CHAPTER

12

Cardiovascular Alterations

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INTRODUCTION

Care of the patient with alterations in cardiac status present unique challenges due to potential serious hemodynamic changes that can affect the prognosis of the critically ill patient. The critical care nurse needs both theoretical knowledge and practice-related understanding of the common cardiac diseases to have the sound clinical judgment necessary for making rapid and accurate decisions. The purpose of this chapter is to identify and explore common cardiac alterations that are likely to be encountered by the critical care nurse caring for adult patients with compromised cardiac status, and to describe the nursing care to optimize patient outcomes.

NORMAL STRUCTURE AND FUNCTION OF THE HEART

The heart muscle is approximately the size of a person's closed fist and lies within the mediastinal space of the thoracic cavity between the lungs, directly under the lower half of the sternum, and above the diaphragm (Figure 12-1). It is covered by the pericardium, which has an inner visceral layer and an outer parietal layer. Certain diseases can cause this covering to become inflamed and can subsequently diminish the effectiveness of the heart as a pump. Several cubic milliliters of lubricating fluid are present between these layers. Some pathological conditions can increase the amount and the consistency of this fluid, affecting the pumping ability of the heart. The heart muscle itself is composed of three layers. The outer layer, or epicardium, covers the surface of the heart and extends to the great vessels; the middle, muscular layer, or myocardium, is responsible for the heart's pumping action; and the inner endothelial layer, or endocardium, covers the heart valves and the small muscles associated with the opening and closing of those valves. These layers are damaged or destroyed when a patient has a myocardial infarction (MI).

Functionally, the heart is divided into right-sided and left-sided pumps that are separated by a septum. The right side is considered to be a low-pressure system, whereas the left side is a high-pressure system. Each side has an atrium that receives the blood, and a ventricle that pumps it out. The right atrium receives deoxygenated blood from the body through the superior vena cava and inferior vena cava. Blood travels from the atrium to the ventricles by means of a pressure gradient between the chambers. The right ventricle pumps the deoxygenated blood to the lungs through the pulmonary artery for oxygen and carbon dioxide exchange. The left atrium receives the newly oxygenated blood by way of the pulmonary veins from the lungs, and the left ventricle pumps the oxygenated blood through the aorta to the systemic circulation (Figure 12-2).

The four cardiac valves maintain the unidirectional blood flow through the chambers of the heart. The valves also assist in producing the pressure gradient needed between the chambers for the blood to flow through the heart. There are two types of valves: the atrioventricular (AV) valves, which separate the atria from the ventricles; and the semilunar (SL) valves, which separate the pulmonary artery from the right ventricle and the aorta from the left ventricle (Figure 12-3). The AV valves are the tricuspid valve, which

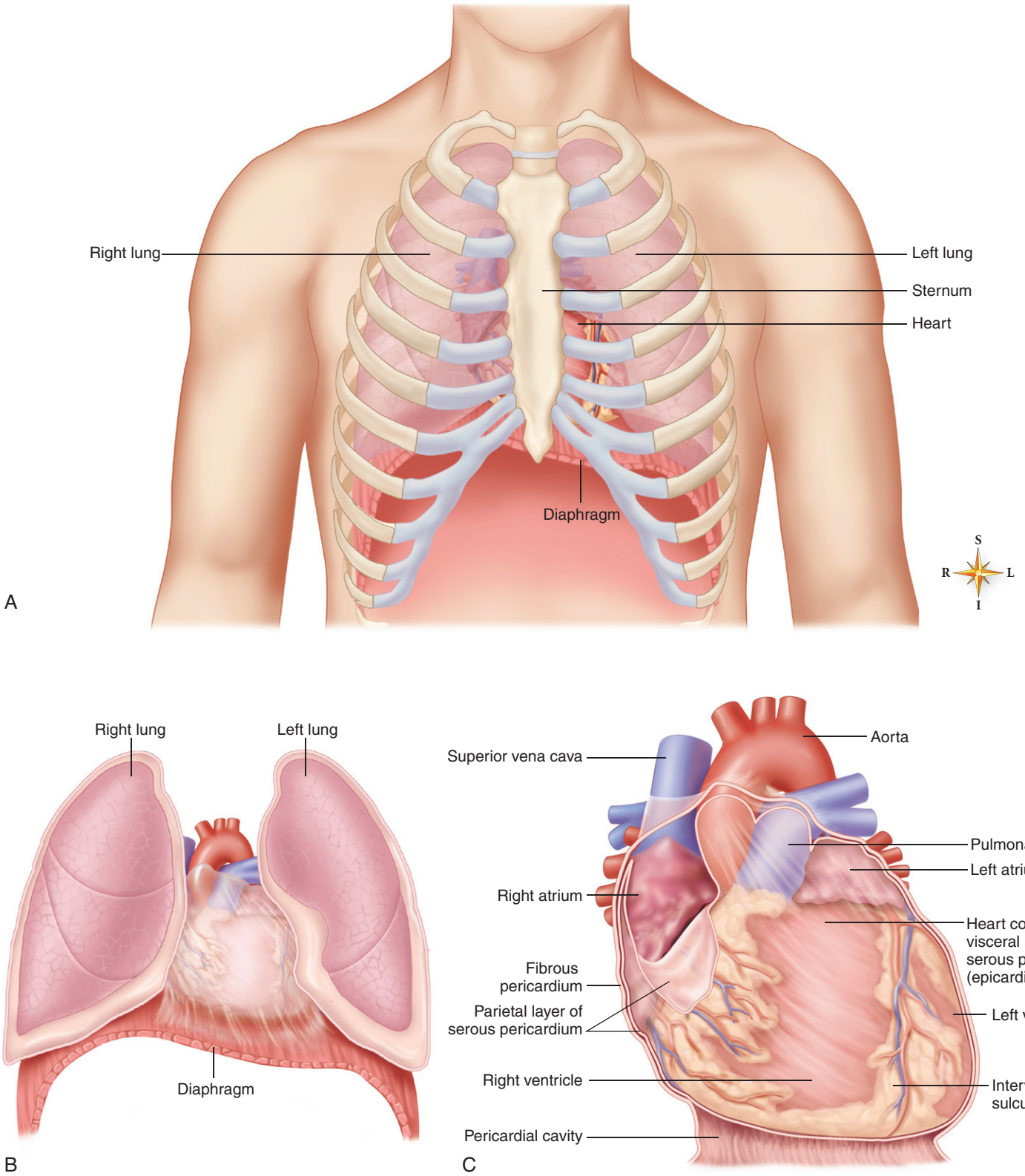

FIGURE 12-1 Location of the heart. **A,** Heart in mediastinum showing relationship to lungs and other anterior thoracic structures. **B,** Anterior view of isolated heart and lungs. Portions of the parietal pleura and pericardium have been removed. **C,** Detail of heart resting on diaphragm with pericardial sac opened.

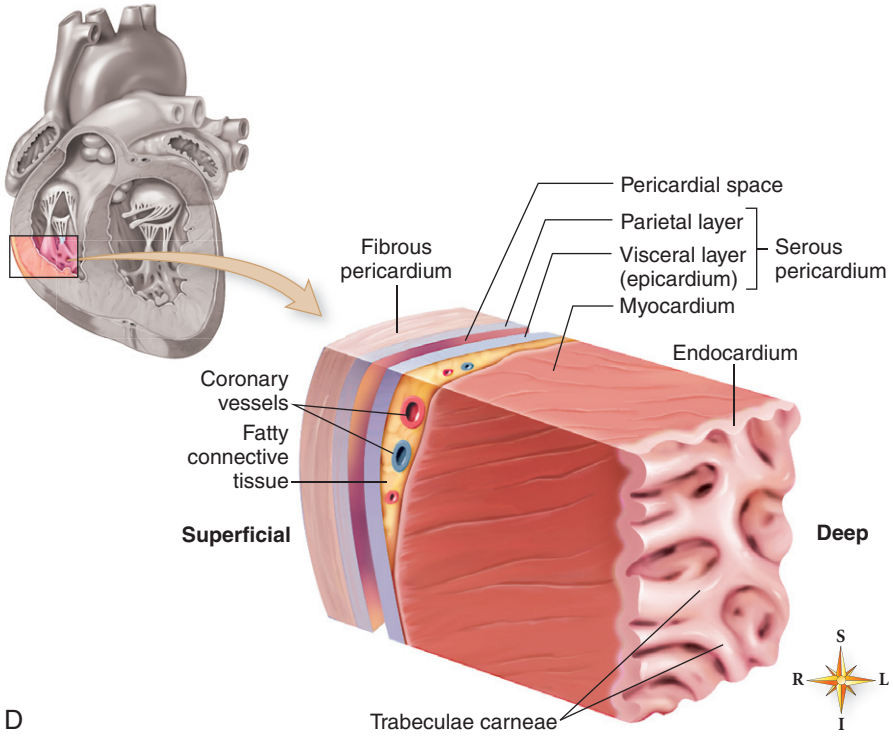

FIGURE 12-1, cont'd **D,** Wall of the heart. The cutout section of the heart wall shows the outer fibrous pericardium and the parietal and visceral layers of the serous pericardium (with the pericardial space between them). Note that a layer of fatty connective tissue is located between the visceral layer of the serous pericardium (epicardium) and the myocardium. Note also that the endocardium covers beamlike projections of myocardial muscle tissue, called *trabeculae carneae*. (From Patton KT, Thibodeau GA. *Anatomy and Physiology*. 8th ed. St. Louis: Mosby; 2013.)

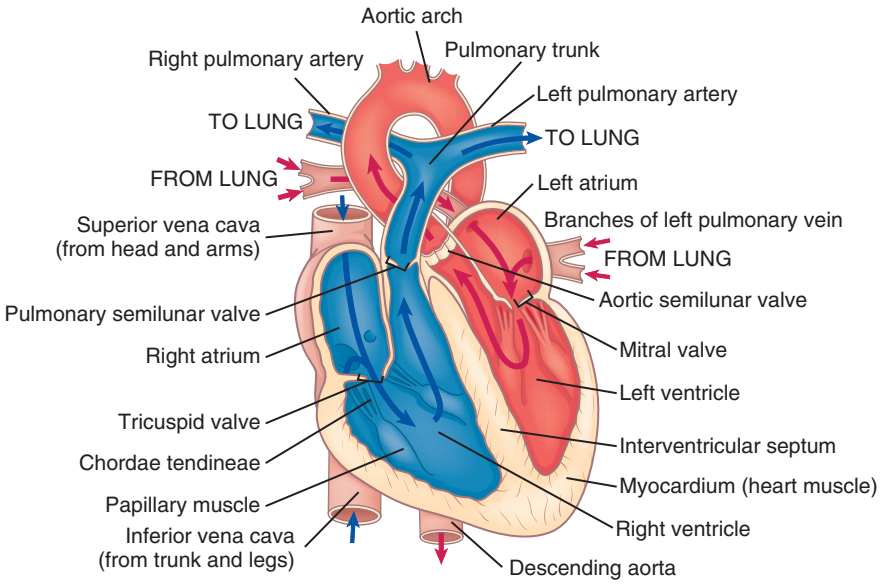

FIGURE 12-2 Structures that direct blood flow through the heart. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

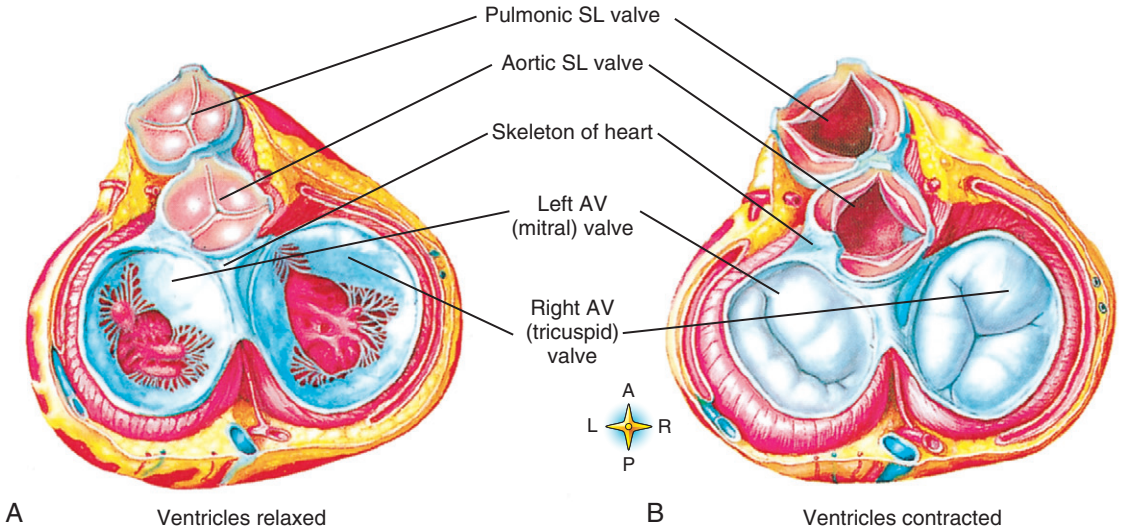

FIGURE 12-3 **A,** The atrioventricular (AV) valves in the open position and the semilunar (SL) valves in the closed position. **B,** The AV valves in the closed position and the SL valves in the open position. (From Patton KT, Thibodeau GA. *Anatomy and Physiology*. 7th ed. St. Louis: Mosby; 2010.)

lies between the right atrium and the right ventricle, and the mitral valve, located between the left atrium and the left ventricle. Each AV valve is anchored by chordae tendineae to the papillary muscles on its ventricular floor. The semilunar valves are the pulmonic valve, which lies between the right ventricle and the pulmonary artery, and the aortic valve, located between the left ventricle and the aorta. These semilunar valves are not anchored by chordae tendineae. Instead, their closing is passive and is caused by differences in pressure between the chamber and the respective great vessel.

Autonomic Control

The autonomic nervous system (sympathetic and parasympathetic) exerts control over the cardiovascular system. The sympathetic nervous system releases norepinephrine, which has alpha- and beta-adrenergic effects. Alpha-adrenergic effects cause arterial vasoconstriction. Beta-adrenergic effects increase sinus node discharge (positive chronotropic), increase the force of contraction (positive inotropic), and accelerate the AV conduction time (positive dromotropic).

The parasympathetic nervous system releases acetylcholine through stimulation of the vagus nerve. It causes a decrease in the sinus node discharge and slows conduction through the AV node.

In addition to this innervation, receptors help to control cardiovascular function. The first receptors are the *chemoreceptors*, which are sensitive to changes in the partial pressure of arterial oxygen (PaO_2), the partial pressure of arterial carbon dioxide ($PaCO_2$), and pH blood levels. Chemoreceptors stimulate the vasomotor center in the medulla; this center controls vasoconstriction and vasodilation. Second are *baroreceptors*, which are sensitive to stretch and pressure. If blood pressure increases, the baroreceptors cause the heart rate to decrease. If the blood pressure decreases, the baroreceptors stimulate an increase in heart rate (Figure 12-4).

Coronary Circulation

Many cardiac problems result from a complete or a partial occlusion of a coronary artery. The blood supply to the myocardium is derived from the coronary arteries that branch off the aorta immediately above the aortic valve (Figure 12-5). Two major branches exist: the left coronary artery, which splits into the left anterior descending and the left circumflex branches; and the right coronary artery. Knowledge of the portion of the heart that receives its blood supply from a particular coronary artery allows the critical care nurse to anticipate problems related to occlusion of that vessel (Box 12-1). Variations in the branching and the exact placement of the coronary arteries are common.

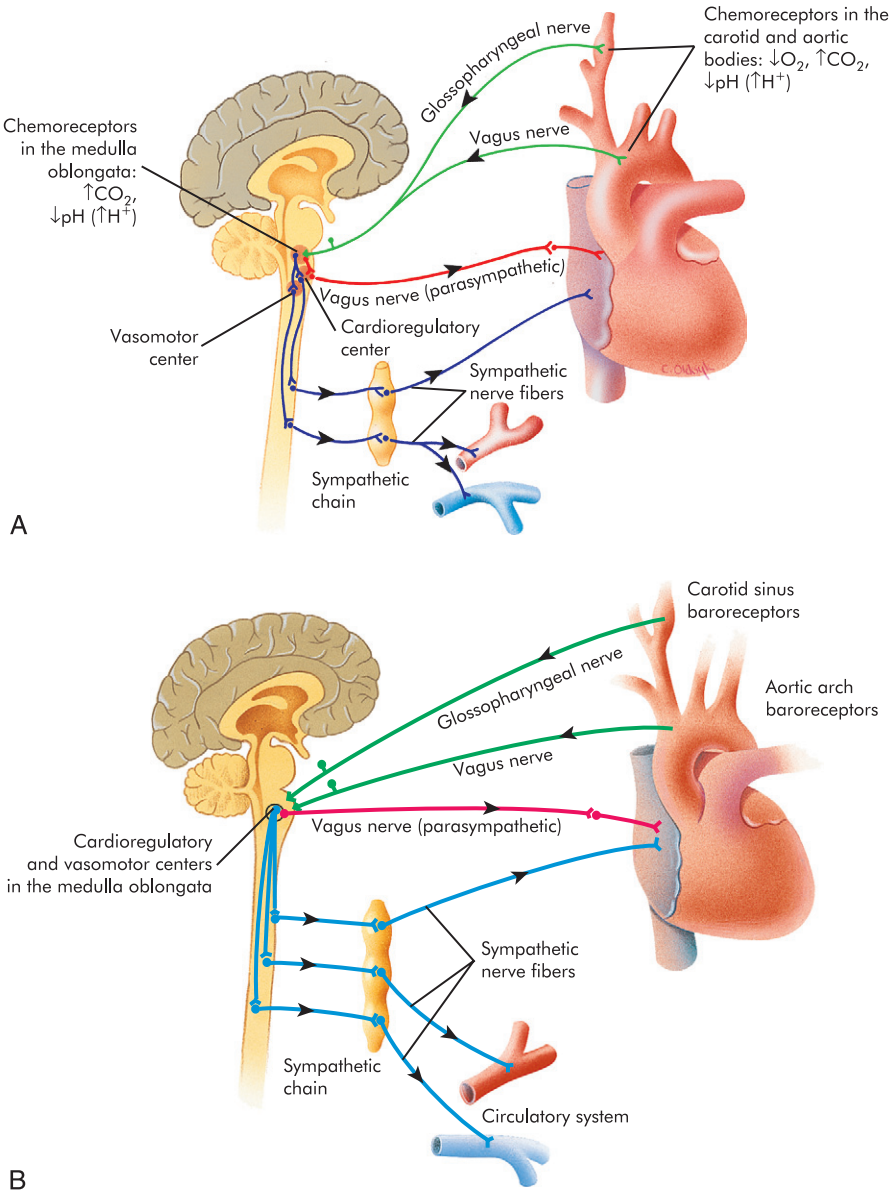

FIGURE 12-4 **A,** Chemoreceptor and **B,** baroreceptor reflex control of blood pressure. (From Seeley RR, Stephens TD, Tate P: *Anatomy and Physiology*. 3rd ed. St. Louis: Mosby; 1995.)

Blood flow to the coronary arteries occurs during ventricular diastole, when the aortic valve is closed and the sinuses of Valsalva are filled with blood. Myocardial fibers are relaxed at this time, thus promoting blood flow through the coronary vessels. The coronary veins return blood from the coronary circulation back into the heart through the coronary sinuses to the right and left atria.

Other Cardiac Functions

Knowledge of properties of cardiac muscle and the normal conduction system of the heart is essential since many patients have cardiac dysrhythmias (see Chapter 7). Hemodynamic concepts of the cardiovascular system are also important in understanding pathological disorders such as heart failure (HF). (Refer to Chapter 8 for hemodynamic content.)

