leads are moved to the torso so that arm or leg movement during exercise does not interfere with ECG recording. Careful attention is paid to skin preparation and electrode attachment to permit interpretable recordings during maximal exercise. It may be necessary to wrap material or fishnet over the electrodes and cables on the patient's torso to reduce movement artifact.

The critical care nurse may be responsible for explaining the general format of an exercise test to the patient and family. It is important that patients understand why the test is indicated and what will be expected of them. Patients should be reassured that someone will observe them closely throughout the test and encouraged to express any concerns before, during, and after the procedure. Patients also should understand that they may have to continue exercising after the development of angina but will not be expected to exercise more than is safe.

## RADIONUCLIDE IMAGING

The noninvasive assessment of cardiac structure and function using radiotracers has increased dramatically in the past few years. In particular, radionuclide perfusion studies are playing a larger role in the diagnosis and treatment of patients with coronary artery disease. The ability of perfusion studies to separate ischemic, viable myocardium from infarcted, nonviable myocardium is used by clinicians to select noninvasive versus invasive strategies, such as angioplasty or coronary bypass grafting, for treating the underlying coronary artery disease in patients with more severe disease.

Radionuclide perfusion studies provide information not only about the presence of coronary artery disease but about the location and quantity of ischemic and infarcted myocardium. In addition, they offer advantages over exercise ECG when ischemic changes cannot be assessed easily on the ECG, such as in patients with LBBB, with paced rhythm, or those receiving digitalis.

New on the horizon and not discussed in detail here is positron emission tomography (PET). This modality is highly sensitive and specific for diagnosing coronary artery disease. However, it is not superior to single-photon emission computed tomography (SPECT) in diagnostic accuracy. The equipment required for PET is expensive and is available in only a few centers. Because it offers the ability to image and quantify myocardial metabolism and blood flow, it is useful in distinguishing viable myocardium and evaluating the response of the myocardium to treatment with pharmacological agents.

### *Perfusion Imaging*

*Procedure.* Cardiac radionuclide perfusion imaging is based on the fact that a radioactive tracer is taken up in abnormal myocardial cells in either increased or decreased amounts compared with normal myocardium. After injection of the tracer, a gamma camera is used to record an image of radioactive counts from the entire myocardium. An abnormal area with decreased uptake, or "cold spot" imaging, is the type of study used to assess myocardial perfusion. An abnormal area with increased myocardial uptake, or "hot spot" imaging, is the type of study used to assess myocardial necrosis.

Perfusion studies are performed most commonly in conjunction with exercise testing so that radionuclide scans obtained at rest and with exercise can be compared. Typically, at rest, the radiotracer is spread uniformly throughout the myocardium, and the camera reads counts equally from throughout the myocardium. A similar scan is obtained during exercise in patients without significant coronary artery stenosis as blood flow increases uniformly to meet myocardial oxygen demands.

However, in patients with significant coronary artery disease, the image obtained during exercise is altered. The amount of coronary blood flow is limited in stenotic arteries, and the quantity of tracer in myocardial segments supplied by stenotic arteries is diminished or absent compared with segments supplied by nonstenotic arteries. The presence of an area of decreased tracer uptake during exercise compared with rest is known as a *reversible perfusion defect*. In patients with previous infarction, decreased uptake may be present on both the rest and exercise scans in the

infarcted segments; this pattern is known as a *fixed perfusion defect* and usually signifies nonviable myocardium. It is possible for patients to have fixed perfusion defects in some myocardial segments, reversible defects in others, and normal perfusion in the remaining segments.

Because of the many patients who are physically unable to exercise, pharmacological agents may be used to mimic the heart's response to exercise. Vasodilating agents, such as dipyridamole, adenosine, and dobutamine, administered intravenously mimic exercise conditions in the heart by dilating nonstenotic coronary arteries. Coronary blood flow is increased preferentially through normal, non-stenosed arteries; this results in relative hypoperfusion in myocardial segments supplied by stenosed coronary arteries. A radiotracer injected during the peak action of the pharmacological agent produces images similar to those seen with exercise. As of this writing, only dipyridamole is approved by the U.S. Food and Drug Administration (FDA) for use in perfusion imaging.

Two methods are used to record radioactive images—planar and tomographic. With the planar technique, images of the heart are obtained by the gamma camera from three views: anterior, left anterior oblique (45 degrees to the left of the anterior view), and left lateral (Fig. 17-18). Tomographic or SPECT images are obtained by rotating the head of the camera over a 180-degree arc from the left lateral to the anterior position while stopping to make 32 to 64 recordings of 20 to 40 seconds each. A computer uses the recorded images to reconstruct multiple slices of the heart along its short axis and both horizontal and vertical long axes.

Three radioactive tracers, thallium-201, technetium (Tc)-99m sestamibi, and Tc-99m teboroxime, are approved for perfusion imaging. Most experience in radionuclide perfusion studies has occurred with thallium because this agent has been available since 1974. Characteristics of the three agents differ and are responsible for the varying imaging protocols used.