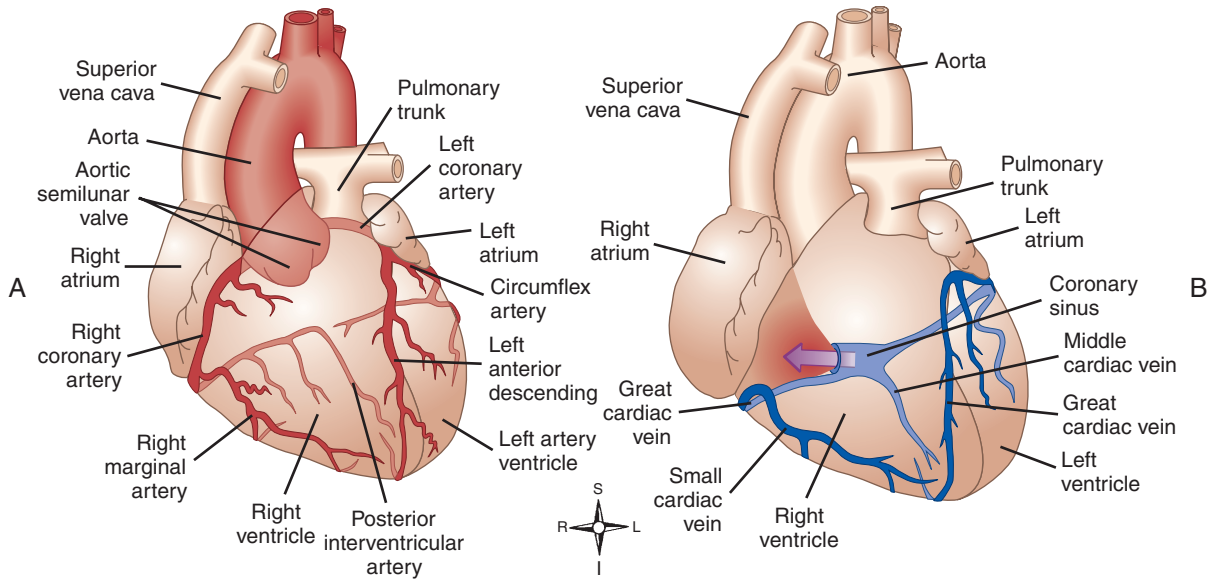

FIGURE 12-5 The coronary vessels. **A,** Arteries. **B,** Veins. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

BOX 12-1 CORONARY ARTERY DISTRIBUTION

Right Coronary Artery
- Right atrium
- Right ventricle
- Sinoatrial node
- Atrioventricular bundle
- Posterior portion of the left ventricle

Left Anterior Descending Artery
- Anterior two thirds of the intraventricular septum
- Anterior left ventricle

Circumflex Artery
- Left atrium
- Posterior left ventricle

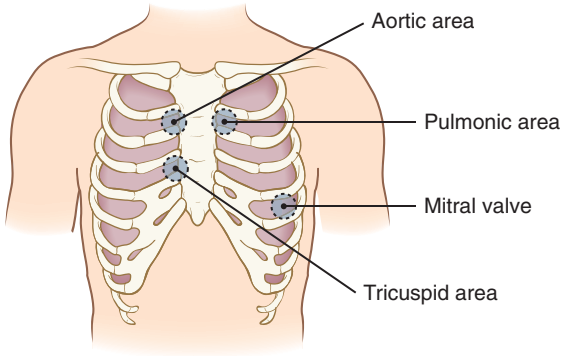

FIGURE 12-6 Chest areas from which each valve sound is best heard. (Modified from Hall JE, Guyton AC. *Guyton and Hall Textbook of Medical Physiology*. 12th ed. Philadelphia: Saunders; 2011.)

Heart Sounds

The vibrations produced by vascular walls, flowing blood, heart muscle, and heart valves create sound waves known as heart sounds. Auscultation of these sounds with a stethoscope over the heart provides valuable information about valve and cardiac function (Figure 12-6). Ventricular systole occurs when the pulmonic and aortic valves open to allow blood to be pumped to the lungs (right ventricle-pulmonic valve) and systemic circulation (left ventricle-aortic valve). Ventricular diastole occurs when the tricuspid and mitral valves open to allow the ventricles to fill with blood.

The first heart sound is known as S_1. This sound has been described as "lub." It is caused by closure of the tricuspid and mitral valves. It is best heard at the apex of the heart (fifth intercostal space, left midclavicular line) and represents the beginning of ventricular systole.

The second heart sound is known as S_2. It has been described as "dub" and is caused by closure of the pulmonic and aortic valves. It is best heard at the second intercostal space at the left or right sternal border and represents the beginning of ventricular diastole. The first and second heart sounds are best heard with the diaphragm of the stethoscope.

A third heart sound, S_3 or ventricular or protodiastolic gallop, is a normal variant in young adults, but usually represents a pathological process in the older adult. The sound is caused by rapid left ventricular filling and may be produced at the time when the heart is already overfilled or poorly compliant. The S_3 sound is low pitched and can best be heard with the bell of the stethoscope at the fifth intercostal space, at the left midclavicular line. It occurs immediately after S_2. Together with S_1 and S_2, S_3 produces a "lub-dubba" or "ken-**tuk**'e" sound. S_3 is often heard in patients with HF or fluid overload.

A fourth heart sound, S_4 or presystolic or atrial gallop, is produced from atrial contraction that is more forceful than normal. Together with S_1 and S_2, S_4 produces a "te-lubb-dubb" or "**ten**'-ne-see" sound. S_4 can be normal in elderly patients, but it is often heard after an acute myocardial infarction (AMI), when the atria contract more forcefully against ventricles distended with blood.

In the severely failing heart, all four sounds (S_4, S_1, S_2, and S_3) may be heard, producing a "gallop" rhythm (quadruple gallop), so named because it sounds like the hoof beats of a galloping horse. It can best be heard with the bell of the stethoscope at the fifth intercostal space, at the left midclavicular line. In addition, it is often documented S_4, S_1, S_2, S_3 because of the order in which the sounds are heard. Summation gallop is when the third and fourth heart sounds are superimposed and is usually an indication of heart disease.

Heart Murmur

A heart murmur is a sound caused by turbulence of blood flow through the valves of the heart. A murmur is usually a rumbling, blowing, harsh, or musical sound. It is important to distinguish the sound, anatomical location, loudness, and intensity of a murmur and determine whether extra heart sounds are heard. Table 12-1 gives a grading of heart murmurs.

TABLE 12-1 GRADING OF HEART MURMURS

Intensity of Murmur Graded from I to VI Based on Increasing Loudness

Grade I	Lowest intensity, usually not audible by inexperienced providers
Grade II	Low intensity, usually audible by inexperienced providers
Grade III	Medium intensity without a thrill
Grade IV	Medium intensity with a thrill
Grade V	Loudest murmur audible when stethoscope is placed on the chest; associated with a thrill
Grade VI	Loudest intensity, audible when stethoscope is removed from chest; associated with a thrill

Murmurs are audible when a septal defect is present; when a valve (usually aortic or mitral) is stenosed, or when the valve leaflets fail to approximate (valve insufficiency). The presence of a new murmur warrants special attention, particularly in a patient with AMI. A papillary muscle may have ruptured, causing the mitral valve to not close correctly, which can be indicative of severe damage and impending complications (HF and pulmonary edema). Auscultation of heart sounds is a skill developed from practice in listening to many different patients' hearts and in correlating the sounds heard with the patients' pathological conditions.

CORONARY ARTERY DISEASE

Coronary artery disease (CAD) is a broad term used to refer to the narrowing or occlusion of the coronary arteries. Other terms used to describe CAD include coronary heart disease and atherosclerotic heart disease.

Pathophysiology

CAD is the progressive narrowing of one or more coronary arteries by atherosclerosis. CAD results in ischemia when the internal diameter of the coronary vessel is reduced by 70% (Figure 12-7).[27]

Atherosclerosis is an inflammatory disease progressing from endothelial injury to fatty streak, plaque, and complex lesion. The process begins with injury to the endothelium due to cardiac risk factors such as smoking, hypertension, diabetes, and hyperlipidemia (Box 12-2). Once injury occurs, endothelial cells become inflamed causing release of cytokines. Macrophages adhere to the injured endothelium and release enzymes and toxic oxygen radicals that create oxidative stress, oxidize low-density lipoproteins (LDLs) and further injure the vessel. Inflammation with oxidative stress and activation of macrophages occurs. Oxidized LDL penetrate the arterial wall and are engulfed by macrophages, creating foam cells (Figure 12-8). Accumulation of foam cells lead to fatty streak formation.[31] By the age of 20 years, most individuals have fatty streaks, an accumulation of serum lipoproteins in the intima of the vessel wall, in their coronary arteries.[19] The dysfunctional formation of a fatty streak leads to the presence of fibrotic plaque. Growth factors are also released, including angiotensin II, fibroblast growth factor, and platelet-derived growth factor, which stimulate smooth muscle proliferation in the affected vessel. Over time, a collagen cap is formed from connective tissue (fibroblasts and macrophages) and LDL (Figure 12-7, *C*).

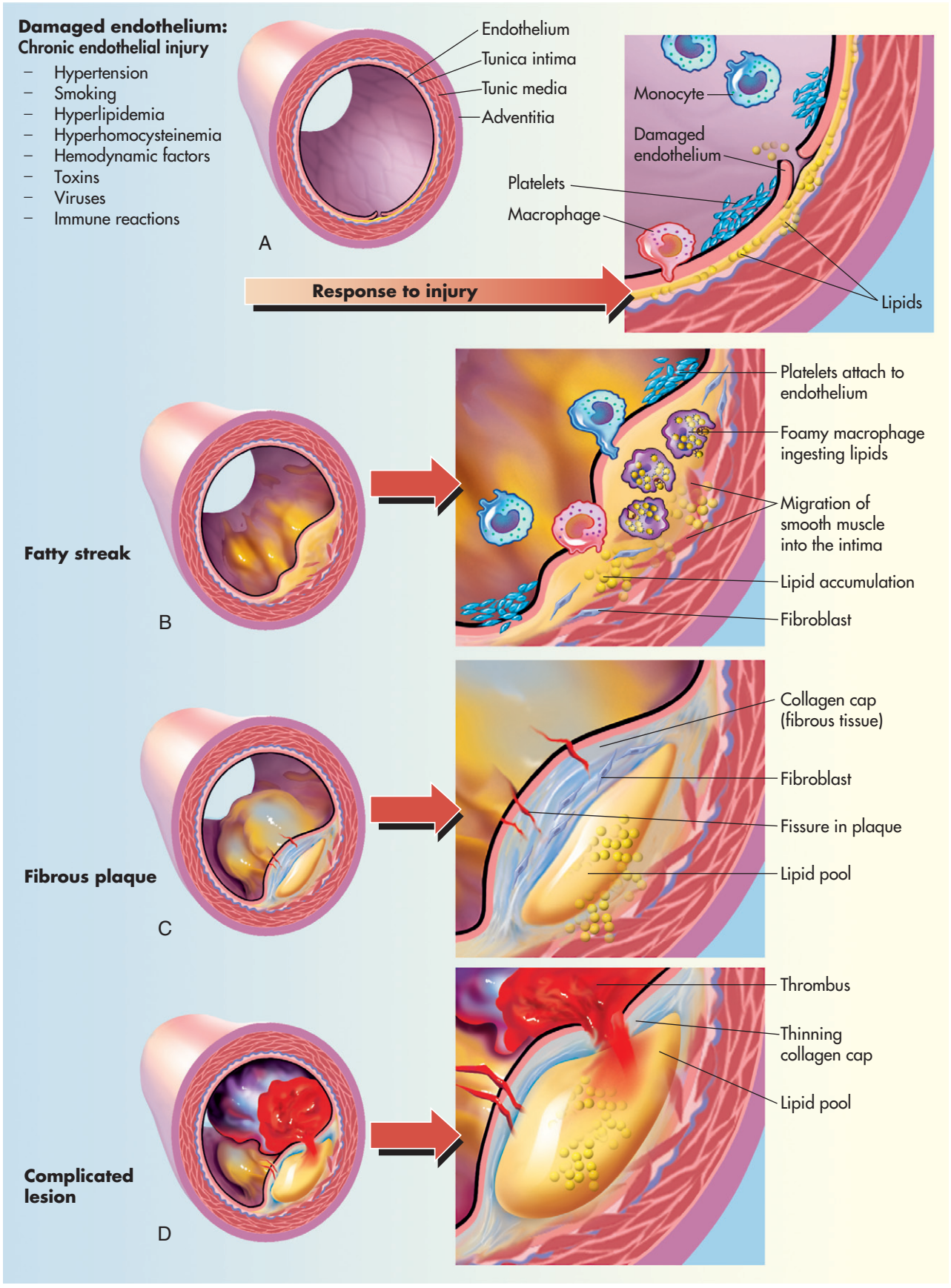

FIGURE 12-7 Progression of atherosclerosis. **A,** Damaged endothelium. **B,** Fatty streak. **C,** Fibrous plaque. **D,** Complicated lesion. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

BOX 12-2 RISK FACTORS FOR CORONARY ARTERY DISEASE

Several risk factors predispose persons to coronary artery disease (CAD). Some risk factors cannot be changed (e.g., gender, heredity, and age). Other risk factors are modifiable: smoking, high blood cholesterol, high blood pressure, physical inactivity, overweight or obesity, and diabetes.

Gender

Men have a greater risk of heart attacks than women and have heart attacks earlier in life.

Heredity

Family history of early heart disease is an unmodifiable risk for CAD. A positive history is defined as having a first degree relative (parent, sister, brother, or child) with CAD having been diagnosed before age 55 years in male relatives and before age 65 years in female relatives.[33]

Age

Men in their mid 40s, and women once they reach menopause, are considered at higher risk for CAD.

Smoking

Smokers have a higher risk of CAD. Smoking increases low-density lipoprotein (LDL) levels and damages the endothelium of coronary vessels. These are predisposing factors for the development of atherosclerosis. Smoking also causes vasoconstriction of coronary vessels, thus decreasing blood supply.

Blood Cholesterol

Serum cholesterol or lipid levels play a key role in the development of atherosclerosis. Elevated total cholesterol (>200 mg/dL) is considered a risk factor for CAD. Cholesterol is insoluble in plasma and must be transported by lipoproteins that are soluble. High-density lipoproteins (HDLs) are considered the good cholesterol. HDLs assist in transporting cholesterol to the liver for removal. A high HDL level (>40 mg/dL for men and >50 mg/dL for women) may reduce the incidence of CAD, whereas a low HDL level (<40 mg/dL) is considered a risk factor for developing CAD.

LDLs are considered the bad cholesterol. LDLs transport and deposit cholesterol to the arterial vessels, thus facilitating the process of atherosclerosis. An LDL level <100 mg/dL is optimal. Other non-HDL lipoproteins also contribute to the development of CAD. Very-low-density lipoproteins are largely composed of triglycerides and contribute to an increased risk of CAD.

High Blood Pressure

A blood pressure (BP) greater than 120/80 mm Hg is considered prehypertension. A BP greater than 140/89 mm Hg or taking antihypertensive medication is a risk factor for CAD. Hypertension causes direct injury to the vasculature, leading to the development of CAD. Oxygen demands are also increased. The heart muscle enlarges and weakens over time, thereby increasing the workload of the heart.

Physical Inactivity

Lack of physical activity is a risk factor for CAD. Regular aerobic exercise reduces the incidence of CAD. Exercise also helps to control other risk factors such as high blood pressure, diabetes, and obesity.

Overweight and Obesity (See Bariatric Considerations)

Obesity increases the atherogenic process and predisposes persons to CAD. In addition, obesity is related to hypertension and diabetes, two other major risk factors. The waist-to-hip ratio and body mass index (BMI) are important assessments.

Diabetes

Diabetes is associated with increased levels of LDL and triglycerides. Glycation associated with diabetes decreases the uptake of LDL by the liver and increases the hepatic synthesis of LDL.

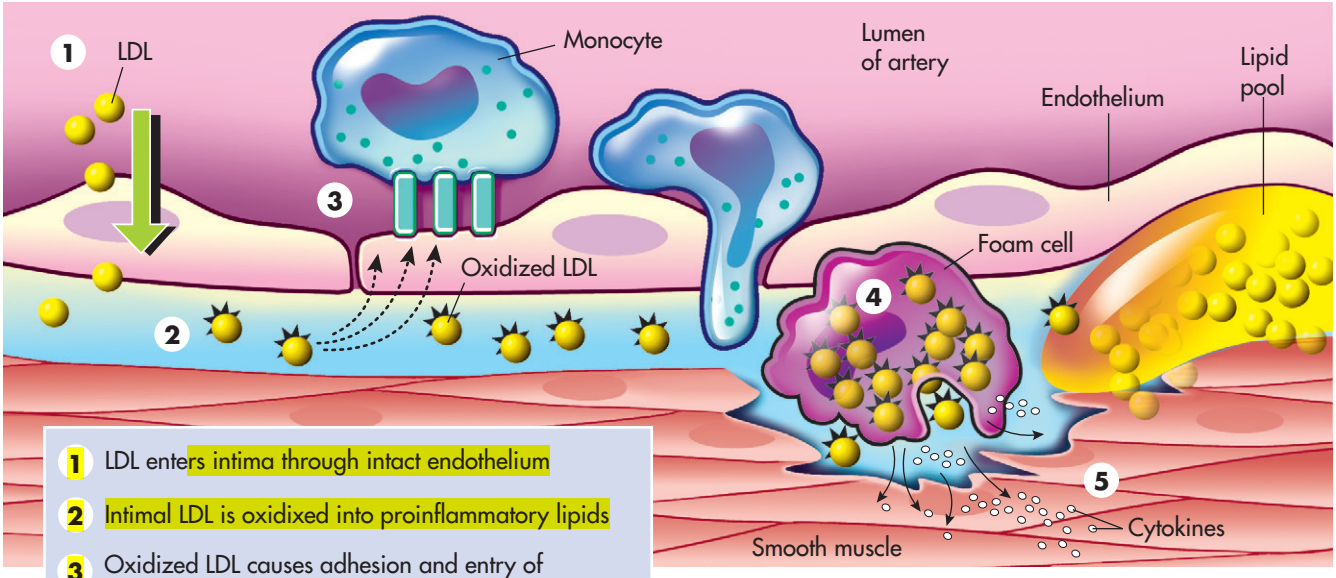

FIGURE 12-8 Low-density lipoprotein oxidation. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010; Modified from Crawford MH, Dim Arco JP, eds: *Cardiology*. London: Mosby-Wolfe; 2001.)

BARIATRIC CONSIDERATIONS

Obesity is a risk factor of coronary artery disease (CAD). Persons with a greater proportion of fat through the abdomen ("apple-shaped") have been shown to have a higher incidence of CAD than those with greater fat distribution over the hips ("pear-shaped"). The waist-to-hip ratio is used to help identify this risk. Body mass index is another way to determine the degree of overweight.

Bariatric surgery may result in improved cardiac function and may assist in preventing heart failure. Weight loss in patients with morbid obesity has been associated with changes in left ventricular structure and improved right ventricular function.

Reference

Garza CA, Pellikka PA, Somers VK, Sarr MG, Collazo-Clavell ML, Korenfeld Y, Lopez-Jimenez F. Structural and functional changes in left and right ventricles after major weight loss following bariatric surgery for morbid obesity. *American Journal of Cardiology.* 2010;105:550-556.

Plaques may rupture, with the contents interacting with blood, producing a thrombus. The thrombus can occlude a coronary artery, with resulting injury and infarction. Rupture of the plaque starts the coagulation cascade with the initiation of thrombin production, the conversion of fibrinogen to fibrin, and platelet aggregation at the site. After injury to the endothelium, platelets are exposed to proteins that bind to receptors, causing adhesion of platelets at the site of injury. Next, the platelets are activated and change shape. They release thromboxane A_2 and serotonin. Each platelet has thousands of glycoprotein IIb/IIIa (Gp IIb/IIIa) receptors that are activated and bind with von Willebrand factor and fibrinogen, which is converted to fibrin strands. At the same time, the platelets aggregate with one another. This process of adhesion, activation, and aggregation causes a rapidly growing thrombus that compromises coronary blood flow.[31]

Assessment

Patient Assessment

A thorough history and cardiovascular assessment provide data to develop a comprehensive plan of care for the critically ill patient with cardiovascular disease. The history includes subjective data regarding medical history, prior hospitalizations, allergies, and family medical history. Several risk factors that are associated with CAD are also assessed: hypertension, hyperlipidemia, diabetes, male gender, age, tobacco use, lack of physical activity, obesity, and family history (see Box 12-2). Knowledge of prior hospitalizations is also important so medical records can be obtained for review. Records are especially useful if the patient was hospitalized for a cardiac event or underwent cardiac diagnostic testing. Information regarding the patient's current medications, both prescription and over-the-counter, includes assessment of the patient's understanding and use of these medications. For example, when considering nitroglycerin (NTG) administration, it is necessary to know the history of the patient's use of phosphodiesterase type 5 (PDE5) inhibitors taken for erectile dysfunction, such as sildenafil (Viagra). These medications potentiate the hypotensive effects of nitrates such as NTG; thus concurrent use is contraindicated. It is also important to determine whether the patient has any food or drug allergies.

A psychosocial or personal history is important for the planning of the critically ill patient's care. This history includes major stress events and everyday stressors (Box 12-3).

Before beginning the physical examination, the nurse determines recent and recurrent symptoms that may be related to the patient's current problems. Such information gathering includes the presence or absence of fatigue, fluid retention, dyspnea, irregular heartbeat (palpitations), and chest pain (see box, "Clinical Alert: Assessment of the Patient with Chest Pain [PQRST]"). The physical examination itself encompasses all the body systems and is not limited to the cardiovascular system. Because all the body systems are interrelated and interdependent, it is imperative that a total evaluation be completed regarding the physical status of the patient. Patients whose primary problems are cardiovascular most commonly exhibit alterations in circulation and oxygenation. Thus all systems should be examined from this perspective.