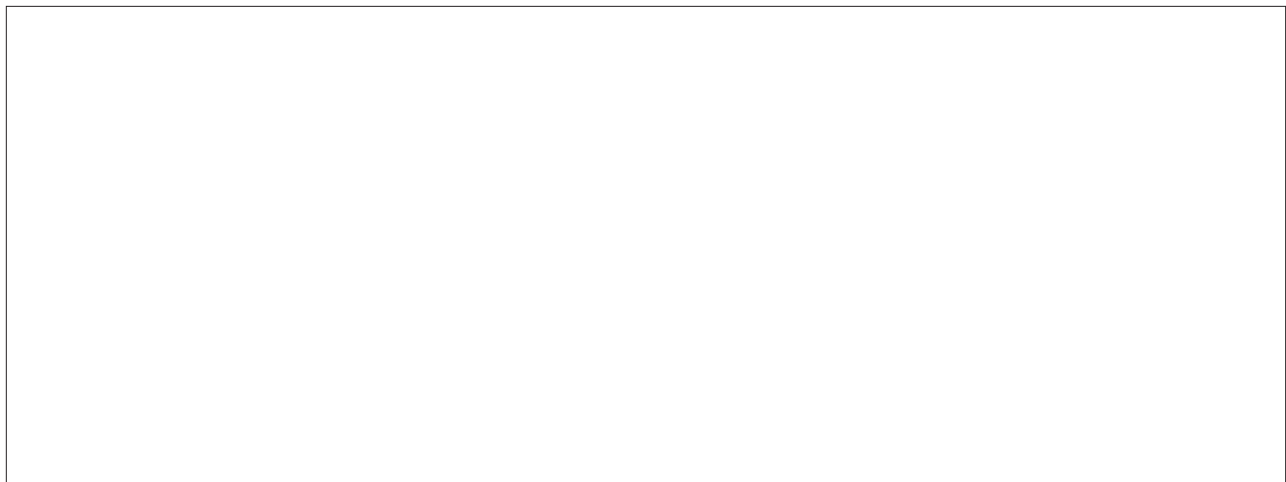
**Thallium Protocol.** The cardiac half-life of thallium is approximately 7.5 hours, meaning that 50% of the tracer still is present in myocardial cells 7.5 hours after it is admin-

istered. It also redistributes readily, so thallium in normally perfused areas moves to previously underperfused areas after the myocardial blood flow demands in that territory have decreased. The standard protocol for thallium perfusion studies begins first with the exercise portion; thallium is injected at the peak of exercise, and imaging starts within 5 minutes of injection. The rest portion is obtained 2 to 4 hours later. Because of redistribution, no additional thallium is required. However, in some patients with perfusion defects on both the rest and exercise scans, significant redistribution may not occur, and it is recommended that an additional dose of thallium be administered.

**Sestamibi Protocol.** Perfusion imaging with sestamibi typically begins with the rest scan first. Because significant uptake also occurs in the liver, imaging is delayed for approximately 60 minutes. This delay allows sestamibi to be cleared from the liver but not the heart. In addition, a glass of milk or small fatty meal is taken shortly after radiotracer injection to enhance hepatic clearance. A second dose of sestamibi is administered during peak exercise, and the exercise scan is obtained 60 minutes after injection, again allowing time for hepatic clearance. Because sestamibi redistributes very slowly, the image obtained 60 minutes after peak exercise reflects the perfusion conditions at the time of injection. Initially, perfusion studies with sestamibi were performed on 2 different days, but it now is customary to complete both portions of the study in 1 day. It has been shown that exercise sestamibi myocardial perfusion SPECT can provide incremental prognostic information in patients who have not suffered a previous MI or undergone cardiac catheterization and who are determined to be at low risk.

**Teboroxime Protocol.** Because of the very short cardiac half-life of teboroxime, two injections of the tracer are required. As with sestamibi, hepatic uptake also occurs. Redistribution is not an issue because of the short half-life. Imaging must begin within 2 to 5 minutes of injection and be completed within 15 minutes. The sequence of imaging, exercise versus rest, is not of concern, and typically the two scans are obtained 60 to 90 minutes apart.

Planar imaging usually is performed with the patient in the supine position, although some laboratories place



**figure 17-18** Ventricular segments of the heart projected on radionuclide planar views. ANT, anterior; LAO, left anterior oblique; LLAT, left lateral.

patients on their right side to obtain the left lateral image. When technetium-99m is used as the radiotracer, scans may be obtained with the patient in a sitting or standing position to avoid hepatic interference. With tomographic studies, it is extremely important that the patient not move during image acquisition because computer reconstruction of the images requires the same reference points. If significant movement occurs, the entire tomographic scan may have to be repeated.

**Nursing Assessment and Management.** All the directions and precautions that pertain to exercise ECG also apply to exercise radionuclide imaging. When pharmacological agents are used in place of exercise, minor side effects, such as flushing, headache, and nausea, may occur. Serious side effects due to the radiotracer are extremely rare. Medications to counteract serious side effects should be readily available. Some patients who receive sestamibi report a metallic taste several minutes after injection. Patients often are anxious about the radiation involved and the appearance of the equipment. It is important for the nurse to allay these anxieties.

### Infarct Imaging

**Procedure.** Infarct or “hot-spot” imaging may be useful in patients who present to the hospital several days after MI when serum cardiac enzymes have returned to normal. Accumulation of the radiotracer in the area of myocardial necrosis compared with the surrounding normal myocardium is responsible for the hot-spot image obtained.

Tc-99m Sn-pyrophosphate, the only radiotracer currently approved by the FDA for infarct imaging, is sensitive for 1 to 5 days after onset of symptoms. Because aneurysm formation in the area of a previous infarction may result in a false-positive study, a second pyrophosphate scan may be performed 7 to 10 days after symptom onset. In patients with recent infarction, little or no radiotracer uptake is seen on the repeat scan. The diagnostic sensitivity of pyrophosphate imaging in patients with a small or non-transmural infarction is poor.

Indium-111 antimyosin is a monoclonal antibody that binds to damaged myocytes and is under investigation as an imaging agent for myocardial necrosis. Planar or tomographic images are obtained 24 to 48 hours after injection of the indium-labeled antibody. Although the study usually is performed within 1 week of an MI, a positive scan may be obtained for up to 1 year after myocardial necrosis. Initial results suggest that antimyosin is more sensitive than pyrophosphate scans for the detection of infarction. In addition, antimyosin may be useful in other clinical conditions that result in myocardial necrosis, such as myocarditis and rejection after cardiac transplantation. The pattern of radiotracer uptake is more diffuse and global in these conditions, compared with the localized pattern of uptake in infarction.

**Nursing Assessment and Management.** No special preparation is required for patients undergoing infarct imaging other than an explanation of the procedure. Views usually are obtained with the patient in the supine position. If an antimyosin tomographic study is to be performed, the importance of not moving during image acquisition should

be reinforced. No serious side effects have been reported with either pyrophosphate or antimyosin administration.

Table 17-5 outlines some of the tests that are used to detect the presence of myocardial ischemia.

## ANGIOCARDIOGRAPHY

### Purpose

Radionuclide angiocardiology for the assessment of cardiac performance has been in clinical use since the 1970s. Such studies may include information about right and left ventricular ejection fractions, left ventricle regional wall motion abnormalities, ventricular volumes, and cardiac shunts. The measurement of left ventricular ejection fraction, the percentage of blood ejected with each contraction of the left ventricle, has been a key prognostic index for patients with MI or cardiac arrest.

Two approaches are used for the evaluation of cardiac performance. The technique used most commonly is known as equilibrium angiocardiology. It is performed easily at the bedside in patients too critically ill to be transported to the laboratory. The other technique, first-pass angiocardiology, likely will enjoy wider use in the future because it can use technetium radiotracers, such as technetium-99m or sestamibi, and can be performed at the same time as perfusion imaging.

### Procedure

With *equilibrium radionuclide studies*, an aliquot of the patient's blood is drawn, and the erythrocytes are tagged with Tc-99m radiotracer. The blood sample is then returned intravenously to the patient. Imaging can begin within a few minutes after administration and is performed serially over a period of 4 to 6 hours because the radiotracer-tagged erythrocytes remain within the vascular system. An ECG signal from the patient is used to separate radioactive counts acquired during systole from those during diastole; imaging continues over several hundred cardiac cycles, and images are averaged for both systole and diastole to obtain a representative cardiac cycle.

At the end of diastole, when the left ventricle is maximally filled with blood containing tagged erythrocytes, the amount of radioactivity is greatest. As the ventricle contracts during systole, blood is ejected into the aorta. The amount of blood and therefore radioactivity in the left ventricle is lowest at the end of systole. Because radioactive counts are proportional to the blood volume, the difference in counts obtained at the end of systole and the end of diastole permits the calculation of left ventricular ejection fraction. Left ventricular impairment caused by a previous infarction or cardiomyopathy usually results in a reduction in left ventricular ejection fraction from the normal values of 55% to 70%. Comparisons between ejection fractions at rest and with exercise also can be made. An inability to increase left ventricular ejection fraction by at least 5% with exercise is considered abnormal and may represent ischemic myocardium.

Left ventricular volumes and wall motion also can be assessed with equilibrium angiocardiology. By tracing the images obtained during the end of diastole and the end of systole, abnormalities in systolic or diastolic volumes can be ascertained. In addition, global versus regional impairment of ventricular function can be differentiated, including



**table 17-5** ■ Diagnostic Tests Used to Detect Myocardial Ischemia

Procedure	Abnormal Findings	Special Considerations
Standard 12-lead ECG	Transient ST segment and T-wave changes in patients with chest pain at rest or of prolonged duration	
Holter monitoring	Transient ST segment and T-wave changes occurring at rest or with activity	Only two ECG leads monitored
Stress echocardiogram	Segmental wall motion abnormality associated with echocardiogram obtained during exercise	May be used in patients with ventricular conduction defects Pharmacological agents may be used in patients who cannot exercise.
Exercise ECG	Transient ST segment and T-wave changes occurring with exercise	Cannot be used in patients who are unable to exercise or who have left bundle branch block or paced rhythm Does not provide good information on the location of the coronary artery disease
Radionuclide perfusion stress study	“Cold spot” image or perfusion defect associated with scan obtained during exercise	May be used in patients with ventricular conduction defects Pharmacological agents may be used in patients who cannot exercise.
Online ischemia analysis	Myocardial ischemia dynamic analysis (MIDA)* analyzing eight leads to detect ST segment levels indicating ischemia and QRS complex changes corresponding to infarct evolution	Noninvasive Hastens clinical decision making Graphic trends monitored online Reocclusion readily identified Helps differentiate chest pain related to ischemia from nonischemic symptoms

\*MIDA CoroNet, Hewlett-Packard, Andover, MA, Product Literature

the identification of aneurysm formation after infarction. Baseline data may provide information about the etiology of the ventricular impairment, and serial measurements often are used to assess response to treatment.