BOX 12-3 QUESTIONING OF ACTIVITIES FOR STRESS REDUCTION

- What, if any, is the critically ill patient's exercise routine, including the type, amount, and regularity of the activity?
- What is the critically ill patient's daily food pattern and intake?
- What is the critically ill patient's sleep pattern?
- What are the critically ill patient's habitual social patterns in using tobacco, alcohol, drugs, coffee, tea, and caffeinated sodas?

! CLINICAL ALERT

***Assessment of the Patient with Chest Pain* (PQRST)**

P	Provocation
Q	Quality
R	Region/Radiation
S	Severity
T	Timing (when began) and Treatment

The examination is performed in an orderly, organized manner and involves the techniques of inspection, palpation, percussion, and auscultation. A baseline assessment is provided in Table 12-2.

TABLE 12-2 MAJOR SYSTEMS ASSESSMENT FOR CARDIOVASCULAR DISEASE

SYSTEM	ASSESSMENT
Neurological	Level of consciousness, orientation to person, place, time, events; presence of hallucinations, depression, withdrawal, restlessness, apprehensiveness, irritability, cooperativeness, response to tactile stimuli; type, location of pain; how pain is relieved; trembling; pupils (size, equality, response); paresthesias; eye movements; hand grips (strength and equality); leg movement
Skin	Color (mottling, cyanosis, pallor), temperature, dryness, turgor, presence of rashes, broken areas, pressure areas, urticaria, incision site, wounds
Cardiovascular	BP (bilaterally); apical heart rate and radial pulses; pulse deficit; monitor leads on patient in correct anatomical placement; regularity of rhythm, presence of ectopy; PR interval, QRS, and QT intervals; heart sounds; presence of abnormalities (rubs, gallops, clicks); neck vein distention with head of bed at what angle; edema (sacral and dependent); calf pain; varicosities; presence of pulses: bilateral carotid, radial, femoral, posterior tibial, dorsalis pedis; capillary refill in extremities; hemodynamic measurements; temporary pacemaker settings; medications to maintain BP or rhythm
Respiratory	Rate, depth, and quality of respirations; oxygen needs; accessory muscle use; cough, sputum: type, color, suctioning frequency; symmetry of chest expansion and breath sounds, breath sounds (crackles, wheezing); interpretation of ABGs; chest tube with description of drainage, fluctuation in water seal, bubbling, suction applied; tracheostomy or endotracheal tube; ventilator used; ventilator settings; ventilator rate versus patient's own breaths
Gastrointestinal	Abdominal size and softness, bowel sounds, nausea and vomiting, bowel movement, dressing and/or drainage, NG tube with description of drainage, feeding tube: type and frequency of feedings, drains
Genitourinary	Voiding or indwelling urinary catheter, urine color, quality and quantity; vaginal or urethral drainage
Intravenous	Volume of fluid, type of solution, rate; intravenous site condition
Wounds	Dry or drainage, type, color, amount, odor; hematoma, inflamed, drains, hemovac, dressing changes, cultures

ABG, Arterial blood gas; *BP*, blood pressure; *NG*, nasogastric.

Diagnostic Studies

Many diagnostic studies are fundamental for the care and treatment of critically ill patients with CAD. The following sections contain brief descriptions of common diagnostic studies the cardiac patient may encounter.

12-Lead electrocardiography (ECG; also commonly referred to as EKG). This noninvasive test is usually preliminary to most other tests performed. It is useful in identification of rhythm disturbances, pericarditis, pulmonary diseases, left ventricular hypertrophy, and myocardial ischemia, injury, and infarction. The importance of this basic test should not be underestimated.

Chest x-ray. The chest x-ray is usually performed in the anteroposterior view. The chest x-ray study is used for detecting cardiomegaly, cardiac positioning, degree of fluid infiltrating the pulmonary space or pericardial space, and other structural changes that may affect the physical ability of the heart to function in a normal manner.

Holter monitor. This test is used to detect suspected dysrhythmias. The patient is connected to a small portable recorder (about the size of a large cellular phone) by three to five electrodes; the recorder is worn for 24 to 48 hours. The patient engages in normal daily activities, and keeps a log of all activities and symptoms during the monitoring period. The recording is analyzed for abnormalities and correlated with the documented activities and symptoms.

Exercise tolerance test (ETT) or stress test. This is a noninvasive test in which the patient is connected to an ECG machine while exercising. Physical stress causes an increase in myocardial oxygen consumption. If oxygen demand exceeds supply, ischemia may result. The stress test is used to document exercise-induced ischemia, and it can identify those individuals prone to cardiac ischemia during activity when resting ECGs are normal. The exercise usually involves pedaling a stationary bike or walking on a treadmill. The patient is constantly monitored, the pulse and blood pressure are checked at intervals, and the ECG printout is analyzed at the end of the testing period. Changes in the ST segments of the ECG can indicate ischemia. Beta-blockers are often held the morning of an ETT so that an adequate heart rate can be attained during the test. Patients return to their room or go home after the heart rate returns to baseline.

Pharmacological stress testing. If a patient is physically unable to perform the exercise, a pharmacological stress test can be done. This is done in conjunction with radionuclide scintigraphy or echocardiography. Medications such as regadenoson, dipyridamole, or adenosine are used because they cause vasodilation of normal coronary arteries. If an area of the blood vessel is stenosed, it does not dilate and shows up as hypoperfusion with radionuclide scanning, or as hypokinesis with echocardiography. Alternatively, dobutamine can be used, which increases heart rate and contractility. Areas that are not perfused well because of blockages are evident when scanned.

Nuclear stress testing. This test can be done with exercise to increase the sensitivity of the test and/or be used for

patients who have an ECG that precludes an accurate interpretation of ST-segment changes. It is also used in conjunction with medications for patients who cannot walk on a treadmill. Technetium-99m and thallium-201 are radionuclides given intravenously to image the heart at rest and at stress (induced either by exercise or use of a pharmacological agent). The stress images are compared to the rest images. Perfusion defects seen on rest and stress images are evidence of infarct, whereas defects noted on stress that are normal during the rest study indicate ischemia.

Echocardiography. This is a noninvasive, acoustic imaging procedure that uses ultrasound to visualize the cardiac structures and the motion and function of cardiac valves and chambers. A transducer placed on the chest wall sends ultrasound waves at short intervals. The reflected sound waves, termed echoes, are displayed on a graph for interpretation. Echocardiography is used to assess valvular function, evaluate congenital defects, measure size of cardiac chambers, evaluate cardiac disease progression, evaluate ventricular function, diagnose myocardial tumors and effusions, and, to a lesser degree, measure cardiac output. Ventricular function is evaluated by obtaining an ejection fraction. The ejection fraction is the percentage of blood ejected from the left ventricle during systole, normally 55% to 60%.

Transesophageal echocardiography. This test provides ultrasonic imaging of the heart from a view behind the heart. In transesophageal echocardiography (TEE), an ultrasound probe is fitted on the end of a flexible gastroscope, which is inserted into the posterior pharynx and advanced into the esophagus. TEE shows a clear picture because the esophagus is against the back of the heart and parallel to the aorta. TEE is indicated to visualize prosthetic heart valves, mitral valve function, aortic dissection, vegetative endocarditis, congenital heart defects in adults, cardiac masses and tumors, and embolic phenomena. It is also used intraoperatively to assess left ventricular function. Patients should fast (except for medications) for 6 to 8 hours before the examination. During the procedure, vital signs, cardiac rhythm, and oxygen saturation are monitored. After the procedure, the patient is unable to eat until the gag reflex returns. A rare complication of TEE is esophageal perforation, with signs of sore throat, dysphagia, stiff neck, and epigastric or substernal pain that worsens with breathing and movement, or pain in the back, abdomen, or shoulder.

Multigated blood pool study. The multigated blood pool study (MUGA) scan is used to assess left ventricular function. An isotope is injected and images of the heart are taken during systole and diastole to assess the ejection fraction of the heart. An ejection fraction of 55% to 60% and symmetrical contraction of the left ventricle are considered normal test results.

Cardiac magnetic resonance imaging. Magnetic resonance imaging (MRI) is a noninvasive test used to evaluate tissues, structures, and blood flow. MRI is a technique that uses magnetic resonance to create images of hydrogen. These images are created as the ions are emitted, picked up, and fed into a computer that reconstructs the image that can differentiate between healthy and ischemic tissue. MRI is used to diagnose or evaluate coronary artery disease, aortic aneurysm, congenital heart disease, left ventricular function, cardiac tumors, thrombus, valvular disease, and pericardial disorders. One of the advantages of MRI is that it does not involve exposure to ionizing radiation. Contrast can be used with MRI to enhance results.

MRI cannot be performed on patients with pacemakers, defibrillators, cochlear implants, or some types of brain clips (used for aneurysms). The test can be very stressful for patients who are claustrophobic, and in these situations, open MRI may be indicated. ECG monitoring is difficult to use during MRI; therefore critically ill patients may not be good candidates for this procedure. Specialized monitors have been developed specifically for use in MRI to facilitate cardiac monitoring when indicated.

Cardiac catheterization and angiography. This is an invasive procedure that can be divided into two stages (right-sided and left-sided catheterization). Cardiac catheterization is used to measure pressures in the chambers of the heart, cardiac output, and blood gas content; confirm and evaluate the severity of lesions within the coronary arteries; and to assess left ventricular function.

Right-sided catheterization is performed by placing a pulmonary artery catheter in the femoral or brachial vein and then carefully advancing it into the right atrium, right ventricle, and pulmonary artery. The healthcare provider measures pressures in the right atrium, right ventricle, and pulmonary artery, and the pulmonary artery occlusion pressure. Oxygen saturations can be measured if indicated (i.e., valve disease or septal defect).

Left-sided catheterization is performed to visualize coronary arteries, to note the area and extent of lesions within the native vessel walls and bypass grafts, to evaluate angina-related spasms, to locate areas of infarct, and to perform interventions such as percutaneous angioplasty or stent placement.

Left-sided catheterization is performed by cannulation of a femoral, brachial, or radial artery. The procedure entails positioning a catheter into the aorta at the proximal end of the coronary arteries. Dye is injected into the arteries, and a radiographic picture is recorded as the dye progresses or fails to progress through the coronary circulation. In addition, dye is injected into the left ventricle, and the amount of dye ejected with the next systole is measured to determine the ejection fraction.

After the procedure the catheters are removed. To prevent bleeding from the arterial site, a vascular sealing device made of collagen (e.g., AngioSeal), a clip-mediated device (e.g., StarClose), or a stitch device (e.g., Perclose) may be used to close the puncture site in the artery. A hemostatic bandage (e.g., QuickClot) can also be used. If a sealing or stitch device is not used, firm pressure is applied for 15 to 30 minutes. Commercial devices (e.g., FemoStop; Figure 12-9) are available to assist in applying pressure to the site. Depending on the diagnostic study results and the patient's status, patients are usually discharged within 6 to 8 hours of completion of the test.

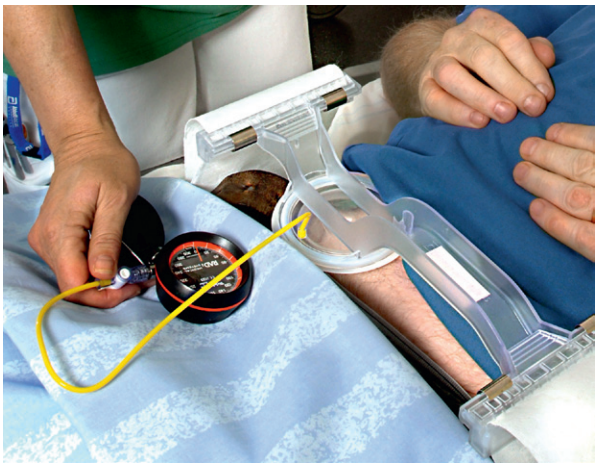

FIGURE 12-9 FemoStop in correct position. (Courtesy RADI Medical Systems, Inc. Sweden.)

BOX 12-4 NURSING CARE AFTER CARDIAC CATHETERIZATION AND ANGIOGRAPHY

- Maintain the patient on bed rest (time varies depending on the size of the catheter used and the method for preventing arterial bleeding).
- Keep the extremity used for catheter insertion immobile.
- Observe the insertion site for bleeding or hematoma, especially if the patient is receiving postprocedure anticoagulant therapy.
- Mark the hematoma with a marker around outer perimeter, to aid in assessing for an increase in bleeding.
- Assess for bruits.
- Maintain head-of-bed elevation no higher than 30 degrees.
- Monitor peripheral pulses, color, and sensation of the extremity distal to insertion site (every 15 minutes × 4; every 30 minutes × 4; every 1 hour × 2). In addition, monitor the opposite extremity pulse to assess for presence of equal pulses and color, and sensation bilaterally.
- Observe cardiac rhythm.
- Encourage fluid intake if not contraindicated.
- Monitor intake and output.
- Observe for an adverse reaction to dye (angiography).
- Assess for chest pain, back pain, and shortness of breath and notify health care provider.

Nursing care for a patient undergoing cardiac catheterization involves the preprocedure instruction (the procedure will be performed using local anesthesia, and the patient may feel a warm or hot flush sensation or flutter of the catheter as it moves about) and the postprocedure instruction. The postprocedure routine is noted in Box 12-4.

Electrophysiology study. An electrophysiology study is an invasive procedure that involves the introduction of an electrode catheter percutaneously from a peripheral vein or artery into the cardiac chamber or sinuses and the performance of programmed electrical stimulation of the heart. Electrophysiology studies aid in recording intracardiac ECGs, diagnosing cardiac conduction defects, evaluating the effectiveness of antidysrhythmic medications, determining the proper choice of pacemaker programming, and mapping the cardiac conduction system before ablation.

Laboratory Diagnostics

Other diagnostic measures include the evaluation of serum electrolyte studies and cardiac enzymes. Because many books are available regarding the reading and interpretation of laboratory values, this section presents a brief overview of the more important blood studies that should or may need to be assessed in the patient with a cardiovascular alteration.[10]

Serum electrolytes. Electrolytes are important in maintaining the function of the cardiac conduction system. Imbalances in sodium, potassium, calcium, and magnesium can result in cardiac dysrhythmias. Therefore analysis of serum electrolytes is a routine part of the assessment and treatment of the cardiac patient. Table 12-3 reviews ECG changes that may alert the nurse to possible electrolyte abnormalities.

Serum enzymes. Enzymes are proteins that are produced by all living cells and released into the bloodstream. When cells are injured or diseased, more enzymes are released. Assessments of enzyme levels released from cardiac muscle are useful in the diagnosis of AMI.[9]

- *Creatine kinase (CK)* enzymes increase within 2 to 6 hours after the onset of myocardial muscle damage. Peak levels occur within 18 to 36 hours, and levels return to baseline in 3 to 6 days. Total CK can be elevated from a variety of diseases and conditions, such as muscle injury and acute renal failure and therefore is nonspecific.

TABLE 12-3 ECG CHANGES ASSOCIATED WITH ELECTROLYTE IMBALANCES

ELECTROLYTE IMBALANCE	PANIC VALUE	MANIFESTATIONS
Hypokalemia	<2.5 mEq/L	U wave, increased ventricular ectopy
Hyperkalemia	>6.6 mEq/L	Tall, peaked T waves, conduction blocks, ventricular fibrillation
Hypocalcemia	<7.0 mg/dL	Prolonged ST segment and QT interval
Hypercalcemia	>12.0 mg/dL	Shortened ST segment and QT interval
Hypomagnesemia	<0.5 mEq/L	Prolonged PR and QT intervals, broad, flat T waves, PVCs, ventricular tachycardia or fibrillation
Hypermagnesemia	>3.0 mEq/L	Prolonged PR and QT intervals, widened QRS

PVCs, Premature ventricular contractions.
Modified from Chernecky CC, & Berger BJ. (2008). *Laboratory tests and diagnostic procedures* (5th ed.). Philadelphia: Saunders.

- *CK_2-MB (heart)* is a fraction of the total CK that is specific for cardiac muscle. Normal values of CK_2-MB are 0% to 6% of the total CK, or 0.3 to 4.9 ng/mL. Values are elevated after AMI, cardiac surgery, and blunt cardiac trauma. The initial rise in CK_2-MB levels after an AMI occurs within 4 to 8 hours after the onset of damage. Peak levels occur in 18 to 24 hours, and levels return to baseline within 3 days. Total CK and CK_2-MB are usually ordered at the initial assessment and serially at 8, 16, and 24 hours after the onset of chest pain to assist in the diagnosis of AMI.
- *Troponin I and troponin T*. Serum troponin levels are useful in the early diagnosis of AMI. Levels are normally undetectable in healthy people and elevate as early as 1 hour after myocardial cell injury. Testing for troponin can be done quickly in the field or the emergency department and aids in the early diagnosis of AMI. The normal value of troponin I is less than 0.5 mcg/L, and that of troponin T is less than 0.1 mcg/L.
- *Myoglobin*. Serum myoglobin is released within 30 to 60 minutes after AMI. Normal values are less than 72 ng/mL in men, and less than 58 ng/mL in women. Myoglobin levels rise before CK and CK_2-MB and are useful in the early diagnosis of AMI. Myoglobin alone is not specific for AMI, but when used in combination with other tests, it can aid in the diagnosis. Some institutions order myoglobin levels every 2 hours. A doubling of levels from one sample to the next sample is indicative of AMI.

Nursing Diagnoses

CAD is a broad diagnostic area, and thus several nursing diagnostic categories apply. With the complications of CAD, such as angina, MI, and HF, the diagnostic categories are more specific. Nursing diagnoses of patients with CAD include the following:

- Pain related to decreased coronary artery tissue perfusion
- Anxiety/fear related to treatments and invasive procedures used for diagnostic testing
- Knowledge deficit related to understanding of anatomy and pathophysiology of the heart and its functions, complexity of treatment, new condition, emotional state
- Health-seeking behaviors related to desire for information regarding altered health status or a disease process or condition

Interventions

Nursing Interventions

Nursing interventions are patient centric and encompass health assessment and patient education. Assessment of the patient's psychosocial status and family support, as well as the patient's history and physical examination findings, are used to guide interventions. The nurse instructs the patient about risk factor modification and signs and symptoms of progression of CAD that warrant medical treatment.

Medical Management

The goals of medical management are to reduce the risk factors for progression of CAD. This includes achieving target levels of LDL. The National Cholesterol Education Project of the National Heart, Lung, and Blood Institute recommends that an optimal LDL level is less than 100 mg/dL, but the target level should be adjusted in relation to the patient's number of major risk factors for CAD.[4] These include family history, age, smoking, hypertension, and diabetes. The key to lessening the burden of coronary heart disease in the United States is primary prevention, and one way this can be accomplished is through thorough management of cholesterol levels (see box, "Laboratory Alert," for LDL, high-density lipoproteins [HDL], and triglyceride levels).

Strategies for risk factor modification include a low-fat, low-cholesterol diet, exercise, weight loss, smoking cessation, and control of other risks such as diabetes and hypertension. If LDL levels are not at target values after 6 months of risk factor modification, patients are started on lipid-lowering medications.

Medications to reduce serum lipid levels. Lipid-lowering drugs include statins, bile acid resins, ezetimibe, and nicotinic acid (Table 12-4). The statins are officially classified as 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors. The statins lower LDL more than other types of lipid-lowering drugs. They work by slowing the production of cholesterol and increasing the liver's ability to remove LDL from the body. Some commonly used drugs are lovastatin, atorvastatin, pravastatin, simvastatin, and rosuvastatin. The drugs are well tolerated by most patients. It is recommended that statins be given as a single dose in the evening because the body makes more cholesterol at night.

! LABORATORY ALERT

Target Lipid Levels

RISKS	TARGET LOW-DENSITY LIPOPROTEIN LEVEL	TARGET HIGH-DENSITY LIPOPROTEIN LEVEL	TARGET TRIGLYCERIDE LEVEL
No CAD; 0-1 risk factors	<160 mg/dL	>60 mg/dL	<190 mg/dL
No CAD; 2 or more risk factors	<130 mg/dL	>60 mg/dL	<160 mg/dL
CAD or CAD risk equivalent (other atherosclerotic disease, diabetes, multiple risks)	<100 mg/dL	>60 mg/dL	<130 mg/dL

CAD, Coronary artery disease.