*First-pass radioangiography* also uses Tc-99m tracers; however, they are not tagged to any blood components. An image is obtained immediately after IV injection of the radiotracer as it enters the central circulation. The appearance time of the tracer in the various cardiac chambers and right and left ventricular systolic and diastolic counts provide diagnostic information. Because the time required for the tracer to traverse the central circulation is only a few cardiac cycles, the image acquisition time is very short.

Intracardiac shunts may be diagnosed by first-pass techniques. For example, in a patient with a ventricular septal defect and right-to-left shunt, the tracer appears in the left ventricle at the same time or before its appearance in the left atrium. In addition, this technique allows the amount of shunting to be quantified.

Right ventricular ejection fraction and volumes are measured best by first-pass angiography. Because the tracer is present in the right ventricle before it appears in the left ventricle, there is no contamination of counts from the overlapping left ventricle. The methods for measuring

right ventricular volumes and ejection fraction are similar to those used for the left ventricle.

### *Nursing Assessment and Management*

Three planar views similar to those used in perfusion imaging are obtained during equilibrium angiography. If exercise angiography is to be performed, the patient should be instructed to wear comfortable shoes for treadmill walking or bicycle riding. As with exercise testing, emergency equipment should be readily available. Although imaging usually is performed with the patient in the supine position, semierect or erect positioning may be used. It is important that the patient not move during image acquisition for either equilibrium or first-pass studies because of the effect on systolic and diastolic images.

Nurses caring for patients who have undergone radionuclide imaging should be aware of precautions; this information is available through the radiation safety department of their institution. The length of time that any precautions may be necessary is related to the half-life of the radiotracer used. In general, nurses who are pregnant should avoid caring for patients for 24 to 48 hours after the study, and all nurses should wear gloves when handling body fluids during the 24- to 48-hour period.

## COMPUTED TOMOGRAPHY

Computed tomography (CT) uses x-rays to provide a planar image of tissue structures. An x-ray source and detector rotate mechanically around the patient, and multiple cross-sectional images, or slices, are taken along a designated plane. Imaging is slow, which has proven to be a significant factor in the clear acquisition of structures in motion, such as the ventricles. The resultant blurring of images limits CT's effectiveness in viewing certain cardiac structures. However, CT is effective in illuminating nonmoving structures, such as the aorta, pericardium, and great vessels. CT can be used with or without a contrast agent.

### Ultrafast Computed Tomography

Ultrafast CT is a noninvasive technique that is rapidly showing promise in the assessment of myocardial blood flow. It is similar to conventional CT but differs in the speed at which images can be relayed. Ultrafast CT has a distinct advantage: its ability to provide high-quality image acquisition that is not contaminated by the artifactual movement that occurs with cardiac contractions. Images are obtained when an electronic beam passes across four tungsten targets, creating a fan-shaped x-ray view that is transmitted, digitalized, and reconstructed as with traditional CT scanning. Segmental slices of the heart can then be obtained, which are used to evaluate myocardial perfusion. This is especially useful when clear spatial resolution is required. This technique can be used with or without contrast media.

Ultrafast CT has demonstrated an increased sensitivity in the detection of coronary calcium, which has been implicated in the development of coronary atherosclerosis. Studies have shown that the greater the amount of intimal calcium present, the greater the likelihood of obstructive coronary disease. When ultrafast CT is used to identify and quantify coronary calcium, no contrast media are required, radiation exposure is minimal, and the physician need not be present. When ultrafast CT is used with contrast media, it has shown a high degree of accuracy in the assessment of myocardial blood flow when flow is normal or reduced. However, it is less accurate when blood flow is increased.

## MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) is a noninvasive imaging technique that uses a magnetic field and radiofrequency pulses to produce cross-sectional, three-dimensional images of tissues without the use of x-rays. MRI uses powerful magnetic fields that cause protons to become aligned or parallel to the magnetic field. MRI scans are used to view structural cardiovascular abnormalities when other diagnostic techniques (e.g., echocardiogram) are inconclusive or ambiguous. Because they do not visualize bone, they are superior to other techniques, such as a routine radiographic studies. Wall motion abnormalities, wall thickening, congenital defects, cardiac ejection fraction, coronary arteries, and valvular disease can be visualized on MRI. In addition, MRI scans help assess thrombus formation, major vessel malformations, and diseases of the aortic arch. Regions of MI as well as cardiac masses are best illuminated using the MRI technique.<sup>7,8</sup>

MRI does have limitations. The procedure is expensive, and the technology is not readily available in many treatment centers. In addition, patients must often be trans-

ported to a special MRI laboratory, thereby making the examination difficult for critically ill patients who require transport. The patient is placed in a small tube for 60 to 90 minutes; thus, the nurse should explain the procedure to the patient to allay any fears concerning close confinement. The patient should be NPO for at least 4 hours before the procedure, and premedication may be required. MRI is not a hazard to living cells, but there are significant absolute and relative contraindications to its use (Box 17-7).

## Invasive Techniques

### CARDIAC CATHETERIZATION

*Cardiac catheterization* is a generic term that refers to a variety of procedures performed in the catheterization laboratory. Such procedures include selective coronary artery, saphenous vein bypass graft, or internal mammary artery angiography, ventriculography, and right or left heart catheterization. All of the procedures are performed using invasive techniques and require a sterile environment.

#### Procedure

*Coronary angiography* is used to evaluate the presence and location of coronary artery disease. A catheter is introduced into the arterial system retrograde to the ascending aorta under fluoroscopy. The right or left main coronary artery is then selectively cannulated, and a radiopaque dye is injected directly into the artery through the catheter. As dye flows through the artery, the lumen of the artery can be visualized and the image recorded on film. Disease in the coronary artery or one of its branches delays or obstructs the flow of dye and may be visualized on the film as a site of lumen narrowing and slow filling of the artery with dye. In patients who have undergone previous coronary bypass surgery, selective injections of saphenous vein bypass grafts or internal mammary arteries can be performed in a similar manner.

#### box 17-7

#### Contraindications to Magnetic Resonance Imaging (MRI)

ABSOLUTE	RELATIVE (INDIVIDUAL ASSESSMENT REQUIRED)
Cardiac pacemaker	Prosthetic joints
Aneurysm clips	Certain foreign objects in body (e.g., dental braces)
Epicardial pacing wires	Nonmetallic prosthetic heart valves
Metal prosthetic heart valves	Implanted infusion pumps
Implanted cardioverter-defibrillator (ICD)	Cochlear implants
Implanted infusion pumps	Coronary stents (if recently deployed)
Cochlear implants	
Metal intrauterine devices	
Metal debris (e.g., bullets, shrapnel)	

*Radionuclide ventriculography* is an excellent tool to assess regional decreases in contractility in areas of stenosed vessels and is useful in the evaluation of left ventricular ejection fraction at peak exercise. Ventriculography, which commonly is performed in conjunction with selective coronary angiography, is accomplished by injecting dye directly into the left ventricle. A catheter is directed retrograde into the left ventricle through the arterial system under fluoroscopy. Dye is injected rapidly, and an image of the left ventricle cavity is recorded on film as the ventricle contracts. Left ventricular ejection fraction, the percentage of blood present in the left ventricle during diastole that is ejected during systole, can be calculated from the film images. Outlines of the ventricle during diastole and systole are traced, and the areas inside each outline are proportional to the amount of blood present. In addition, regional ventricular wall motion abnormalities caused by MI or severe ischemia can be visualized. The competence of the mitral valve also may be evaluated during ventriculography. In patients with mitral regurgitation, dye is observed being ejected not only into the aorta during systole, but into the left atrium through an incompetent mitral valve. In patients with suspected aortic regurgitation, dye may be injected into the aorta; if regurgitation is present, the dye flows retrograde into the left ventricle during diastole.

*Left heart catheterization* is performed to measure intracardiac or intravascular pressures in the structures of the left side of the heart. The chambers are accessed with a catheter introduced retrograde through the arterial system under fluoroscopy. If either the mitral or aortic valve is stenosed, the pressures required to eject blood forward are higher than normal because of the small valve orifice. For example, with normal mitral valve function, the left atrial pressure and left ventricular pressure are nearly equal during ventricular diastole, because blood flows easily from the left atrium through the mitral valve into the left ventricle. In contrast, mitral stenosis results in a left atrial pressure that is significantly higher than the pressure in the left ventricle during ventricular diastole because the left atrium has to generate more pressure to force blood forward through the stenosed valve. This difference in pressure is known as a *gradient* and is related to the degree of stenosis. Similar pressure comparisons are made in the left ventricle and aorta during systole to evaluate aortic stenosis. If a cardiac output measurement is available, the area of either the mitral or aortic valve opening may be calculated.

Mitral or aortic valve regurgitation also may be assessed by pressure measurements and with ventriculography, as previously described. The abnormal retrograde flow of blood into the left atrium during ventricular systole that occurs with mitral regurgitation produces higher-than-normal left atrial pressures. In patients with severe mitral regurgitation, the pressure in the left atrium may nearly equal the peak systolic pressure in the left ventricle. Similar pressure measurements are made in the left ventricle and aorta to evaluate aortic regurgitation.

*Right heart catheterization* is performed to measure intracardiac and intravascular pressures in structures of the right side of the heart. A catheter is inserted antegrade through the venous system under fluoroscopy; the procedure is similar to the insertion of a PA catheter. Pressures are recorded from the vena cava, right atrium, right ventri-

cle, PA, and pulmonary capillary wedge position. In addition, blood samples may be drawn from each chamber as the catheter is advanced, and the amount of oxygen present in each blood sample is measured. Because the right side of the heart normally contains venous blood, a significant increase in the amount of oxygen present in a blood sample may indicate a left-to-right intracardiac shunt.

Cardiac output, the amount of blood pumped by the heart in a minute, may be measured during a right heart catheterization using the thermodilution technique. Because cardiac output can be expected to vary with body size, the term *cardiac index*, which takes height and weight into consideration, is used more often.