TABLE 12-4 PHARMACOLOGY

Medications for Lowering Cholesterol and Triglycerides

Antilipemic Agents (HMG-CoA Reductase Inhibitors)

Indications: used to lower total and LDL cholesterol and to help reduce the risk of acute myocardial infarction and stroke

Mechanism of action: competitively inhibit HMG-CoA reductase, the enzyme that catalyzes the rate-limiting step in cholesterol biosynthesis, resulting in lower total and LDL cholesterol levels with increased HDL cholesterol

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Lovastatin (Altocor, Mevacor)	10-80 mg PO once daily (in the evening) or in two divided doses	Headache, dizziness, constipation, weakness, and increased creatine phosphokinase (CPK) levels Instruct patient to take with evening meal. Report severe muscle pain, or weakness, which can be a sign of rhabdomyolysis, a serious side effect. Obtain baseline liver function and lipid profile tests before starting therapy, every 2 months for first year, then at 6 and 12 months, or when dose increased. Do not give in pregnancy. Instruct patient about a low-cholesterol diet.
Atorvastatin (Lipitor)	10-80 mg PO daily	Same
Fluvastatin (Lescol)	20-80 mg daily PO at bedtime	Same
Pravastatin (Pravachol)	10-80 mg daily PO at bedtime	Same
Rosuvastatin (Crestor)	5-40 mg daily PO at bedtime	Same
Simvastatin (Apo-Simvastatin, Zocor)	5-80 mg daily PO at bedtime	Same as above. The FDA has mandated no more new prescriptions of 80 mg. Patients that have been stable for over a year may continue to take 80 mg. Also, no more than 20 mg for patients taking amlodipine or ranolazine, and no more than 10 mg for patients on amiodarone, verapamil, or diltiazem. Contraindicated in patients on gemfibrozil; antifungal medications including itraconazole, ketoconazole, posaconazole; antibiotics such as erythromycin, clarithromycin, telithromycin; and HIV protease inhibitors such as nefazodone, cyclosporine, and danazol.

Antilipemic Agents (Bile Acid Sequestrants)

Indications: used to manage hypercholesterolemia

Mechanism of action: form a nonabsorbable complex with bile acids in the intestine, inhibiting enterohepatic reuptake of intestinal bile salts, which increases the fecal loss of bile salt–bound LDL cholesterol

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Cholestyramine (Novo-Cholamine, Prevalite, Questran, Questran-Light)	*Powder:* 4-24 g 1-2 times a day *Tablet:* 4-16 g 1-2 times a day	Constipation, heartburn, nausea, flatulence, vomiting, abdominal pain, and headache Instruct patient to mix powder with fluid or applesauce. Do not take dry, avoid inhaling product. Patient should take other medications at least 1 hour before taking this medication. Patient should report any stomach cramping, pain, blood in stool, and unresolved nausea or vomiting. Monitor cholesterol and triglyceride levels before and during therapy. Use during pregnancy must be cautious, weighing benefits of use against the possible risks involved.
Colesevelam (Welchol)	625 mg, 3 tablets BID with meals	Same
Colestipol (Colestid)	*Powder:* 5-30 g mixed with liquid in divided doses *Tablet:* 2 g daily-BID, max 16 g/day	Same
Ezetimibe (Zetia)	*Tablet:* One tablet once a day by mouth with or without food	Headache, dizziness, diarrhea, sore throat, runny nose, sneezing, and joint pain. Monitor for signs of liver failure. Follow low-cholesterol, low-fat diet. Keep a written list of all prescription and nonprescription medicines as well as vitamins, minerals, or other dietary supplements.

Continued

TABLE 12-4 PHARMACOLOGY

Medications for Lowering Cholesterol and Triglycerides—cont'd

Antilipemic Agent (Miscellaneous, Niacin)
Indications: adjunctive treatment of hyperlipidemia
Mechanism of action: inhibits VLDL synthesis

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Nicotinic acid (Niacin)	1.5-2 g daily in 3 divided doses *Sustained release:* 500 mg at bedtime, max 2000 mg/day	Headache, flushing, bloating, flatulence, and nausea Instruct patient to take it as directed and not to exceed recommended dosage. Should be taken after meals. Patient should report persistent gastrointestinal disturbances or changes in color of urine or stool. Take with aspirin to reduce flushing.

Antilipemic Agent (Fibric Acid)
Indications: treatment of hypertriglyceridemia in patients who have not responded to dietary intervention
Mechanism of action: inhibits biosynthesis of VLDL, increases HDL, and decreases triglycerides

Gemfibrozil (Lopid)	600 mg BID PO	Stomach upset, fatigue, headache, diarrhea, and nausea. Instruct patient to take before breakfast and dinner. May take with milk or meals if gastrointestinal upset occurs. Patient should report severe abdominal pain, nausea, or vomiting. Use during pregnancy must be weighed against the possible risks.
Fenofibrate (Tricor)	67-200 mg PO	Nausea, abdominal pain, increased liver enzymes, rash, headaches

BID, Twice daily; *HDL,* high-density lipoprotein; *HMG-CoA,* 3-hydroxy-3-methylglutaryl-coenzyme A; *LDL,* low-density lipoprotein; *PO,* orally; *VLDL,* very-low-density lipoprotein.
From Skidmore-Roth L. (2011). *Mosby's 2011 Nursing Drug Reference.* St. Louis: Mosby.

LDL levels are reassessed in 4 to 6 weeks, and dosages are adjusted as needed.[28] A disadvantage of the medications is that they can cause liver damage; therefore it is important to ensure that the patient has liver enzymes drawn periodically to assess liver function. The medications can also cause myopathies, although rare, and patients are instructed to contact their healthcare provider if they develop any muscle aches.

The bile acid resins combine with cholesterol-containing bile acids in the intestines to form an insoluble complex that is eliminated through feces. These drugs lower LDL levels by 10% to 20%. Bile acid resins include cholestyramine and colestipol. The drugs are mixed in liquid and are taken twice daily. They are associated with side effects such as nausea and flatulence and are contraindicated in biliary obstruction. The drugs interfere with absorption of many medications. It is recommended that other medications be given 1 hour before or 4 hours after administration of the resins to promote absorption.[11]

Ezetimibe (Zetia) works in the digestive tract by blocking the absorption of cholesterol from food. It is often used in conjunction with other cholesterol reducing medications. Ezetimibe can cause liver disease; therefore liver function tests need to be monitored and the drug is contraindicated in severe hepatic disease. Although the drug lowers lipid levels, it has not been proven to reduce heart disease. The medication is also being tested in combination with statins to achieve LDL goals.

Nicotinic acid, or niacin, reduces total cholesterol, LDL, and triglyceride levels, and it increases HDL. The drug is available over-the-counter; however, its use in lowering cholesterol must be under the supervision of a healthcare provider. A long-acting, once-daily dose, is available by prescription. The drug dosage is gradually increased to the maximum effective daily dose. Common side effects include a metallic taste in mouth, flushing, and increased feelings of warmth. Major side effects include hepatic dysfunction, gout, and hyperglycemia. Because nicotinic acid affects the absorption of other drugs, it must be given separately from other medications. Administering niacin with food and aspirin can reduce some of the side effects.[28]

If triglyceride levels are elevated, patients may be prescribed agents that specifically lower triglyceride levels. One agent is gemfibrozil, a fibric acid derivative that lowers triglycerides and increases HDL levels. This drug is associated with many gastrointestinal side effects.

If a patient does not respond adequately to single-drug therapy, combined-drug therapy is considered to lower LDL levels further. For example, statins may be combined with bile acid resins. Patients must be carefully monitored when two or more lipid-lowering agents are given simultaneously.

Medications to prevent platelet adhesion and aggregation. Drugs are often prescribed for the patient with CAD to reduce platelet adhesion and aggregation. A single dose of 81 to 325 mg of an enteric-coated aspirin per day is commonly

prescribed. To prevent platelet aggregation, other agents that may be prescribed with aspirin such as clopidogrel (Plavix) or prasugrel (Effient). In addition, Brilinta (ticagrelor) has just been approved.

Patient Outcomes

Several outcomes are expected after treatment. These include relief of pain; less anxiety related to the disease; adherence to health behavior modification to reduce cardiovascular risks; and the ability to describe the disease process, causes, factors contributing to the symptoms, and the procedures for disease or symptom control.

ANGINA

Angina is chest pain or discomfort caused by myocardial ischemia that occurs from an imbalance between myocardial oxygen supply and demand. CAD and coronary artery spasms are common causes of angina.

Pathophysiology

Angina (from the Latin word meaning *squeezing*) is the chest pain associated with myocardial ischemia; it is transient and does not cause cell death; but it may be a precursor to cell death from MI. The neural pain receptors are stimulated by accelerated metabolism, chemical changes and imbalances, and/or local mechanical stress resulting from abnormal myocardial contractions. The oxygen circulating via the vascular system to the myocardial cells decreases, causing ischemia to the tissue, resulting in pain.

Angina occurs when oxygen demand is higher than oxygen supply. Box 12-5 shows factors influencing oxygen supply and demand that may result in angina.

Types of Angina

Different types of angina exist: stable, unstable, and variant. *Stable angina* occurs with exertion and is relieved by rest. It is sometimes called chronic exertional angina. *Unstable angina* pain is often more severe, may occur at rest, and requires more frequent nitrate therapy. It is sometimes described as crescendo (increasing) in nature. During an unstable episode, the ECG may show ST-segment depression, T-wave inversions, or no changes at all. The patient has an increased risk of MI within 18 months of onset of unstable angina; therefore medical or surgical interventions, or both, are warranted. Patients are often hospitalized for diagnostic workup and treatment. The treatment of unstable angina is discussed more completely in the section, "Acute Coronary Syndrome."

Variant, or *Prinzmetal, angina* is caused by coronary artery spasms. It often occurs at rest and without other precipitating factors. The ECG shows a marked ST elevation (usually seen only in AMI) during the episode. The ST segment returns to normal after the spasm subsides. AMI can occur with prolonged coronary artery spasm, even in the absence of CAD.

Assessment

Assessment of the patient with actual or suspected angina involves continual observation of the patient and monitoring of signs, symptoms, and diagnostic findings. The patient must be monitored for the type and degree of pain (see box, "Clinical Alert: Symptoms of Angina").

BOX 12-5 FACTORS THAT INFLUENCE OXYGEN DEMAND AND SUPPLY

Increased Oxygen Demand

- **Increased heart rate:** exercise, tachydysrhythmias, fever, anxiety, pain, thyrotoxicosis, medications, ingestion of heavy meals, adapting to extremes in temperature
- **Increased preload:** volume overload
- **Increased afterload**: hypertension, aortic stenosis, vasopressors
- **Increased contractility**: exercise, medications, anxiety

Reduced Oxygen Supply

- Coronary artery disease
- Coronary artery spasms
- Anemia
- Hypoxemia

! CLINICAL ALERT

Symptoms of Angina

- Pain is frequently retrosternal, left pectoral, or epigastric. It may radiate to the jaw, left shoulder, or left arm.
- Pain may be associated with dyspnea, light-headedness, or diaphoresis.
- Pain can be described as chest pressure, burning, squeezing, heavy, or smothering.
- Pain usually lasts 1 to 5 minutes.
- Classic placing of clenched fist against the chest (sternum) may be seen, or may be absent if the sensation is confused with indigestion.
- Pain usually begins with exertion and subsides with rest.

The precipitating factors that can be identified as bringing on an episode of anginal pain include physical or emotional stress, exposure to temperature extremes, and ingestion of a heavy meal. It is important to know what factors alleviate the anginal pain, including stopping activity or exercise and taking nitroglycerin sublingual tablets or spray.

Diagnostic Studies

Diagnostic studies for angina include the following: history and physical examination, including assessment of pain and precipitating factors; laboratory data, including blood studies for anemia (hemoglobin and hematocrit values), cardiac enzymes (CK_2-MB, cardiac troponin I, cardiac troponin T levels), and cholesterol and triglyceride levels; ECGs during resting periods; stress testing; and coronary angiography. Complications of untreated or unstable angina include MI,

HF, dysrhythmias, psychological depression, and sudden death.

Nursing Diagnoses

Several nursing diagnoses and interventions are identified for patients with angina. These include the following[11]:

- Acute chest pain related to myocardial ischemia
- Knowledge deficit related to unfamiliarity with disease process and treatment
- Activity intolerance related to chest pain, side effects of prescribed medications, imbalance between oxygen supply and demand

Interventions

Nursing Interventions

Nursing interventions for the patient with angina are aimed at assessing the patient's description of pain, noting exacerbating factors and measures used to relieve the pain; evaluating whether this is a chronic problem (stable angina) or a new presentation; assessing for appropriateness of performing an ECG to evaluate ST-segment and T-wave changes; monitoring vital signs during chest pain and after nitrate administration; and monitoring the effectiveness of interventions.

The patient is instructed to relax and rest at the first sign of pain or discomfort, and to notify the nurse at the onset of any type of chest pain so that nitrates and oxygen can be administered. The nurse also offers assurance and emotional support by explaining all treatments and procedures and by encouraging questions. The nurse begins to assess the patient's knowledge level regarding the causes of angina, diagnostic procedures, the treatment plan, and risk factors for CAD. For those patients that smoke, readiness to quit should be assessed. Smoking cessation is encouraged. Patients who wish to stop smoking can be referred to the American Heart Association, American Lung Association, or American Cancer Society for support groups and interventions.

Medical Interventions

Unstable angina can be treated by conservative management, early intervention with percutaneous intervention, or surgical revascularization. Conservative intervention for the patient experiencing angina includes the administration of nitrates, beta-adrenergic blocking agents, calcium channel blocking agents, and ranolazine (Table 12-5). Angioplasty, stenting, and bypass surgery are approaches to revascularization.

TABLE 12-5 PHARMACOLOGY

Drugs for Acute Coronary Syndromes

Nitrates

Indications: angina

Mechanism of action: directly relaxes smooth muscle, which causes vasodilation of the systemic vascular bed; decrease myocardial oxygen demands; secondary effect is vasodilation of responsive coronary arteries

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Nitroglycerin (Tridil, Nitro-Bid, Nitro-Dur, Nitrostat)	*SL:* 0.4 mg SL every 5 minutes for up to 3 doses *Topical:* 0.5-2 inches every 6 hours *Transdermal:* One patch each day *IV:* continuous infusion started at 5 mcg/min and titrated up to 200 mcg/min maximum	Headache, flushing, tachycardia, dizziness, and orthostatic hypotension Instruct patient to call 911 if chest pain does not subside after the third SL dose. For topical dosing, patient should have a nitrate-free interval (10-12 hours/day) to avoid development of tolerance. Instruct patient not to combine nitrate use with medications used for treatment of erectile dysfunction (e.g., vardenafil, tadalafil, sildenafil).
Isosorbide dinitrate (Isordil)	*PO:* 5-40 mg BID-TID	Same
Isosorbide mononitrate (Imdur)	*PO:* 30-60 mg every day; maximum daily dose 240 mg	Same Do not crush or dissolve.

Beta-Blockers

Indications: used to treat angina, acute myocardial infarction, dysrhythmias and heart failure

Mechanism of action: block beta-adrenergic receptors, which results in decreased sympathetic nervous system response such as decreased heart rate, blood pressure, and cardiac contractility

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Metoprolol (Lopressor, Toprol XL)	*PO:* 50-100 mg BID *PO Toprol XL:* 12.5-200 mg daily *IV:* 5 mg	Bradycardia, hypotension, atrioventricular blocks, asthma attacks, fatigue, impotence, may mask hypoglycemic episodes.

TABLE 12-5 PHARMACOLOGY

Drugs for Acute Coronary Syndromes—cont'd

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
		Teach patient to take pulse and blood pressure on regular basis and not to abruptly stop taking beta-blockers. Close glucose monitoring is needed if diabetic. Patient should have ECG monitoring during IV administration. Monitor for worsening signs of heart failure.
Propranolol (Inderal)	*PO:* 80-320 mg in divided doses 2-4 times a day *IV:* 0.5-3.0 mg IVP at rate of 1 mg/minute	Same
Labetalol (Trandate, Normodyne)	*PO:* 200-400 mg BID *IV:* 2 mg over 2 minutes at 10-minute intervals; slow IVP	Same Acts on both alpha, beta$_1$, and beta$_2$ receptors During IV administration, monitor blood pressure continuously; maximum effect occurs within 5 minutes.
Carvedilol (Coreg)	*PO:* 3.125-50 mg BID	Same; take with meals. Dose is doubled every 2 weeks until desired effect.

Calcium Channel Blockers

Indications: used to treat hypertension, tachydysrhythmias, vasospasms, and angina

Mechanism of action: inhibit the flow of calcium ions across cellular membranes, with resulting increased coronary blood flow and myocardial perfusion and decreased myocardial oxygen requirements

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Verapamil (Calan, Isoptin)	*PO:* 80-120 mg TID	Dizziness, flushing, headaches, bradycardia, atrioventricular blocks, and hypotension. Teach patient to monitor pulse and blood pressure, especially if taking nitrates and/or beta-blockers. Tablets cannot be crushed or chewed. Instruct patient to make position changes slowly.
Nifedipine (Procardia)	*PO:* 10 mg TID *PO sustained release:* 30-60 mg daily	Same Important that short-acting formulation (capsule) is swallowed whole and not punctured or chewed.
Diltiazem (Cardizem, Cardizem CD)	*PO:* 30 mg QID *PO sustained release:* 120-360 mg daily	Same

Antiplatelet Agents

Indications: unstable angina, acute myocardial infarction, and coronary interventions

Mechanism of action: inhibit clotting mechanisms within the clotting cascade or prevent platelet aggregation

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Aspirin	*PO:* 81-325 mg daily	Bleeding, epigastric discomfort, bruising, and gastric ulceration Instruct patient to take medication with food. Do not crush or chew the enteric-coated forms. Instruct patient to be aware of additive effects with OTC drugs containing aspirin or salicylate or other NSAIDs.
Clopidogrel (Plavix)	*PO:* 300-mg loading dose, then 75 mg daily (in combination with aspirin)	Same
Prasugrel (Effient)	*PO:* 60-mg loading dose, then 10 mg daily	Anemia, edema, headaches, dizziness. Not recommended for patients >75 years of age, weighing <60 kg, or with history of TIA.
Ticagrelor (Brilinta)	*PO:* 180-mg (two 90-mg tablets) loading dose (in combination with 325 mg aspirin), then 90 mg daily (with 81 mg aspirin)	Contraindicated in patients with history of intracranial hemorrhage, active pathological bleeding, or severe hepatic impairment. Higher incidence of bradydysrhythmias noted in clinical studies. Comparison of ticagrelor vs. clopidogrel reported a higher incidence of bleeding (11.6% vs 11.2%) and dyspnea (14% vs 8%).

Continued

TABLE 12-5 PHARMACOLOGY

Drugs for Acute Coronary Syndromes—cont'd

Glycoprotein IIb/IIIa Inhibitors

Indications: acute coronary syndromes and coronary intervention patients

Mechanism of action: antiplatelet agent and glycoprotein IIb/IIIa inhibitor; act by binding to the glycoprotein IIb/IIIa receptor site on the surface of the platelet

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Abciximab (ReoPro)	*IV:* 0.25 mg/kg IV bolus followed by a continuous infusion at 0.125 mg/kg/min for 12 hours	Bleeding, bruising, hemorrhage, thrombocytopenia, and hypotension. Avoid IM injections and venipunctures. Observe and teach patient bleeding precautions and activities to avoid that may cause injury. Assess infusion insertion site for bleeding or hematoma formation. Assess puncture site used for coronary intervention frequently. Abciximab is not reversible because of its binding to platelets. For hemorrhage, give fresh frozen plasma and platelets. Monitor CBC and aPTT daily.
Tirofiban (Aggrastat)	*IV:* 0.4 mcg/kg/min for 30 minutes, then continued at 0.1 mcg/kg/min for 12-24 hours Reduce loading and maintenance infusion by 50% in patients with impaired renal function (creatinine clearance <30 mL/min)	Same Tirofiban stops working when the infusion is discontinued. Platelet function is restored 4 hours after stopping the infusion.
Eptifibatide (Integrilin)	*IV:* 180 mcg/kg loading dose over 2 minutes, followed by continuous infusion of 2 mcg/kg/min for 18-24 hours or until hospital discharge. Reduce maintenance dose by 50% (to 1 mcg/kg/minute) in patients with creatinine clearance <50 mL/min; contraindicated if creatinine clearance <20 mL/min Concurrent aspirin, thienopyridine, and heparin therapy is recommended	Same Eptifibatide stops working when the infusion is discontinued. Platelet function is restored 4 hours after stopping the infusion.