Arterial and venous accesses usually are achieved with percutaneous techniques from femoral sites. Typically, a needle is inserted into the artery or vein. A guide wire is then inserted through the needle and advanced to the appropriate position in or near the heart. After removing the needle, a catheter may be placed over the guide wire and advanced to the desired position. Changing catheters over guide wires allows specific preformed catheters to be used during the procedure. In some patients, percutaneous access cannot be accomplished from a femoral site, and a cutdown at a brachial site may be required. A bolus of IV or intra-arterial heparin is administered to patients requiring arterial access to prevent clot formation on the guide wire or catheter.

### *Nursing Assessment and Management*

Patients undergoing elective cardiac catheterization are NPO for at least 6 hours before the procedure. Because the dye may be nephrotoxic, hydration with IV fluids may be started before the procedure and continued afterward. Patients with low cardiac output or renal impairment are especially susceptible to dye nephrotoxicity, and their renal function should be monitored closely after the procedure. Patients may be prescribed a mild sedative before the procedure. When percutaneous access is used, pressure usually is applied over the site until bleeding has ceased. A pressure dressing, and in some laboratories, a sandbag, is left in place for several hours after the procedure.

Patients typically are placed on bed rest for 6 hours after the procedure and instructed not to flex the affected extremity. After the procedure, the access site should be checked frequently for signs of bleeding, swelling, or hematoma formation. If a femoral arterial access site was used, peripheral pulses in the affected extremity should be monitored. In addition, bleeding may occur in the retroperitoneal space in patients who have undergone femoral arterial access. Close monitoring of blood pressure and heart rate and an awareness that retroperitoneal bleeding frequently presents as low back pain are useful in preventing a significant bleed. Nursing considerations for patients undergoing cardiac catheterization can be found in Box 17-8.

Before catheterization, patients should be informed about the procedures that will be performed and questioned about any possible dye allergies. They should be instructed that they will be placed on a table with rounded sides and that their body will be strapped down so that they will not move as the table rotates from side to side. If they are to undergo ventriculography, they should be instructed that they may experience a temporary hot flash or flushing



### box 17-8 nursing interventions for The Patient Undergoing Cardiac Catheterization

#### Preprocedure

- Explain procedure to patient and family.
- Verify that the patient has taken nothing by mouth for at least 6 hours before the procedure *except* prescribed medications as advised by the physician.
- Ensure ordered preoperative laboratory studies have been completed and results are available.
- Verify patient allergy information; alert physician if patient is allergic to radiographic dye, medications, or specific foods.
- Ensure that informed consent has been obtained.
- Establish intravenous access per institutional protocol or physician order.
- Place patient on cardiac monitoring system with blood pressure and pulse oximetry monitoring.
- Provide supplemental oxygen as ordered/indicated.
- Premedicate patient per physician order.
- Obtain vital signs before transfer to catheterization laboratory.

#### During Procedure

- Continually assess patient vital signs, oxygenation, level of consciousness, and cardiac rhythm per institutional protocol.
- Alert attending physician to significant changes in vital signs, oxygenation, and presence of malignant cardiac arrhythmias (e.g., premature ventricular contractions, ventricular tachycardia, ventricular fibrillation).
- Be prepared to initiate cardiac resuscitation with emergency equipment and medications.

#### Postprocedure

- Ensure patient vital signs are stable before transfer.
- Check catheterization site dressing for bleeding and integrity.
- Check distal pulses below catheterization site; if femoral site was used, check distal pulse, extremity color, capillary refill, and neurosensory status.
- Keep extremity straight and instruct patient not to bend leg or arm.
- Maintain intravenous infusion per physician order or institutional protocol.
- Maintain supplemental oxygenation support as ordered or indicated.
- Encourage oral fluids as ordered.
- Check patient's coagulation status per institutional protocol before sheath removal.
- When catheter is removed:
  - Apply direct pressure over invasive site for 20 to 30 minutes to prevent bleeding or apply commercial hemostatic compression device per institutional protocol.
  - Check distal extremity for pulse, color, capillary refill, and sensorium.
  - Remind patient to lie flat for 4 to 6 hours per institutional protocol.
  - Check site dressing every 4 to 6 hours for bleeding and integrity.

when dye is injected into the left ventricle. Postcatheterization procedures also should be explained to the patient, including bed rest and monitoring of the access site and vital signs. If the patient is to be discharged after the procedure and a cutdown was used for access, the patient should be instructed to make a follow-up appointment with the physician for suture removal. A summary of patient teaching for patients undergoing cardiac catheterization can be found in Box 17-9.

#### INTRAVASCULAR ULTRASONOGRAPHY

IVUS is a newer approach to the assessment of coronary anatomy. Using catheters of various frequencies, miniaturized ultrasound crystals are interfaced to ultrasound imaging consoles. Generally, the higher the frequency, the greater the image resolution. IVUS differs from angiography in that IVUS provides a circumferential assessment of the vessel with a 360-degree view, whereas angiography provides a rapid means of assessing the diameter of the vessel lumen in silhouette. IVUS is capable of providing cross-sectional images of coronary arteries that can illuminate arterial wall layers as well as injured media and adventitia. Vessel diameter and the cross-sectional dimensions can be determined. This evaluation permits identification of plaque composition on the vessel wall and its morphology.