Antithrombin Agents

Indications: prevention of or delay in thrombus formation

Mechanism of action: enhances inhibitory effects of antithrombin III, preventing conversion of fibrinogen to fibrin and prothrombin to thrombin

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Heparin	*IV:* 5000-7000 units bolus, followed by infusion of 1000 units/hr, titrated to aPTT	Bleeding, bruising, thrombocytopenia. Monitor aPTT. Monitor for signs of bleeding and hematoma formation. Avoid IM injections. Do not rub the site after giving the injection. Increased bleeding risk when dosing unfractioned heparin in patients with renal insufficiency. The aPTT should be monitored aggressively and the patient should be evaluated closely for signs and symptoms of bleeding.
Enoxaparin (Lovenox)	*Subcutaneous:* 1 mg/kg every 12 hours, in conjunction with aspirin For patients with creatinine clearances <30 mL/minute, dosage is 1 mg/kg every 24 hours.	Bleeding, bruising, local site hematomas, and hemorrhage. Instruct patient to report persistent chest pain, unusual bleeding or bruising. Do not rub the site after giving the injection.

TABLE 12-5 PHARMACOLOGY

Drugs for Acute Coronary Syndromes—cont'd

Analgesic

Indications: pain relief and anxiety reduction during acute myocardial infarction

Mechanism of action: binds to opioid receptors in the central nervous system and causes inhibition of ascending pain pathways, altering perception and response to pain

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Morphine	*IV:* 2-4 mg IVP every 5-10 minutes	Hypotension, respiratory depression, apnea, bradycardia, nausea, and restlessness. Titrated for chest pain. Monitor level of consciousness, blood pressure, respiratory rate, and oxygen saturation during therapy. Effects are reversed with naloxone (Narcan).

Angiotensin-Converting Enzyme Inhibitors

Indications: used to treat hypertension, heart failure, and patients after myocardial infarction

Mechanism of action: prevent the conversion of angiotensin I to angiotensin II resulting in lower levels of angiotensin II, which causes an increase in plasma renin activity and a reduction of aldosterone secretion; also inhibit the remodeling process after myocardial injury

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Enalapril (Vasotec)	*PO:* 2.5-40 mg BID	Hypotension, bradycardia, renal impairment, cough, and orthostatic hypotension. Monitor urine output. Monitor potassium levels. Avoid use of NSAIDs. Instruct patient to avoid rapid change in position such as from lying to standing. Contraindicated in pregnancy.
Fosinopril (Monopril)	*PO:* 20-40 mg daily or BID	Same
Captopril (Capoten)	*PO:* 6.25 mg TID increasing gradually to 100 mg TID	Same

aPTT, Activated partial thromboplastin time; *BID,* twice daily; *CBC,* complete blood count; *ECG,* electrocardiogram; *IVP,* intravenous push; *NSAIDs,* nonsteroidal antiinflammatory drugs; *OTC,* over-the-counter; *PO,* orally; *QID,* four times daily; *SL,* sublingual; *TID,* three times daily.
From Skidmore-Roth L. (2011). *Mosby's 2011 Nursing Drug Reference.* St. Louis: Mosby.

Nitrates are the most common medications for angina. They are direct-acting smooth muscle relaxants that cause vasodilation of the peripheral or systemic vascular bed.[28] Nitrate therapy is beneficial because it decreases myocardial oxygen demand. The vasodilating effect causes relief of pain and lowering of blood pressure. Nitroglycerin is available in quick-acting forms such as sublingual tablets or spray, or intravenous infusion. Long-acting forms are delivered orally or by ointments and skin patches (transdermal). Oral isosorbide (Isordil, Ismo, Imdur) is another vasodilator. Side effects of these vasodilators include headache, flushing, tachycardia, dizziness, and orthostatic hypotension. Instructions for NTG therapy are detailed in Box 12-6.

Beta-adrenergic blocking agents may also be used to treat angina. They block adrenergic receptors, thereby decreasing heart rate, blood pressure, and cardiac contractility.[28] Examples include atenolol, metoprolol, propranolol, labetalol, carvedilol, nadolol, timolol, and pindolol. The side effects of these agents include bradycardia, AV block, asthma attacks, depression, erectile dysfunction, hypotension, memory loss, and masking of hypoglycemic episodes. The patient is taught to take these agents as prescribed, not to stop taking them abruptly, and to monitor heart rate and blood pressure at regular intervals.

Calcium channel blockers inhibit the flow of calcium ions across cellular membranes, an effect that causes direct increases in coronary blood flow and myocardial perfusion.[28] These drugs are used for treating tachydysrhythmias, vasospasms, and hypertension, as well as for treating angina. Calcium channel blockers are divided into two categories: dihydropyridines and non-dihydropyridines. Dihydropyridines are primarily used to treat hypertension. These drugs typically end in "pine," such as amlodipine,

BOX 12-6 INSTRUCTIONS REGARDING NITROGLYCERIN

If the client is discharged on sublingual or buccal nitroglycerin, instruct client to:

- Have tablets readily available.
- Take a tablet before strenuous activity and in stressful situations.
- Take one tablet when chest pain occurs and another every 5 minutes up to a total of three times if necessary; obtain emergency medical assistance if pain persists.
- Place the tablet under the tongue or in the buccal pouch and allow it to dissolve thoroughly.
- Store tablets in a tightly capped, original container away from heat and moisture.
- Replace tablets every 6 months or sooner if they do not relieve discomfort.
- Avoid rising to a standing position quickly after taking nitroglycerin.
- Recognize that dizziness, flushing, and mild headache are common side effects.
- Report fainting, persistent or severe headache, blurred vision, or dry mouth.
- Avoid drinking alcoholic beverages.
- Caution use of drugs for erectile dysfunction (e.g., Viagra, Levitra) when taking nitrates because hypotensive effects are exaggerated.

If nitroglycerin skin patches are prescribed:

- Provide instructions about correct application, skin care, the need to rotate sites and to remove the old patch, and frequency of change.
- The patch should only be worn 12 to 14 hours per day to prevent development of nitrate tolerance.

From Skidmore-Roth, L. (2011). *Mosby's 2011 Nursing Drug Reference*. St. Louis: Mosby.

felodipine, nifedipine, and isradipine. Non-dihydropyridines such as verapamil (Calan, Isoptin) and diltiazem (Cardizem) are more effective for treating angina and dysrhythmias. The side effects of calcium channel blockers include dizziness, flushing, headaches, decreased heart rate, and hypotension. Ankle edema is a major side effect with the dihydropyridine-type calcium channel blocker. When prescribed a calcium channel blocker, the patient is taught to monitor blood pressure for hypotension and heart rate for bradycardia, especially if the agents are taken in combination with nitrates and beta-blockers.

Outcomes

The outcomes for patients with angina are that they will verbalize relief of chest discomfort, appear relaxed and comfortable, verbalize an understanding of angina pectoris and its management, describe their own cardiac risk factors and strategies to reduce them, and perform activities within limits of their ischemic disease, as evidenced by absence of chest pain or discomfort and no ECG changes reflecting ischemia.[11]

ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) includes the diagnoses of unstable angina (UA) (previously defined) as well as acute myocardial infarction (AMI). AMI is defined as non–ST segment elevation myocardial infarction (NSTEMI) or ST segment elevation myocardial infarction (STEMI) by ECG characteristics. Prompt recognition and treatment results in improved outcomes for all ACSs.[24]

Pathophysiology

AMI is caused by an imbalance between myocardial oxygen supply and demand. This imbalance is the result of decreased coronary artery perfusion. Most cases of AMI are secondary to atherosclerosis. Other causes (<5%) include coronary artery spasm, coronary embolism, and blunt trauma. Reduced blood flow to an area of the myocardium causes significant and sustained oxygen deprivation to myocardial cells. Normal functioning is disrupted as ischemia and injury lead to eventual cellular death. Myocardial dysfunction occurs as more cells become involved.

Prolonged ischemia from cessation of blood flow to the cardiac muscle results in infarction and evolves over time. Cardiac cells can withstand ischemic conditions for 20 minutes; after that period, irreversible myocardial cell damage and cellular death begins. The amount of cell death increases, extending from the endocardium to the epicardium, as the duration of the occlusion increases. The extent of cell death determines the size of the MI. Contractility in the infarcted area becomes impaired. A nonfunctional zone and a zone of mild ischemia with potentially viable tissue surround the infarct. The ultimate size of the infarct depends on the fate of this ischemic zone. Early interventions, such as the administration of thrombolytics, can restore perfusion to the ischemic zone and can reduce the area of myocardial damage.

Based on the ECG, AMI is classified as a STEMI or NSTEMI. STEMI usually occurs because of plaque rupture leading to complete occlusion of the artery. NSTEMI usually results from a partially occluded coronary vessel. Most infarcts occur in the left ventricle; however, right ventricular infarction commonly occurs in patients with inferior wall infarction.[4] The treatment for a RV infarct is usually fluid therapy. Patients with right ventricular infarctions require cardiac pacing more frequently than left ventricular infarcts secondary to conduction defects, which are common.

The severity of the MI is determined by the success or lack of success of the treatment and by the degree of collateral circulation present at that particular part of the heart muscle. The *collateral circulation* consists of the alternative routes, or channels, that can develop in the myocardium in response to chronic ischemia or regional hypoperfusion. Through this small network of extra vessels, blood flow can be improved to the threatened myocardium.

TABLE 12-6 MYOCARDIAL INFARCTION BY SITE, ELECTROCARDIOGRAPHIC CHANGES, AND COMPLICATIONS

LOCATION OF MI	PRIMARY SITE OF OCCLUSION	PRIMARY ECG CHANGES	COMPLICATIONS
Inferior MI	RCA (80%-90%) LCX (10%-20%)	II, III, aV_F	First- and second-degree heart block, right ventricular infarction
Inferolateral MI	LCX	II, III, aV_F, V_5, V_6	Third-degree heart block, left HF, cardiomyopathy, left ventricular rupture
Posterior MI	RCA or LCX	No lead truly looks at posterior surface Look for reciprocal changes in V_1 and V_2—tall, broad R waves; ST depression and tall T waves Posterior leads V_7, V_8, and V_9 may be recorded and evaluated	First-, second-, and third-degree heart blocks, HF, bradydysrhythmias
Anterior MI	LAD	V_2-V_4	Third-degree heart block, HF, left bundle branch block
Anterior-septal MI	LAD	V_1-V_3	Second- and third-degree heart block
Lateral MI	LAD or LCX	V_5, V_6, I, aVL	HF
Right ventricular	RCA	V_4R Right precordial leads V_1R-V_6R may be recorded and evaluated	Increased RAP, decreased cardiac output, bradydysrhythmias, heart blocks, hypotension, cardiogenic shock

AV, Atrioventricular; *ECG,* electrocardiographic; *HF,* heart failure; *LAD,* left anterior descending; *LCX,* left circumflex artery; *MI,* myocardial infarction; *RAP,* right atrial pressure; *RCA,* right coronary artery.

Assessment

Patient Assessment

Patient assessment includes close observation to identify the classic signs and symptoms of AMI. Chest pain is the paramount symptom. It may be severe, crushing, tight, squeezing, or simply a feeling of pressure. It can be precordial, substernal, or in the back, radiating to the arms, neck, or jaw. The skin may be cool, clammy, pale, and diaphoretic; the patient's color may be dusky or ashen; and slight hyperthermia may be present. The patient may be dyspneic and tachypneic, and may feel faint or have intermittent loss of sensorium. Nausea and vomiting commonly occur. Hypotension may be present and is often associated with dysrhythmias, particularly ventricular ectopy, bradycardia, tachycardia, or heart block. The type of dysrhythmia present depends on the area of the MI. The patient may be anxious or restless, or may exhibit certain behavioral responses including denial, depression, and a sense of impending doom. *Women are more likely to have atypical signs and symptoms* such as fatigue, diaphoresis, indigestion, arm or shoulder pain, nausea, and vomiting.[23]

Some individuals have ischemic episodes without knowing it, thereby having a *silent* infarction. These occur with no presenting signs or symptoms. This is more common in diabetic patients secondary to neuropathy. Assessment of a patient experiencing an AMI takes all the foregoing signs and symptoms into account during the history and physical examination. Risk factors for an AMI are also considered.

Diagnosis

Diagnosis of AMI is based on symptoms, analysis of a 12-lead ECG, and cardiac enzyme values. The ECG is inspected for ST-segment elevation (>1 mm) in two or more contiguous leads. ST-segment depression (≥0.5 mm) and new onset left bundle branch block also suggest an AMI. The type of AMI can be determined by the particular coronary artery involved and the blood supply to that area (Table 12-6).

Elevated serum cardiac enzymes are used to confirm the diagnosis of AMI: total CK, CK_2-MB, troponins I and T, and myoglobin. These tests are ordered immediately when a diagnosis of AMI is suspected and periodically (usually every 6-8 hours) during the first 24 hours to assess for increasing levels. Emergency cardiac catheterization may be performed in institutions with interventional cardiology services. Criteria for the diagnosis of ACS are summarized in Table 12-7.

Nursing Diagnoses

Nursing diagnoses and collaborative problems for the patient with AMI are described in the "Nursing Care Plan for the Patient with Acute Myocardial Infarction."

TABLE 12-7 CRITERIA FOR DIAGNOSIS OF ACUTE CORONARY SYNDROME

MAJOR CRITERIA	MINOR CRITERIA	
A diagnosis of an ACS can be made if one or more of the following major criteria are present:	In the absence of a major criterion, a diagnosis of ACS requires the presence of at least one item from both columns I and II	
	I	II
• ST-elevation or left bundle branch block (LBBB) in the setting of recent (<24 hours) or ongoing angina • New, or presumably new, ST-segment depression (≥0.05 mV) or T-wave inversion (≥0.2 mV) with rest symptoms • Elevated serum markers of myocardial damage (i.e., troponin I, troponin T, and CK_2-MB)	• Prolonged (i.e., >20 minutes) chest, arm/shoulder, neck, or epigastric discomfort • New onset chest, arm/shoulder, neck, or epigastric discomfort at rest, minimal exertion or ordinary activity • Previously documented chest, arm/shoulder, neck, or epigastric discomfort which has become distinctly more frequent or longer in duration	• Typical or atypical angina • Male age >40 years or female age >60 years • Known CAD • Heart failure, hypotension, or transient mitral regurgitation by examination • Diabetes • Documented extracardiac vascular disease • Pathological Q-waves on ECG • Abnormal ST-segment or T-wave abnormalities not known to be new

ACS, Acute coronary syndrome; *CAD,* coronary artery disease; *CK_2-MB,* creatine kinase MB band; *ECG,* electrocardiogram.
From Veterans Health Administration, Department of Defense. (2003). *VA/DoD clinical practice guideline for management of ischemic heart disease*. Washington, D.C.: Veterans Health Administration, Department of Defense.

NURSING CARE PLAN

for the Patient with Acute Myocardial Infarction

NURSING DIAGNOSIS

Acute Chest Pain related to myocardial infarction, ischemia, or reduced coronary artery blood flow

PATIENT OUTCOMES

Chest pain relieved

- Verbalizes relief of pain
- Appears comfortable

NURSING INTERVENTIONS	RATIONALES

• Assess for characteristics of AMI pain:
  • Occurs suddenly
  • More intense
  • Quality varies
  • Not relieved with rest or nitrates
  • Atypical symptoms in older patients, women, and patients with diabetes and heart failure
	• Assist in identification of AMI to provide early treatment

• Note time since onset of first episode of chest pain; if less than 6 hours, patient may be a candidate for thrombolytic therapy	• Provide timely intervention
• Assess prior treatments for pain; patient may have taken sublingual nitroglycerin and a single dose of aspirin before arriving to hospital	• Assess response to prior treatment; assess need for aspirin as part of treatment protocol
• Monitor heart rate and blood pressure during pain episodes and during medication administration	• Assess nonverbal indicators of pain and response to treatment
• Assess baseline ECG for signs of MI: T and ST changes and development of Q waves and compare to previous ECGs if available	• Identify ischemia, injury, evolving AMI
• Monitor serial cardiac enzymes	• Assist in diagnosis and confirmation of AMI
• Continually reassess chest pain and response to medication; ongoing pain signifies prolonged myocardial ischemia and warrants immediate intervention	• Assess response to treatment; ensure that pain is controlled
• Assess for contraindications to thrombolytic agents; absolute contraindications include active internal bleeding, bleeding diathesis, or history of hemorrhagic stroke or intracranial hemorrhage	• Ensure that medication is administered safely when warranted; prevent complications associated with the medication

NURSING CARE PLAN

for the Patient with Acute Myocardial Infarction—cont'd

NURSING INTERVENTIONS	RATIONALES
• Assess for relative contraindications or warning conditions	• Risks of thrombolytic agents are weighed against benefits
• Maintain bed rest during periods of pain	• Reduce oxygen demand of the heart
• Administer oxygen therapy at 4-6 L/min; maintain oxygen saturation above 90%	• Ensure adequate oxygenation to the myocardium to prevent further damage
• Initiate IV nitrates according to protocol	• Nitrates are both coronary dilators and peripheral vasodilators causing hypotension
• Administer morphine sulfate according to unit protocol	• Reduce the workload on the heart through venodilation
• Administer IV beta-blockers according to protocol	• Reduce mortality in acute-phase MI
• Administer oral aspirin	• Decrease platelet aggregation to improve mortality
• Administer angiotensin-converting enzyme inhibitors	• Reduce progression to heart failure and death in patients with large MIs with LV dysfunction and in diabetic patients having an MI
• Administer thrombolytic agents according to unit protocol	• Restore perfusion
• Monitor for signs of bleeding: puncture sites, gingival bleeding, and prior cuts; observe for presence of occult or frank blood in urine, stool emesis, and sputum	• Assess for complications of thrombolytic therapy so that treatment can be initiated as needed
• Assess for intracranial bleeding by frequent monitoring of neurological status; changes in mental status, visual disturbances, and headaches are frequent signs of intracranial bleeding	• Assess for complication of intracranial bleeding associated with thrombolytic therapy
• Administer IV heparin according to unit protocol adjusting aPTT dose to 1.5 to 2 times normal	• Maintain coronary artery vessel patency after thrombolysis
• Prepare for possible cardiac catheterization, percutaneous transluminal coronary angioplasty or stent or coronary artery bypass graft surgery if signs of reperfusion are not evident and infarction evolves or if primary angioplasty is indicated	• Facilitate rapid intervention to restore coronary artery perfusion

NURSING DIAGNOSIS

Decreased Cardiac Output related to dysrhythmias, sympathetic nervous system stimulation, or electrolyte imbalances

PATIENT OUTCOMES

Normal cardiac rhythm with adequate cardiac output

- Strong peripheral pulses
- Normal blood pressure
- Clear breath sounds
- Good capillary refill
- Adequate urine outputs
- Clear mentation

NURSING INTERVENTIONS	RATIONALES
• Monitor heart rate and rhythm continuously; anticipate common dysrhythmias—PVCs, ventricular tachycardia, atrial flutter, and atrial fibrillation	• Detect and treat dysrhythmias
• Assess for signs of decreased cardiac output	• Identify complications to ensure timely treatment
• Monitor PR, QRS, and QT intervals	• Detect abnormal conduction of impulses early
• Monitor continuous ECG in appropriate leads	• See Chapter 7 for best practice for monitoring
• Institute treatment according to advanced cardiac life support (ACLS) guidelines or unit protocol	• Surveillance may prevent lethal dysrhythmias
• Assess peripheral and central pulses	• Detect reduced stroke volume and cardiac output
• Assess for mental status changes—restlessness and anxiety	• Detect early signs of hypoxemia
• Assess respiratory rate, rhythm, and breath sounds; rapid, shallow respirations and presence of crackles, wheezes or Cheyne-Stokes respirations	• Assess respiratory symptoms associated with low cardiac output
• Assess urine output via indwelling urinary catheter	• Assess adequacy of renal perfusion
• Auscultate for presence of S_3, S_4, or systolic murmur	• S_3 denotes LV dysfunction; S_4 indicates a noncompliant ventricle; a systolic murmur may be caused by papillary muscle rupture

Continued

NURSING CARE PLAN

for the Patient with Acute Myocardial Infarction—cont'd

NURSING INTERVENTIONS	RATIONALES
• Assess pulse oximetry and arterial blood gas results; maintain oxygen saturation of at least 90%	• Ensure adequate oxygenation
• If inferior wall MI, evaluate right-sided 12-lead ECG; assess for signs of a right ventricular MI and right ventricular failure (see Box 12-12)	• Identify right ventricular MI and potential complication of right-sided heart failure
• Anticipate insertion of hemodynamic monitoring catheter	• Guide management of fluids and medications
• Administer IV fluids	• Maintain fluid balance
• Monitor for signs of left and right ventricular failure (Box 12-12)	• For left-sided failure anticipate diuretics, vasodilators, inotropics, and oxygen as indicated • For right-sided failure anticipate fluid resuscitation and possible inotropic and peripheral vasodilator therapy
• Carefully administer nitrates and morphine sulfate for pain	• Reduced preload and filling pressures associated with these medication may compromise cardiac output

NURSING DIAGNOSIS

Fear related to change in health status, threat of death, threat to self-concept, critical care environment

PATIENT OUTCOMES

Fear is decreased or resolved

- Verbalizes reduced fear
- Demonstrates positive coping mechanisms

NURSING INTERVENTIONS	RATIONALES
• Assess level of fear noting nonverbal communication	• Identify fear and anxiety
• Assess coping factors	• Coping patterns are highly individualized
• Acknowledge awareness of patient's fears	• Validate feelings and communicate acceptance of those feelings
• Allow patient to verbalize fears of dying	• May reduce anxiety
• Offer realistic assurances of recovery	• Reduce anxiety by providing accurate information
• Maintain confident, assured manner	• Staff anxiety may be perceived by the patient
• Explain care provided and rationale so it is understandable	• Allay anxiety; lack of understanding can add to fear
• Assure the patient that monitoring will ensure prompt intervention	• Provide a measure of safety
• Reduce unnecessary external stimuli	• Anxiety may escalate with excessive noise
• Provide diversional materials	• Decrease anxiety and prevent feelings of isolation
• Establish rest periods	• Ensure dedicate periods to facilitate physical and mental rest
• Refer to other support persons as appropriate	• Additional specialty expertise may be required
• Administer mild sedative as prescribed	• Medication may be required to reduce anxiety

NURSING DIAGNOSIS

Risk for Activity Intolerance related to weakness or imbalance between oxygen supply and demand

PATIENT OUTCOMES

Tolerates progressive activity

- Heart rate and blood pressure within expected range and no complaints of dyspnea or fatigue
- Verbalizes realistic expectations for progressive activity

NURSING INTERVENTIONS	RATIONALES
• Assess respiratory and cardiac status before initiating activity	• Physical deconditioning may occur with prolonged bed rest
• Observe and document response to activity	• Assess response to activity progression
• Encourage adequate rest	• Provide time for energy conservation and recovery
• Provide small, frequent meals	• Facilitate digestion and reduce energy needs
• Instruct patient not to hold breath while exercising or moving about in bed and not to strain for bowel movements	• Valsalva maneuver affects endocardial repolarization
• Maintain progression of activity per cardiac rehabilitation protocol	• Provide gradual increase in activity as tolerated
• Provide emotional support when increasing activity	• Reduce anxiety about overexertion

AMI, Acute myocardial infarction; *aPTT,* activated partial thromboplastin time; *ECG,* electrocardiogram; *IV,* intravenous; *LV,* left ventricular; *PVCs,* premature ventricular contractions; *RAP,* right atrial pressure.

Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes*, 7th ed. St. Louis, Mosby, 2011.

Complications

Complications of AMI include cardiac dysrhythmias, heart failure, thromboembolism, rupture of a portion of the heart (e.g., ventricular wall, interventricular septum, or papillary muscle), pericarditis, infarct extension or recurrence, and cardiogenic shock (see Chapter 11). Dysrhythmias, heart failure, and pericarditis are discussed later in this chapter.

Medical Interventions

Treatment goals for AMI are to establish reperfusion, reduce infarct size, prevent and treat complications, and provide emotional support and education.[1] Medical treatment of AMI is aimed at relieving pain, providing adequate oxygenation to the myocardium, preventing platelet aggregation, and restoring blood flow to the myocardium through thrombolytic therapy or acute interventional therapy, such as angioplasty. Hemodynamic monitoring is also used to assess cardiac function and to monitor fluid balance in some patients.

Pain Relief

The initial pain of AMI is treated with morphine sulfate administered by the IV route. The dose is 2 to 4 mg IV push over 5 minutes. Patients must be observed for hypotension and respiratory depression (see Table 12-5).

Nitrates. Nitroglycerin (NTG) may be given to reduce the ischemic pain of AMI. NTG increases coronary perfusion because of its vasodilatory effects. It is usually started at doses of 5 to 10 mcg/min IV and titrated to a total dose of 50 to 200 mcg/min until chest pain is absent, pulmonary artery occlusion pressure decreases, and/or systolic blood pressure decreases. Caution should be used in administering NTG to patients with inferior or right ventricular infarctions because it can lead to profound hypotension.

Oxygen

Oxygen administration is important for assisting the myocardial tissue to continue its pumping activity and for repairing the damaged tissue around the site of the infarct. Treatment with oxygen via nasal cannula at 4 to 6 L/min assists in maintaining oxygenation. Rest also helps to improve oxygenation. The goal is to maintain oxygen saturation above 90%. However, recent guidelines suggest that routine use of supplemental oxygen may not be necessary in patients with uncomplicated ACS without signs of heart failure or hypoxemia.[24]

Antidysrhythmics

Dysrhythmias are common after AMI. Drugs to treat cardiac dysrhythmias are administered when the heart's natural pacemaker develops an abnormal rate or rhythm (see Chapter 7).

Prevention of Platelet Aggregation

Alterations in platelet function contribute to occlusion of the coronary arteries. Aspirin (325 mg) is given immediately to all patients with suspected AMI. Aspirin blocks synthesis of thromboxane A_2, thus inhibiting aggregation of platelets. In addition, a thienopyridine, such as clopidogrel (Plavix), prasugel (Effient), or ticagrelor (Brilinta); or a Gp IIb/IIIa inhibitor may added.[33] Heparin is used with other antiplatelet agents.

Thrombolytic Therapy

One common treatment for STEMI is thrombolytic therapy. Research has shown that occlusion of the coronary vessel does not cause immediate myocardial cell death. Ischemia begins within minutes of the vessel occlusion, and prolonged injury results in AMI.[26] The goals are to dissolve the lesion that is occluding the coronary artery and to increase blood flow to the myocardium. For treatment to be considered, the patient must be symptomatic for less than 6 hours, have pain for 20 minutes that was unrelieved by NTG, and have a 12-lead ECG with an ST-segment elevation of 1 mm or greater in two or more contiguous ECG leads or an ST-segment depression of 0.5 mm or greater. Table 12-8 lists some of the common thrombolytics currently available.

A summary of the use of thrombolytics includes the following:

- Fibrinolysis reduces mortality and salvages myocardium in STEMI.
- Fibrinolysis is not effective in the treatment of unstable angina or NSTEMI.
- Thrombolysis should be instituted within 30 to 60 minutes of arrival. The sooner treatment is initiated, the better the outcome.
- Patients treated within the first 70 minutes of onset of symptoms have 75% reduction in mortality rates and greater than 50% reduction in infarct size.
- The worst possible complication of fibrinolysis is intracranial hemorrhage. Bleeding from puncture sites commonly occurs.

Nursing care of the patient includes rapid identification of whether the patient is a suitable candidate for IV thrombolytics, thus ensuring as little delay as possible before the therapy; and screening for contraindications. Next, the nurse secures three vascular access lines and obtains necessary laboratory data. Initial ECG monitoring is documented before starting the infusion, at various times throughout the infusion, and at the end of the infusion. Finally, the patient is monitored for complications, including reperfusion dysrhythmias (premature ventricular contractions, sinus bradycardia, accelerated idioventricular rhythm, or ventricular tachycardia), oozing at venipuncture sites, gingival bleeding, reocclusion or reinfarction, and symptoms of hemorrhagic stroke.

TABLE 12-8 PHARMACOLOGY

Thrombolytics

MEDICATION	DOSE/ROUTE	HALF-LIFE
Alteplase (tissue plasminogen activator; t-PA)	*3-hour infusion:* For adults weighing >65 kg, 100-mg dose; administer 60 mg over the first hour (6-10 mg as IV bolus over 1-2 minutes followed by infusion of remaining dose), 20 mg over second hour, and 20 over third hour For adults weighing <65 kg, 1.25 mg/kg dose; administer 60% first hour (6%-10% as a IV bolus followed by infusion of remaining dose), 20% over second hour, and 20% over third *Accelerated 90-minute infusion:* For adults weighing >67 kg, 100-mg dose; administer 15-mg bolus IV over 1-2 minutes, followed by infusion of 50 mg over the first 30 minutes, and 35 mg over next 60 minutes For adults weighing ≤67 kg, administer 15-mg bolus IV over 1-2 minutes, followed by infusion of 0.75 mg/kg over the first 30 minutes (not to exceed 50 mg), and 0.50 mg/kg over next 60 minutes (not to exceed 35 mg)	4-5 minutes
Reteplase (r-PA)	10 units IV bolus; repeat 10-unit dose in 30 minutes; administer over 2 minutes. Give through a dedicated IV line Do not give repeat bolus if serious bleeding occurs after first IV bolus	13-16 minutes
Tenecteplase (TNK)	Total dose 30 to 50 mg, based on weight (see package insert) given IV over 5 seconds	20-24 minutes
Streptokinase (SK)	1.5 million units IV infusion over 60 minutes	23 minutes

IV, Intravenous.
From Skidmore-Roth L. (2011). *Mosby's 2011 Nursing Drug Reference*. St. Louis: Mosby.

Percutaneous Coronary Intervention

Primary percutaneous coronary intervention (PCI) is performed in the management of AMI with improved outcomes over thrombolytic therapy. PCI should be performed within 90 minutes of arrival to the emergency department, with a target of less than 60 minutes (termed *door to balloon* time).[16] Primary PCI has been demonstrated to be more effective than thrombolysis in opening acutely occluded arteries in settings where it can be rapidly performed by experienced interventional cardiologists.[16] If patients present to a facility without PCI capabilities, they should be transferred to a PCI-capable facility to receive a PCI within 90 minutes of being assessed, or triaged to receive fibrinolytic therapy at the receiving facility.

Facilitated Percutaneous Coronary Intervention

Facilitated PCI is the use of additional agents, fibrinolysis, Gp IIb/IIIa inhibitors, or both to pretreat the patient awaiting primary PCI. It was thought that facilitated PCI would improve outcomes; however, administration of these agents before PCI is associated with higher rates of death, reinfarction, and bleeding complications. Therefore facilitated PCI is not recommended; PCI is the preferred treatment.

Additional research is needed to test if fibrinolytic therapy is preferable to delayed PCI in facilities without an interventional cardiology service. American College of Cardiology guidelines recommend treating the affected vessel when feasible and deferring surgical or PCI-based revascularization of other vessels until the patient's condition has stabilized and the most appropriate treatment strategy has been determined.[16]

Medications

Several medications may be ordered for the patient with AMI. Patients whose chest pain symptoms are suggestive of serious illness need immediate assessment in a monitored unit and early therapy to include an IV line, oxygen, aspirin, NTG, and morphine. Early therapy consists of aspirin, heparin or low–molecular weight heparin, nitrates, beta-blockers, clopidogrel, bivalirudin, and fondaparinux.

Nitrates. Nitrates are vasodilators that reduce pain, increase venous capacitance, and reduce platelet adhesion and aggregation. Sublingual NTG is often given in the emergency department. IV NTG is effective for relieving ischemia (see Table 12-5).

Beta-blockers. Beta-blockers are used to decrease heart rate, blood pressure, and myocardial oxygen consumption. Morbidity and mortality after AMI have been reduced by the use of beta-blockers. Commonly used drugs include metoprolol, atenolol, and carvedilol. The patient is carefully assessed for hypotension and bradycardia. Beta-blockers should be started within 24 hours of AMI unless otherwise contraindicated.

Angiotensin-converting enzyme inhibitors. After an AMI, the area of ventricular damage changes shape or *remodels*. The ventricle becomes thinner and balloons out, thus reducing contractility. Cardiac tissue surrounding the area of infarction undergoes changes that can be categorized as (1) myocardial stunning (a temporary loss of contractile function that persists for hours to days after perfusion has been restored); (2) hibernating myocardium (tissue that is persistently ischemic and undergoes metabolic adaptation to

prolong myocyte survival until perfusion can be restored); and (3) myocardial remodelling (a process mediated by angiotensin II, aldosterone, catecholamines, adenosine, and inflammatory cytokines that causes myocyte hypertrophy and loss of contractile function in the areas of the heart distant from the site of infarctions). Angiotensin-converting enzyme inhibitors (ACEI) should be started within 24 hours to reduce the incidence of ventricular remodelling. The drugs can be discontinued if the patient exhibits no signs of ventricular dysfunction (see Table 12-5). ACEIs should be prescribed for patients with UA, NSTEMI, STEMI with a left ventricular ejection fraction (LVEF) of 40% or less, and patients with a history of hypertension, diabetes or chronic kidney disease unless contraindicated.[4] The most common side effect with ACEI is cough, which is chronic and nonproductive. If side effects occur with ACEI, angiotensin receptor blockers (ARBs) should be initiated.[2]

Novel Stem Cell Treatment

Autologous bone marrow stem cell therapy is being used to prevent ventricular remodeling and improve cardiac function. The stem cells are implanted either within the heart or the heart muscle (see box, "Evidence-Based Practice").

Outcomes

Patient outcomes are generalized to encompass the wide spectrum of patients who have experienced an AMI, uncomplicated or complicated, that require medical or surgical intervention. Outcomes include verbalization of relief of pain and fear, adequate cardiac output, ability to tolerate progressive activity, and demonstration of positive coping mechanisms.

INTERVENTIONAL CARDIOLOGY

Several interventions are done to treat ACS. Primary PCI is recommended for treatment of acute STEMI. The goal is to treat the patient to prevent AMI. Intervention is also used after AMI to prevent further damage of the myocardium. PCIs consist of percutaneous transluminal coronary angioplasty (PTCA or angioplasty), percutaneous transluminal coronary rotational atherectomy, directional coronary atherectomy, laser atherectomy, and intracoronary stenting. An early, invasive PCI procedure is indicated for patients with UA/NSTEMI who are hemodynamically unstable or continue to have angina, or have an elevated risk for clinical events.[33] For the purposes of this book, only PTCA and stenting are

EVIDENCE-BASED PRACTICE

Stem Cell Therapy to Improve Cardiac Function

Problem

Novel treatments are needed to prevent ventricular remodeling and improve cardiac function. Remodeling occurs after acute myocardial infarction and can lead to heart failure. Bone marrow stem cell therapy has been studied for about a decade.

Clinical Question

Does bone marrow stem cell therapy prevent ventricular remodelling and improve cardiac function?

Evidence

Strauer and Steinhoff provide an excellent discussion of the role of bone marrow stem cell therapy in the treatment of acute myocardial infarction. They summarize the research and also depict graphically the process of getting stem cells to the cardiac muscle. Tuty Kuswardhani and Soetjitno conducted a systematic review and meta-analysis of 10 randomized controlled trials to assess effect of therapy on cardiac function and secondary outcomes. In comparison to placebo, the analysis found stem cell therapy superior in improving left ventricular function. Therapy was not associated with a reduction in mortality, but it protected patients from recurrent myocardial infarction and rehospitalizations for heart failure. They concluded that stem cell therapy was effective and safe.

Implications for Nursing

Rehospitalization for heart failure secondary to ventricular remodeling after an infarction is a common occurrence. Novel treatment is needed to prevent these physiological changes from occurring. Nurses need to be aware of novel and cutting-edge therapies to improve patient outcomes. Knowledge of stem cell therapy is therefore important to understand. Since the stem cell therapy is autologous, the procedure is safe, clinically justified, and should not be associated with ethical issues related to the practice. Related nursing care is similar to that of post–cardiac catheterization.

Level of Evidence

A—Meta-analysis

References

Strauer B-E, & Steinhoff G. 10 Years of intracoronary and intramyocardial bone marrow stem cell therapy of the heart. *Journal of the American College of Cardiology,* 2011;58:1095-1104.

Tuty Kuswardhani RA, & Soejitno A. Bone marrow-derived stem cells as an adjunctive treatment for acute myocardial infarction: A systematic review and meta-analysis. *Acta Medica Indonesiana*, 2011;43(3):168-177.

discussed. The postprocedure care for all patients who undergo PCI consists of the same interventions.

Percutaneous Transluminal Coronary Angioplasty

The purpose of PTCA is to compress intracoronary plaque to increase blood flow to the myocardium. It is usually the treatment of choice for patients with uncompromised collateral flow, noncalcified lesions, and lesions not present at bifurcations of vessels. PTCA is performed in the cardiac catheterization laboratory. A balloon catheter is inserted in the manner of coronary angiography, but it is threaded into the occluded coronary artery and is advanced with the use of a guidewire across the lesion. The balloon is inflated under pressure one or several times to compress the lesion (Figure 12-10).

The optimal goal after PTCA is open coronary arteries (Figure 12-11). This procedure best treats fixed, noncalcified lesions that are accessible for dilation. Single-vessel disease remains the classic indication for PTCA. PTCA is not recommended for a lesion of the left main coronary artery.[15]

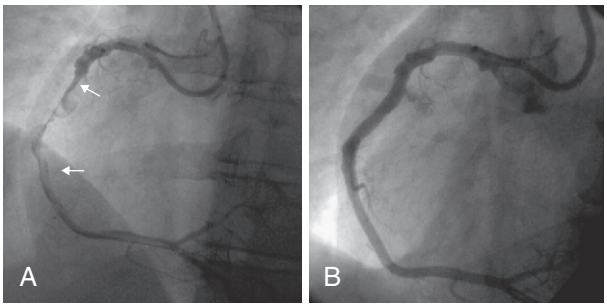

FIGURE 12-11 A, A thrombotic occlusion of the right coronary artery is noted *(arrows)*. **B,** Right coronary artery is opened and blood flow restored following angioplasty and placement of a 4-mm stent. (From Lewis SL. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. St. Louis: Mosby; 2011.)

Complications

Complications of PTCA are commonly due to the angiography and include hematoma at the catheter insertion site, AMI, stroke or transient ischemic attack, pseudoaneurysm,

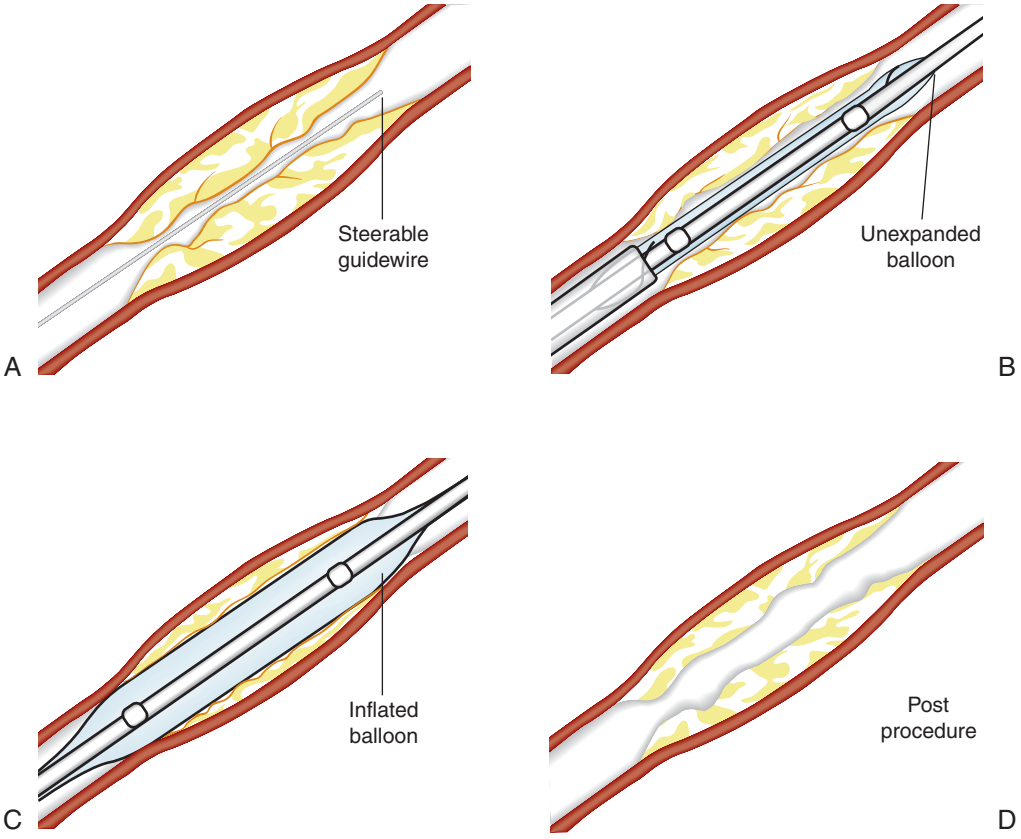

FIGURE 12-10 Coronary angioplasty procedure. **A-D,** Order of procedure. (From Moser DK, Riegel B. *Cardiac Nursing*. St. Louis: Mosby; 2008.)

dysrhythmias, infection, acute kidney injury, and coronary artery dissection. Mortality rates for PTCA are less than 1%, with the risk of AMI less than 1.5%.[33]

Intracoronary Stent

Intracoronary stents are tubes that are implanted at the site of stenosis to widen the arterial lumen by squeezing atherosclerotic plaque against the artery's walls (as does PTCA). However, the stent also keeps the lumen open by providing structural support. Stent designs differ, but most designs have springs, slots, or mesh tubes about 15 mm in length, with some resembling the spiral bindings used in notebooks. These are tightly wrapped around a balloon catheter, which is inflated to implant the stent.