In addition to better assessment of coronary and peripheral arterial atherosclerotic lesions, IVUS has proved useful in the assessment of arterial dissections and clots as well as aortic and pulmonary arterial disorders, and it can provide guidance during various catheter-based therapeutic procedures. Studies have shown that IVUS has been useful in the identification of atheromatous disease in vessels that appear normal with angiography and in the recognition of whether such lesions are soft, fibrous, or calcified. Another common use of IVUS has been for the evaluation of coronary artery stents. Data suggest that IVUS is superior to routine angiography in the determination of expansion and underexpansion of the intracoronary stent postimplantation. IVUS catheters are also used for intracardiac echocardiographic studies. Using 10-MHz ultrasound transducers, atrial and ventricular chambers can be visualized to a greater extent, and views of the structures of the left side of the heart can be obtained through the right atrium and right ventricle.

IVUS techniques have proven to be superior for intravascular diagnostic assessment over angiographic procedures alone. Their ability to characterize plaque composition, arterial wall characteristics, areas at risk, and intracoronary stent expansion has made IVUS a powerful tool in the armamentarium of cardiac diagnostic testing.





### box 17-9 Patient Teaching for the Patient Undergoing Cardiac Catheterization

#### Preprocedure

- Instruct the patient not to take anything by mouth for at least 6 hours before the procedure *except* prescription medications as advised by the physician to reduce the chance of nausea and vomiting during the procedure.
- Tell the patient that an intravenous line will be placed to allow fluid and medication administration before, during, and after the procedure.
- Tell the patient that preoperative medication will be given before transport.
- Inform the patient that only a patient gown will be worn during the procedure.
- Advise the patient that the catheterization laboratory is usually cool, and the procedure table is firm and may be uncomfortable after a prolonged time.
- Tell the patient that he may be asked to turn his head, hold his breath, or cough during the procedure.
- Advise the patient that he may experience some discomfort during the procedure but that local anesthesia will be administered to minimize pain.
- Inform the patient that he will be placed on a cardiac monitor for the duration of the procedure and for a few hours after the procedure.
- Tell the patient that he will have to lie flat for several hours after the procedure to minimize the chance of bleeding from the catheter site.
- Inform the patient that he will be encouraged to take oral fluids as tolerated after the procedure to assist in elimination of the radiographic dye.
- Encourage the patient and family to ask questions.

#### During Procedure

- Instruct the patient to inform the physician and team if he is experiencing chest pain.
- Remind the patient to lie still.
- Reassure the patient and allay anxiety.
- Encourage and answer the patient's questions.

#### Postprocedure

- Remind the patient to lie still and keep the extremity straight.
- Instruct the patient to verbalize any chest pain or shortness of breath if present.
- Tell the patient when the catheter sheath is due for removal.
- Encourage the patient to take oral fluids as ordered.
- Advise the patient that the physician will review the catheterization findings with him.

duction system of the heart, identification of abnormal or accessory conduction pathways, and stimulation of atrial or ventricular tissues to induce arrhythmias. All of the procedures are performed using invasive techniques and require a sterile environment.

Patients presenting with symptoms suggestive of supraventricular tachycardia, ventricular tachycardia (VT), or syncope frequently are studied with electrophysiological testing. The intracardiac electrodes are used to stimulate atrial or ventricular tissue at various pacing rates and numbers of extra stimuli. The induction and subsequent recording of a supraventricular tachycardia provides information about the mechanism of the arrhythmia. If an accessory pathway is identified as the mechanism, radiofrequency or surgical ablation of the pathway may be successful in eliminating future episodes of the tachycardia.

The successful induction of VT with electrophysiological testing is of both diagnostic and prognostic value for the risk of sudden cardiac death. Treatment with pharmacological agents can be evaluated with subsequent studies. Antiarrhythmics that prevent induction or slow the rate of a VT in a patient who was inducible in the control state may be used in the long-term management of the arrhythmia. Ventricular arrhythmias usually are not treated with ablation methods because the areas of ventricular tissue responsible for the tachycardia are not easily identified and are widespread.

#### Procedure

To measure electrical activity from the specialized conduction system of the heart, it is necessary to place electrodes at various intracardiac sites. Special catheters with multiple electrodes are inserted through arterial or venous access and advanced to locations in the heart; a separate access site is required for each electrode. In most studies, venous access is adequate for proper positioning of the electrodes; however, arterial access may be required for blood pressure monitoring. The high right atrium, bundle of His, and right ventricular apex sites typically are used for recording and stimulation. In addition, several body surface leads are recorded simultaneously.<sup>9</sup>

Venous and arterial accesses usually are achieved with percutaneous techniques from femoral sites in a manner similar to that used during cardiac catheterization. A sheath may be left in place during the procedure, however, so that electrode catheters may be repositioned as necessary. In some patients, access cannot be accomplished from a femoral site, and percutaneous access through the jugular vein or a cutdown at a brachial site may be required. A bolus of IV or intra-arterial heparin is administered to patients requiring arterial access to prevent clot formation on the electrode catheter.

Conduction times from the atria to the bundle of His and bundle of His to ventricles are measured. Sites of block—supra-His or infra-His—can be identified and provide information that is used to direct treatment. In addition, the atrium can be paced over a range of rates to identify the rate at which heart block develops. Sinus node function is evaluated by pacing the atrium at various rates, suddenly stopping pacing, and measuring the amount of time it takes for the sinus node to initiate an

## ELECTROPHYSIOLOGY STUDIES

### Purpose

Electrophysiology studies are used both for diagnosing and evaluating interventions in the treatment of arrhythmias. The testing protocol may include the measurement of conduction and recovery times in the specialized con-

impulse. The development of heart block at slow heart rates or prolonged sinus node recovery times may indicate a causal factor in patients presenting with syncope or presyncope.

### *Nursing Assessment and Management*

Patients undergoing electrophysiological testing are NPO for at least 6 hours before the procedure, although a sedative administered orally may be prescribed. When percutaneous access is used, pressure is applied over the site until bleeding has ceased. A pressure dressing usually is left in place for several hours after the procedure.

Patients typically are placed on bed rest for 6 hours after the procedure and instructed not to flex the affected extremity. The access site should be checked frequently for signs of bleeding, swelling, or hematoma formation. If a femoral arterial access site was used, peripheral pulses in the affected extremity should be monitored.

Before electrophysiological testing, patients should be instructed that they will be placed on a table with straps over their torso. They should be informed that they may experience palpitations or syncope if rapid tachyarrhythmias are induced. Poststudy procedures also should be explained to the patient, including bed rest and monitoring of the access site and vital signs. If the patient is to be discharged after the procedure and a cutdown was used for access, the patient should be instructed to make a follow-up appointment with the physician for suture removal.

Patients with VT who are being initiated on antiarrhythmic therapy are at risk for sudden cardiac death because some medications may have adverse proarrhythmic effects. These patients often are kept in a monitored setting for most of their hospital stay until appropriate therapies have been identified. Because most of these patients are otherwise healthy and physically active, it becomes a challenge for the nursing staff to care for these patients as well as the more severely ill patient population.

## ELECTROCARDIOGRAPHIC MONITORING

Cardiac monitoring is used in a variety of settings. Traditionally used in ICUs and operating rooms, cardiac monitors are now found in other inpatient units where it is necessary to monitor continuously a patient's heart rate and rhythm or the effects of a therapy. In addition, cardiac monitors are used outside the hospital in settings such as paramedic ambulances, surgical centers, outpatient rehabilitation programs, and transtelephonic monitoring clinics.

Although the type of monitor may differ in each of these settings, all monitoring systems have three basic components: a display system, a monitoring cable, and electrodes. Electrodes are placed on the patient's chest to receive the electrical current from the cardiac muscle tissue. The electrical signal is then carried by the monitoring cable to a screen, where it is magnified and displayed. The display can be obtained both at the patient's bedside and at a central station, along with displays from other patients' monitors.

## Equipment Features

Two types of cardiac monitoring equipment are in use: continuous hard-wire monitoring systems and telemetry monitoring systems.

### HARD-WIRE MONITORING SYSTEMS

Hard-wire monitors, which are commonly used in ICU settings, require the patient to be linked directly to the cardiac monitor through the ECG cable. Information is displayed and recorded at the bedside along with simultaneous display and recording at a central station. Because this type of cardiac monitoring limits patient mobility, patients using this system usually are confined to bed rest or are allowed to be up at the bedside only. Hard-wire monitors operate on electricity but are well isolated so that water, blood, and other fluids do not pose an electrical hazard as long as the machine is maintained properly.

### TELEMETRY MONITORING SYSTEMS

In telemetry monitoring, no direct wire connection is needed between the patient and the ECG display device. Electrodes are connected by a short monitoring cable to a small battery-operated transmitter that the patient carries in a disposable pouch tied to his or her body. The ECG is then sent by radiofrequency signals to a receiver that picks up and displays the signal on an oscilloscope, either at the bedside or at a distant central recording station. Antennas are built into the receiver and may be mounted in the vicinity of the receiver to widen the range of signal pickup. Batteries are the power source for the transmitter, thus making it possible to avoid electrical hazards by isolating the monitoring system from potential current leakage and accidental shock. Telemetry systems are used primarily for arrhythmia monitoring in areas where the patient is fairly mobile, such as an arrhythmia surveillance or progressive care unit. Because the patient is mobile, stable ECG tracings often are more difficult to obtain. Some hard-wire systems have built-in telemetry capability so that patients may be switched easily from one system to another as monitoring needs change.

### DISPLAY SYSTEMS

Modern electronic technology continues to make sophisticated advances in monitoring equipment, and current display systems incorporate features such as the following:

- Improved freeze/hold modes, which allow the ECG pattern to be held for more detailed examination
- Storage capability that permits retrieval of arrhythmias
- Automatic chart documentation, in which the ECG recorder is activated by alarms or at preset intervals
- Alarm systems for a variety of parameters
- Multilead or 12-lead ECG display, which facilitates complex arrhythmia interpretation
- ST segment analysis for monitoring ischemic events
- Multiparameter displays, which offer display of hemodynamic pressures, temperature, intracranial pressure, and respirations
- Computer systems that store, analyze, and trend monitored data, allowing the information to be retrieved