The procedure for placing a stent is similar to the procedure in PTCA, in which the patient first undergoes cardiac angiography for identification of occlusions in coronary arteries. The balloon catheter bearing the stent is inserted into the coronary artery, and the stent is positioned at the desired site. The balloon is inflated, thereby expanding the stent, which squeezes the atherosclerotic plaque and intimal flaps against the vessel wall. After the balloon is deflated and removed, the stent remains, holding the plaque and other matter in place and providing structural support to keep the artery from collapsing (Figure 12-12).[27]

Aggressive anticoagulation therapy before, during, and after the procedure is necessary for the prevention of coagulation. Before sheath removal, peripheral perfusion is monitored because the sheath may cause occlusion of the femoral artery. Peripheral pulses, skin color, and temperature are monitored. The insertion site is inspected for any oozing or bleeding. After sheath removal, hemostasis is maintained with manual pressure, a femoral compression device, or an arterial puncture sealing device. Pain management and proper hydration aid in recovery. Retroperitoneal bleeding or impaired perfusion may occur after sheath removal. Restenosis can occur as a result of neointimal growth because of the body's natural defense when the inner intimal lining is injured, even slightly, as happens with stent placement. Restenosis occurs in 30% to 40% of patients who undergo this procedure. The Gp IIb/IIIa inhibitors are used after stent placement to prevent acute reocclusion through prevention of platelet aggregation.

After a stent procedure, a patient must take antiplatelet agents such as aspirin and clopidogrel,[27] prasugrel, or ticagrelor. Aspirin is used indefinitely at dose ranges of 81 to 162 mg. Oral clopidogrel, 75 mg/day, should be added to aspirin for 3 to 12 months; it may be used as short as 30 days or given indefinitely. Antibiotics are no longer indicated post stent for prophylaxis protection of endocarditis.[33]

Therapies in intracoronary stenting have advanced using both bare metal stents and drug-eluting stents. Normal reaction from the body to vascular injury is neointimal (new intimal cell) growth. When a stent is placed, minor damage to the inner lining of the artery occurs; thus the body's natural defense is to grow new intimal cells to repair the damage, leading to in-stent restenosis.

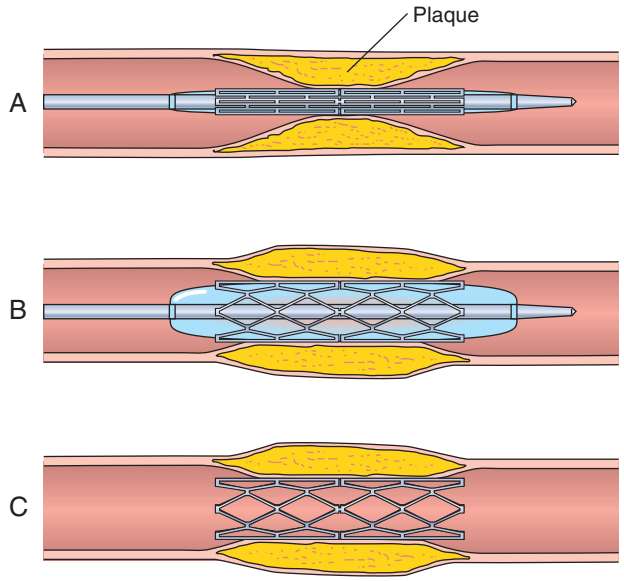

FIGURE 12-12 Placement of coronary artery stent. **A,** The stent is positioned at the site of the lesion. **B,** The balloon is inflated, expanding the stent. The balloon is then deflated and removed. **C,** The implanted stent is left in place. (From Lewis SL. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. St. Louis: Mosby; 2011.)

Drug-eluting stents have the benefit of having an antiproliferative medication coating that reduces in-stent thrombosis. Medication is released slowly over 2 to 4 weeks from the stent to reduce the risk of neointimal growth.[27]

Surgical Revascularization

Surgical approaches used for revascularization include coronary revascularization by coronary artery bypass graft (CABG), minimally invasive CABG, and transmyocardial revascularization (TMR).

Coronary Artery Bypass Graft

CABG is a surgical procedure in which the ischemic area or areas of the myocardium are revascularized by implantation of the internal mammary artery or bypassing of the coronary occlusion with a saphenous vein graft or radial artery graft. The indications for CABG are chronic stable angina that is refractory to other therapies, significant left main coronary occlusion (>50%), triple-vessel CAD, unstable angina pectoris, left ventricular failure, lesions not amenable to PTCA, and PTCA failure.[27]

CABG is performed in the operating room while the patient receives general anesthesia and is intubated. One approach is to make a midsternal, longitudinal incision into the chest cavity. Surgery is done either with cardiopulmonary bypass or without *(off-pump)*. During cardiopulmonary bypass, blood is pumped through an oxygenator, or heart-lung machine, to receive oxygen. Cardioplegia solution is used to stop the heart so surgery can be performed.

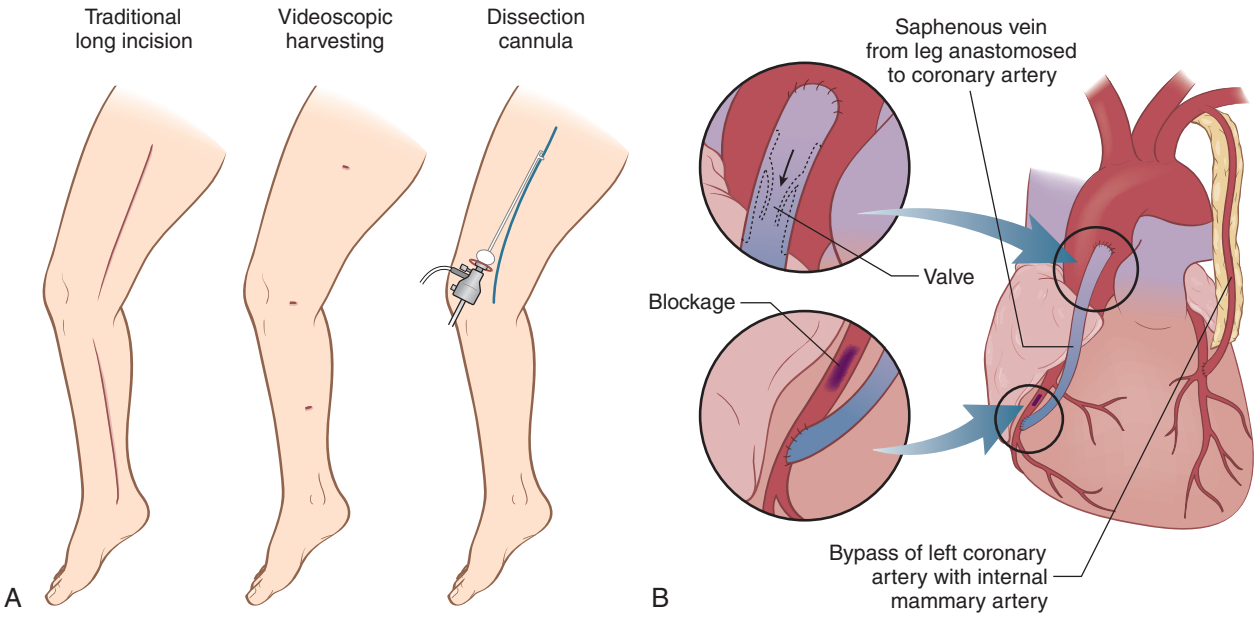

FIGURE 12-13 Coronary artery bypass graft surgery. **A,** Saphenous vein is harvested from the leg using either a traditional long incision or less invasive videoscopic harvesting. **B,** The vein is then anastomosed to the coronary artery.

The coronary arteries are visualized, and a segment of the saphenous vein is grafted or anastomosed to the distal end of the vessel, with the proximal end of the graft vessel anastomosed to the aorta (Figure 12-13). The internal mammary artery is often used for creating an artery-to-artery graft. Internal mammary revascularization has better long-term patency than saphenous vein grafts. It is the preferred graft for lesions of the left anterior descending coronary artery.

Once grafting is done, the cardiopulmonary bypass (if used) is progressively discontinued, chest and mediastinal tubes are inserted, and the chest is closed. Box 12-7 gives information related to chest and mediastinal tubes.[31]

Minimally Invasive Coronary Artery Surgery

Minimally invasive coronary artery surgery is also called limited-access coronary artery surgery.[2] It has been evaluated as an alternative to the standard methods for CABG. Two commonly used approaches include port-access coronary artery bypass (PACAB or PortCAB) and minimally invasive coronary artery bypass (MIDCAB).

In PACAB, the heart is stopped, and the patient undergoes cardiopulmonary bypass. Small incisions (ports) are made in the patient's chest. The surgical team passes instruments through the ports to perform the bypasses using the internal mammary artery, saphenous vein, or radial artery. Procedures to replace damaged valves through limited-access ports are also being done.

The goal of MIDCAB is to avoid using cardiopulmonary bypass. It is performed while the patient's heart is still beating and is intended for use when only one or two arteries will be bypassed. MIDCAB uses a combination of small holes or ports in the chest and a small incision made directly over the coronary artery to be bypassed. The internal mammary artery is commonly used for the graft. The surgeon views and performs the attachment directly, so the artery to be bypassed must be right under the incision.

The American Heart Association's Council on Cardiothoracic and Vascular Surgery has been carefully monitoring these two procedures. MIDCAB appears to be easier on the patient and less expensive than CABG. However, complications may require an open-chest procedure.[2] As these surgical procedures are refined so that they are no more invasive than angioplasty, they will become more common.

Robotically-assisted heart surgery is another type of minimally invasive heart surgery. The cardiac surgeons use a computer console to control surgical instruments on thin robotic arms. Like the other two minimally invasive surgeries just discussed, smaller incisions and quicker recovery times are the primary benefits.

Management after Cardiac Surgery

Patients are usually admitted directly to the critical care unit after cardiac surgery. The patient often has a pulmonary artery catheter, arterial catheter, peripheral IV lines, pleural chest tubes, mediastinal tubes, and an indwelling urinary catheter. The patient is usually mechanically ventilated in the immediate postoperative period. The nurse assesses the patient often and provides rapid interventions to help the patient recover from anesthesia and to prevent complications. The nurse-to-patient ratio is often 1:1 during the first few hours after surgery or until the patient is extubated. Nursing care for these patients is summarized in Box 12-8.

BOX 12-7 KEY POINTS FOR MAINTAINING PLEURAL CHEST AND MEDIASTINAL TUBES

Definitions

- **Pleural chest tube:** The tube is inserted into the pleural space to maintain the normal negative pressure and to facilitate respiration. It is inserted after cardiac surgery if the pleural space is opened. It is also inserted as treatment for pneumothorax or hemothorax.
- **Mediastinal tube:** The tube is inserted into the mediastinal space to provide drainage after cardiac surgery.
- **Drainage system:** A water-seal system assists in maintaining negative pressures (chest tube). Some devices are designed to function without water (dry). Suction (up to 20 cm H_2O) is often applied to facilitate drainage.
- **Autotransfusion:** Reinfusion of autologous drainage from the system back to the patient.

Baseline Assessment

- Make sure that all connections are tight: insertion site to the chest drainage system, suction control chamber to the suction unit.
- Assess that the dressing over insertion site is dry and intact.
- Palpate for subcutaneous crepitus around the insertion site and chest wall.
- Auscultate breath sounds bilaterally.
- Observe the color and consistency of fluid in the collecting tubing (more accurate assessment than fluid in the drainage system); mark the fluid level on the drainage system.
- Assess the drainage system for proper functioning (read instructions for the device being used).
- Check the water in the water-seal level; the water level should fluctuate with respirations in chest tubes (not in mediastinal tubes).
- Check suction control and be sure that suction is on, if ordered.
- Check for intermittent bubbling in the water-seal chamber; it indicates an air leak from the pleural space (pleural tube).

Maintaining the Chest Drainage System

- Keep the tubing coiled on the bed near the patient.
- Record drainage in the medical record per protocol; notify the provider of excessive drainage (volume to report determined by unit parameters or written order; volume varies depending on purpose of the tube and time since insertion).
- Change the dressing according to unit protocol.
- Splint the insertion site to facilitate coughing and deep breathing.
- Ensure that drainage flows into the drainage system by facilitating gravity drainage; *milking* and *stripping* the tubes are not recommended because these procedures generate high negative pressures in the system.
- If the patient is transported (or ambulated) disconnect the drainage system from suction and keep it upright below the level of the chest. Do not clamp the tube.
- Chest x-ray studies are done immediately after insertion and usually daily thereafter.

Assisting with Removal

- Chest and mediastinal tubes are usually removed by the provider.
- Ensure adequate pain medication before removal.
- Apply an occlusive dressing to the site after removal.
- A chest x-ray study is usually done after removal.

Autotransfusion

- An autotransfusion collection system is attached to the chest drainage device.
- Anticoagulants may be ordered to be added to the autotransfusion system (citrate-phosphate-dextrose, acid-phosphate-dextrose, or heparin); these are not usually necessary with mediastinal drainage.
- Reinfuse drainage within the time frame specified by unit policy. It is recommended that reinfusion begin within 6 hours of initiating the collection, and reinfused to the patient within a 4-hour period.
- Evacuate air from the autotransfusion bag; air embolism may occur unless all air is removed.
- Attach a microaggregate filter and infuse via gravity or a pressure bag

BOX 12-8 NURSING INTERVENTIONS AFTER CARDIAC SURGERY

- Monitor for hypotension; administer fluids and vasopressors as ordered or based on protocol.
- Assess for hypovolemia; monitor and trend output from the pleural chest and mediastinal tubes and urine output.
- Monitor hemodynamic pressures, SvO_2, stroke index, cardiac index, PAOP, and RAP; treat the patient per protocol.
- Rewarm the patient gradually (if applicable).
- Monitor and treat fluid and electrolytes, hemoglobin, hematocrit, renal function, and coagulation studies.
- Provide pain relief.
- Monitor for complications: intraoperative AMI, dysrhythmias, heart failure, cardiac tamponade, thromboembolism, impaired renal function, pneumonia, pneumothorax, pleural effusion, cerebral ischemia, or stroke.
- Wean from mechanical ventilation per protocol; extubate; promote pulmonary hygiene every 1 to 2 hours while the patient is awake.
- Assess wounds and provide incisional care per hospital protocol.
- Gradually increase the patient's activity.
- Provide emotional support to the patient and family.

PAOP, Pulmonary artery occlusion pressure; *RAP,* right atrial pressure; *SvO_2,* mixed venous oxygen saturation.

Complications of Cardiac Surgery

Patients who have had cardiac surgery should be closely monitored for complications such as dysrhythmias (atrial fibrillation, atrial flutter, ventricular tachycardia, ventricular fibrillation), MI, shock, pericarditis, pericardial effusion, and cardiac tamponade. The critical care nurse taking care of a patient who has just undergone CABG must have quick critical thinking skills and the ability to assess the whole picture, while prioritizing interventions that need to be performed.

MECHANICAL THERAPIES

Transmyocardial Revascularization

In transmyocardial revascularization (TMR), a high-energy laser creates channels from the epicardial surface into the left ventricular chamber. This procedure is also called *laser revascularization*. The purpose of TMR is to increase perfusion directly to the heart muscle. It is performed on patients who are poor candidates for CABG and whose symptoms are refractory to medical treatment. To do this procedure, a surgeon makes an incision on the left side of the chest and inserts a laser into the chest cavity. With the laser, the surgeon makes channels (1 mm) through the heart's left ventricle in between heartbeats. The laser is fired when the chamber is full of blood so the blood can protect the inside of the heart. Twenty to 40 channels are created.[3] Then the surgeon applies pressure on the outside of the heart. This seals the outer openings but lets the inner channels stay open, to allow oxygen-rich blood to flow through the heart muscle.

TMR has received Food and Drug Administration approval for use in patients with severe angina who have no other treatment options. It has produced early promising results in that the angina of 80% to 90% of patients who have had this procedure has significantly improved (at least 50%) through 1 year after surgery. There are still limited follow-up data as to how long the benefits of this procedure might last.[3] Improvement in symptoms usually occurs over time, not immediately. TMR will not replace CABG or angioplasty as a common method of treating CAD. TMR may be used for patients who are high-risk candidates for a second bypass or angioplasty, for example, patients whose blockages are too diffuse to be treated with bypass alone, or some patients with heart transplants who develop atherosclerosis.

Enhanced External Counterpulsation

Enhanced external counterpulsation (EECP) is a treatment used for angina when the patient is not a candidate for bypass surgery or percutaneous coronary intervention. EECP uses cuffs wrapped around the patients legs to increase arterial blood pressure and retrograde aortic blood flow during diastole. Sequential pressure, using compressed air, is applied from the lower legs to the upper thighs. These treatments take place over the course of a few hours per day for several weeks. There are no definite data that EECP reduces ischemia; however, there is evidence to support a reduction in angina.[21]

CARDIAC DYSRHYTHMIAS

Cardiac dysrhythmias have many causes such as CAD, AMI, electrolyte imbalances, and HF. The various dysrhythmias and patient assessment are discussed in Chapter 7. Emergency treatments of dysrhythmias include medications, transcutaneous pacemakers, and cardioversion and defibrillation (see Chapter 10). Other drugs that may be used to manage dysrhythmias are shown in Table 12-9. Additional surgical and electrical treatments are discussed in the following sections.

Radiofrequency Catheter Ablation

Radiofrequency catheter ablation is a method used to treat dysrhythmias when medications, cardioversion, or both, are not effective or not indicated. The objective of catheter ablation is to permanently interrupt electrical conduction or activity in a region of dysrhythmogenic cardiac tissue. Indications for radiofrequency catheter ablation include the presence of dysrhythmias such as ventricular tachycardia, atrial fibrillation, atrial flutter, and AV nodal reentry tachycardia. The most predominant group are those patients with symptomatic paroxysmal atrial fibrillation.[30]

Radiofrequency ablation is performed percutaneously. The procedure begins with a diagnostic electrophysiology (EP) study to map the areas to be ablated. A catheter with an electrode is positioned at the accessory (abnormal) pathway, and mild, painless radiofrequency energy (similar to microwave heat) is transmitted to the pathway, causing coagulation and necrosis in the conduction fibers without destroying the surrounding tissue. This stops the area from conducting the extra impulses that cause the tachycardia. After each ablation attempt, the patient is retested until there is no recurrence of the tachycardiac rhythm.

A radiofrequency ablation technique called circumferential radiofrequency ablation is used to treat atrial fibrillation. The lines of electrical conduction that may contribute to atrial fibrillation are located where the pulmonary veins connect to the left atrium. Radiofrequency ablation is done in a circular pattern around each pulmonary vein opening. In addition, pulmonary vein isolation may be used to determine sites for ablation.[30]

Pacemakers

Temporary pacemakers are used to treat patients urgently who are waiting for a permanent pacemaker placement or to treat transient bradydysrhythmias. Temporary pacemakers types are external (transcutaneous) or transvenous. External pacing requires large electrodes that are attached to the chest (see Chapter 10). This type of pacing is quite uncomfortable for the patients because of the current of electricity that is required to pace the heart; therefore it is only used on an emergency basis. Transvenous pacing uses a wire passed through the venous system into the heart and connected to an external pulse generator. This type is more comfortable, but is only used for a short period of time due to the risk of infection and venous thrombosis.[13]

TABLE 12-9 PHARMACOLOGY

Medications Used to Treat Dysrhythmias

MEDICATION	INDICATIONS	MECHANISM OF ACTION	DOSE/ROUTE	SIDE EFFECTS	NURSING IMPLICATIONS
Diltiazem (Cardizem)	Atrial fibrillation/ flutter SVT	Inhibits calcium ion influx into vascular smooth muscle and myocardium	*IV:* 0.25 mg/kg actual body weight over 2 minutes May repeat in 15 minutes at dose of 0.35 mg/kg actual body weight *Infusion:* 5-15 mg/hour x 24 hours	Hypotension, edema, dizziness, bradycardia	Often used in conjunction with digoxin for rate control Not used in heart failure Observe for dysrhythmias
Amiodarone (Cordarone)	Atrial fibrillation/ flutter SVT Ventricular dysrhythmias	Prolongs action potential phase 3	*IV:* 150 mg IV in 100 mL 5% dextrose/ water over 10 minutes (15 mg/min). Follow with infusion of 360 mg over next 6 hours at 1 mg/min rate (mix 900 mg in 500 ml solution; 1.8 mg/mL). Follow with maintenance infusion of 540 mg over remaining 18 hours (0.5 mg/min). Maintenance infusion can be continued at 0.5 mg/min for 2 to 3 weeks *PO:* For life-threatening dysrythmias, loading dose of 800-1600 mg/day for 1-3 weeks; decrease dose to 600-800 mg/day for 1 month decrease to lowest therapeutic dose, usually 400 mg/day	Bradycardia, complete atrioventricular block, hypotension Multiple side effects (thyroid, pulmonary, hepatic, neurological, dermatological)	Long half-life Monitor cardiac rhythm Obtain baseline pulmonary and liver function tests
Flecainide (Tambocor)	Ventricular dysrhythmias	Decreases conduction in all parts of the heart; stabilizes cardiac membrane	*PO:* 50-100 mg every 12 hours; increase as needed, not to exceed 400 mg/day	Hypotension, bradycardia, heart block, blurred vision, respiratory depression	Interacts with many other drugs; check drug guide Monitor cardiac rhythm Monitor intake and output Assess electrolytes Assess for central nervous system symptoms
Sotalol (Betapace)	Ventricular dysrhythmias	Nonselective beta-blocker	*PO:* 80 mg BID Increase to 240-320 mg/day	Hematological disorders, bronchospasm	Monitor blood pressure and pulse rate Check baseline liver and renal function before beginning therapy Monitor hydration Watch for QT prolongation Teach patient not to decrease drug abruptly
Ibutilide (Corvert)	Atrial fibrillation/ flutter	Prolongs duration of action potential and refractory period	*IV:* 1 mg IV push over 10 minutes; may repeat after 10 minutes	Hypotension, bradycardia, sinus arrest	Monitor cardiac rhythm Assess for central nervous system symptoms Use usually restricted to electrophysiology personnel
Propafenone (Rythmol)	Ventricular dysrhythmias	Stabilizes cardiac membranes; depresses action potential phase 0	*PO:* 150 mg every 8 hours; 450-900 mg/day	Ventricular dysrhythmias, heart failure, dizziness, nausea/ vomiting, altered taste	Monitor cardiac rhythm Use in patients without structural heart disease

BID, Twice daily; *ECG,* electrocardiogram; *IV,* intravenous; *PO,* orally; *SVT,* supraventricular tachycardia.
From Skidmore-Roth L. (2011). *Mosby's 2011 Nursing Drug Reference.* St. Louis: Mosby.

TABLE 12-10 THE NASPE/BPEG GENERIC (NBG) PACEMAKER CODE (REVISED 2000)

POSITION	I	II	III	IV	V
Category:	**Chamber(s) paced**	**Chamber(s) sensed**	**Response to sensing**	**Rate modulation**	**Multisite pacing**
	O = None	**O** = None	**O** = None	**O** = None	**O** = None
	A = Atrium	**A** = Atrium	**T** = Triggered	**R** = Rate modulation	**A** = Atrium
	V = Ventricle	**V** = Ventricle	**I** = Inhibited		**V** = Ventricle
	D = Dual (A + V)	**D** = Dual (A + V)	**D** = Dual (T + I)		**D** = Dual (A + V)
Manufactures' designation:	**S** = Single (A or V)	**S** = Single (A or V)			

From Bernstein AD, Daubert J-C, Fletcher RD, Hayes DL, Lüderitz B, Reynolds DW, Schoenfeld MH, Sutton R. The Revised NASPE/BPEG Generic Code for antibradycardia, adaptive-rate, and multisite pacing. *Journal of Pacing and Clinical Electrophysiology* 2002;25:260-264.

Permanent pacemakers are used to treat conduction disturbances of the heart. Guidelines for implantation of pacemakers were most recently updated in 2008. They include sinus node dysfunction, atrioventricular block, neurocardiogenic syncope, and some tachycardias.[10] Biventricular pacemakers are used to treat heart failure and will be discussed along with implantable cardioverter-defibrillators. Pacemakers are inserted in the operating room, cardiac catheterization lab, or electrophysiology lab depending on the facility. Depending on the indication, the patient may require atrial and/or ventricular pacing. Leads are inserted through the venous system and into the right atrium and/or right ventricle. A pulse generator is attached to the leads and implanted under the skin, usually on the left side of the chest. Pacemakers are powered by lithium batteries that last approximately 7 to 10 years, at which point a new pulse generator is implanted and attached to the existing functioning leads.

Pacemakers are referred to by a lettered code used to describe their basic function. This code has been modified by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group, currently known as Heart Rhythm Society. The code uses three or four letters to define the chamber paced, chamber sensed, response to pacing, and rate responsiveness, if rate response is being used (Table 12-10).[7]

Cardiac resynchronization therapy (CRT) is permanent pacing with an additional lead placed in the left ventricle. It is indicated to provide therapy for patients with heart failure, with a widened QRS complex and left ventricular ejection fraction of 35% or less, who are on maximum medical therapy and remain symptomatic.[10] Cardiac resynchronization therapy involves biventricular pacing to synchronize contractions of both ventricles. This improves symptoms of heart failure, decreases mortality, and decreases hospital readmissions. It can be implanted as pacemaker device or, as is more common, in combination with a defibrillator.

Defibrillators

Implantable cardioverter-defibrillators (ICDs) are placed in patients for primary or secondary prevention of potentially lethal dysrhythmias. In primary prevention, they are indicated for patients who are at risk of sudden cardiac death (SCD) such as patients with heart failure, patients who have genetic mutations that put them at risk for ventricular dysrhythmias, and certain congenital and structural heart diseases.[10] In secondary prevention, they are implanted in patients who have survived cardiac arrest or sustained ventricular tachycardia (VT). Current indications for ICD therapy are listed in Box 12-9. ICDs are also able to detect fast heart rates, and when necessary deliver a shock to the heart to stop the abnormal heart rhythm.

ICDs are implanted in the same manner as pacemakers by electrophysiologists (cardiologists who specialize in cardiac

BOX 12-9 INDICATIONS FOR AN IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR

- Cardiac arrest resulting from ventricular fibrillation (VF) or ventricular tachycardia (VT) not produced by a transient or reversible cause or in the event of AMI when revascularization cannot be done
- Spontaneous sustained VT in association with structural heart disease
- Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced during electrophysiological study
- Nonsustained VT in patients with coronary artery disease, prior myocardial infarction, left ventricular dysfunction, and inducible VF or sustained VT during electrophysiological study
- Patients with left ventricular ejection fraction of 30% or less, at least 40 days after myocardial infarction and 3 months after coronary revascularization
- Patients with left ventricular ejection fracture less than 35%, NYHA Class II-III in nonischemic heart disease or ischemic heart disease with no coronary revascularization on optimal medical therapy

From Gami A S, Hayes D L, & Friedman P A. (2008). Indications for Pacemakers, ICDs and CRT. In D L Hayes & P A Friedman (Eds.), *Cardiac Pacing, Defibrillation and Resynchronization* (2nd ed.). West Sussex: Wiley-Blackwell.

BOX 12-10 PATIENT AND FAMILY TEACHING FOR AN IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR

Preprocedural Teaching
- Device and how it works
- Lead and generator placement
- Implantation procedure
- Educational materials from the manufacturer

Postprocedural Teaching
- Site care and symptoms of complications
- Hematoma at the site is most common when patient takes anticoagulant or antiplatelet medications
- Restricting activity of the arm on the side of the implant
- Identification (Medic Alert jewelry and ICD card)
- Diary of an event if the device fires
- Response if the device fires (varies from falling, tingling, or discomfort to no awareness of the shock); family members need to help in assessment
- Safety measures:
  - Avoid strong magnetic fields (no magnetic resonance imaging)
  - Avoid sources of high-power electricity
  - Keep cellular phones at least 6 inches from the ICD
  - Inform airline security personnel about the device; avoid the metal detector; the security wand may be used but should not be left over the device
  - The defibrillator therapy must be turned off for surgical procedures using electrocautery
- Everyday activities:
  - Hairdryers, microwaves, and razors are safe
  - Sexual activity can be resumed; tachycardia associated with sexual activity may cause the device to fire; rate adjustments may be needed; If shock occurs during sexual activity, it will not harm the partner
  - Avoid driving for 6 months if the patient has a history of sudden cardiac arrest
- Testing of the device requiring additional electrophysiological studies
- Replacement of the device
- Instruction of family members in cardiopulmonary resuscitation and in how to contact emergency personnel
- Support groups in the local community

ICD, Implantable cardioverter-defibrillator.

rhythms). All ICDs are developed with pacemaker capabilities in the rare instance when the patient needs backup pacing after receiving an ICD shock. ICDs are also capable of providing CRT for patients that require it.

Pacemaker and ICD functions are periodically checked in the office and at home using telemonitoring. These checks help to ensure proper functioning of the devices, and determine when the battery needs to be replaced. The patient is instructed to carry a wallet identification card at all times (see additional patient and family teaching in Box 12-10). Although newer devices are being designed for MRI compatibility, patients who currently have these devices are restricted from undergoing an MRI.

HEART FAILURE

Heart failure (HF) is a complex clinical syndrome that results from the heart's inability to pump blood sufficiently to meet the metabolic demands of the body.[8] HF can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood. CAD is the primary underlying cause of HF; however, several nonischemic causes have been identified: hypertension, valvular disease, exposure to myocardial toxins, myocarditis, untreated tachycardia, alcohol abuse, and sometimes unidentifiable causes (which result in idiopathic dilated cardiomyopathy).

The cardinal manifestations of HF are dyspnea, fatigue, exercise intolerance, and fluid retention, which may lead to pulmonary and peripheral edema. Signs and symptoms of HF consist of progressive exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, fatigability, loss of appetite, abdominal bloating, nausea or vomiting, and eventual organ system dysfunction, particularly the renal system as the failure advances.

The American Heart Association and American College of Cardiology developed a classification system for HF. A patient is classified from stage A to D, based on results of physical examination, diagnostic tests, and clinical symptoms. This terminology helps in understanding that HF is often a progressive condition and worsens over time. HF can be asymptomatic (stages A and B, pre-HF) or symptomatic (stages C and D).[12] HF also has a classification system based on symptoms. The New York Heart Association (NYHA) Heart Failure Symptom Classification System is used to determine functional limitations, and it is also an indicator of prognosis. Class I refers to no symptoms with activity, up to Class IV which indicates dyspnea with little or no exertion.[12] The two classification systems can be used with each other (Table 12-11).

Pathophysiology

HF is impaired cardiac function of one or both ventricles. HF is also classified as systolic or diastolic. Systolic HF results from impaired pumping of the ventricles. Diastolic HF results from impaired filling or relaxation of the ventricles. The most common type of HF is left-sided systolic dysfunction. Right-sided dysfunction is usually a consequence of left-sided HF; however, it can be a primary cause of HF after a right ventricular MI, or it may be secondary to pulmonary pathology. Selected causes of HF are noted in Box 12-11.[12]

TABLE 12-11 ACC/AHA 2001 STAGING COMPARED TO NYHA FUNCTIONAL CLASSIFICATION

ACC/AHA		NYHA	
A	At high risk of developing HF, but without structural heart disease or symptoms of HF	None	
B	Structural heart disease or symptoms of HF	I	Asymptomatic
C	Structural heart disease with prior or current symptoms of HF	II	Symptomatic with moderate exertion
		III	Symptomatic with minimal exertion
		IV	Symptomatic with rest
D	Refractory HF requiring specialized interventions	IV	Symptomatic with rest

ACC, American College of Cardiology; *AHA,* American Heart Association; *HF,* heart failure; *NYHA,* New York Heart Association.
From Institute for Clinical Systems Improvement (ICSI): Health Care Guideline: Heart failure in adults (August 2011). http://www.icsi.org/heart_failure_2/heart_failure_in_adults_.html

BOX 12-11 CAUSES OF HEART FAILURE

Left Heart Systolic Failure
- Myocardial infarction
- Coronary artery disease
- Cardiomyopathy
- Hypertension
- Valvular heart disease
- Tachydysrhythmias
- Toxins: cocaine, ethanol, chemotherapy agents
- Myocarditis
- Pregnancy postpartum cardiomyopathy

Left Heart Diastolic Failure
- Myocardial infarction
- Coronary artery disease
- Hypertrophic heart disease
- Pericarditis
- Infiltrative disease: amyloid sarcoid
- Radiation therapy to the chest
- Age
- Hypertension

Right Heart Systolic Failure
- Right ventricular infarction
- Left-sided heart failure
- Pulmonary embolus
- Pulmonary hypertension
- Chronic obstructive pulmonary disease
- Septal defects

Right Heart Diastolic Failure
- Right ventricular hypertrophy
- Infiltrative disease: amyloid, sarcoid
- Radiation therapy to the chest

In left-sided HF, the left ventricle cannot pump efficiently. The ineffective pumping action causes a decrease in cardiac output, leading to poor perfusion. The volume of blood remaining in the left ventricle increases after each beat. As this volume increases, it backs up into the left atrium and pulmonary veins and into the lungs, causing congestion. Eventually, fluid accumulates in the lungs and pleural spaces, causing increased pressure in the lungs. Gas exchange (oxygen and carbon dioxide) in the pulmonary system is impaired. The backflow can continue into the right ventricle and right atrium and into the systemic circulation (right-sided HF).

When gas exchange is impaired and carbon dioxide increases, the respiratory rate increases to help eliminate the excess carbon dioxide. This phenomenon causes the heart rate to increase, pumping more blood to the lungs for gas exchange. The increased heart rate results in the pumping of more blood from the systemic circulation into the cardiopulmonary circulation, which is already dangerously overloaded, thus a vicious cycle ensues.

As the heart begins to fail to meet the body's metabolic demands, several compensatory mechanisms are activated to improve cardiac output and tissue perfusion. The most noteworthy of these neurohormonal systems are the renin-angiotensin-aldosterone system and the adrenergic nervous system. These interrelated systems act in concert to redistribute blood to critical organs in the body by increasing peripheral vascular tone, heart rate, and contractility. The activation of these diverse systems may account for many of the symptoms of HF and may contribute to the progression of the syndrome. Although these responses may be initially viewed as compensatory, many of them are or become counterregulatory and lead to adverse effects.[8]

The *renin-angiotensin-aldosterone system* plays a major role in the pathogenesis and progression of HF. Angiotensin II is a potent vasoconstrictor and promotes salt and water retention by stimulation of aldosterone release. Sodium

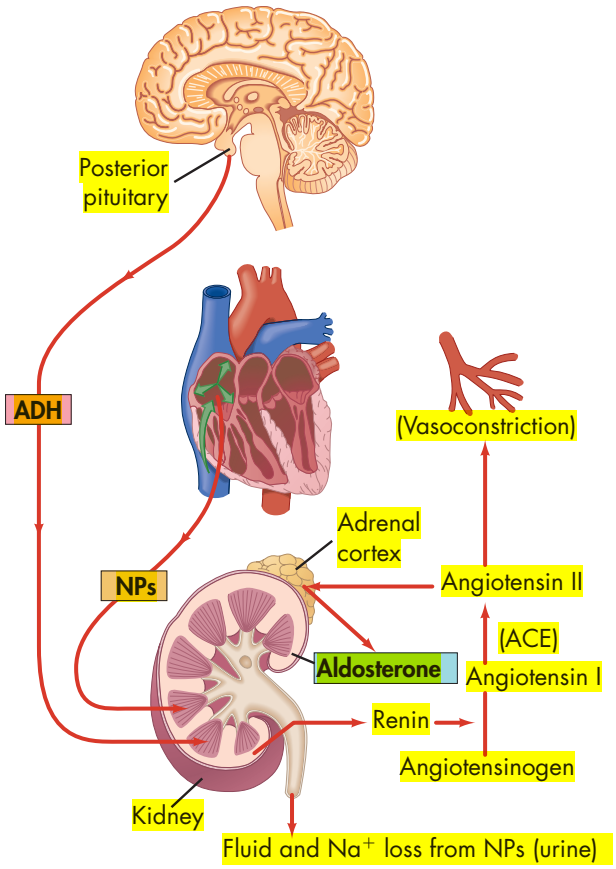

FIGURE 12-14 Three mechanisms that influence total plasma volume. *ACE*, Angiotensin converting enzyme; *ADH*, antidiuretic hormone; *Na*$^+$, sodium; *NPs*, natriuretic peptides. (From McCance KL, Huether SE. *Pathophysiology. The Biologic Basis for Disease in Adults and Children*. 6th ed. St. Louis: Mosby; 2010; Modified from Thibodeau GA, Patton KT: *Anatomy and Physiology*. 5th ed. St. Louis: Mosby; 2003.)

reabsorption increases, and this, in turn, increases blood volume. In patients with impaired function, the heart is unable to handle the extra volume effectively, resulting in edema (peripheral, visceral, and hepatic) (Figure 12-14).

The *adrenergic nervous system* is activated. Although this is initially beneficial in preserving cardiac output and systemic blood pressure, chronic activation is deleterious. Activation (1) produces tachycardia, thereby decreasing preload and contributing to a further decrease in stroke index; (2) causes vasoconstriction, which increases afterload, further decreasing stroke index; and (3) increases contractility, which increases myocardial oxygen demand, thereby decreasing contractility and possibly decreasing stroke index. These changes are progressive. In time, the ventricle dilates, hypertrophies, and becomes more spherical. This process of cardiac remodeling generally precedes symptoms by months or even years.[8]

Assessment

Patient assessment includes the identification of the cause of both right-sided and left-sided HF, the signs and symptoms, and precipitating factors as well as diagnostic studies. Signs and symptoms of HF are presented in Box 12-12.[8]

BOX 12-12 SIGNS AND SYMPTOMS OF HEART FAILURE

Left-Sided Heart Failure: Poor Pump

- Dyspnea/orthopnea
- Cheyne-Stokes
- Paroxysmal nocturnal dyspnea
- Cough (orthopnea equivalent)
- Fatigue or activity intolerance
- Diaphoresis
- Pulmonary crackles
- Elevated pulmonary capillary occlusion pressure
- S_3 and S_4 gallop
- Tachycardia
- Tachypnea
- Hepatojugular reflux

Right Sided Heart Failure: Excess Volume

- Jugular venous distention
- Liver engorgement (hepatomegaly) with ascites in severe cases
- Edema
- Loss of appetite, nausea, vomiting
- Elevated central venous or right atrial pressure

Diagnosis

In diagnosing HF, it is important to identify the etiology or precipitating factors. It is also important to determine whether ventricular dysfunction is systolic or diastolic because therapies are different. Ischemia is responsible for most cases of HF. Identifying ischemia as a cause of HF is important because a majority of these patients may benefit from revascularization.

Diagnosis of the patient with suspected HF includes the following:

- *A complete history including precipitating factors*
- *Physical examination, including assessment of:*
  - Intravascular volume, with examination of neck veins and presence of hepatojugular reflux
  - Presence or absence of edema
  - Perfusion status, which includes blood pressure, quality of peripheral pulses, capillary refill, and temperature of extremities
  - Lung sounds, which may not be helpful. In many cases, the lung fields are clear when the patient is obviously congested, a reflection of chronicity of the disease and adaptation.
- *Chest x-ray study* to view heart size and configuration and to check the lung fields to determine whether they are clear or opaque (fluid filled)
- *Hemodynamic monitoring with pulmonary artery catheter.* Mixed venous oxygen saturation, stroke index, cardiac index, and pulmonary artery pressures are important parameters to assess in the most critically ill patients, especially those who do not respond to conventional therapy. Noninvasive methods of determining hemodynamic parameters also helpful (see Chapter 8).
- *Noninvasive imaging of cardiac structures.* The single most useful test in evaluating patients with HF is the echocardiogram, which can evaluate ventricular enlargement, wall motion abnormalities, valvular structures. It will also determine the left ventricular ejection fraction (LVEF).
- *Arterial blood gases to assess oxygenation and acid-base status*
- *Serum electrolytes.* Many electrolyte imbalances are seen in patients with HF. A low serum sodium level is a sign of advanced or end-stage disease; a low potassium level is associated with diuresis; a high potassium level is seen in renal impairment; blood urea nitrogen and creatinine levels are elevated in low perfusion states, renal impairment, or with overdiuresis.
- *Complete blood count to assess for anemia*
- *B (brain)-type natriuretic peptide (BNP).* BNP is a cardiac hormone secreted by ventricular myocytes in response to wall stretch. BNP and ProBNP assays are useful in the diagnosis of patients with dyspnea of unknown etiology.[11] BNP is a good marker for differentiating between pulmonary and cardiac causes of dyspnea.[9] Plasma concentrations of BNP reflect the severity of HF. In decompensated HF, the BNP concentration increases as a response to wall stress or stretch. As the HF is treated, BNP is used to assess the response to therapies. The normal BNP concentration is less than 100 pg/mL. A BNP level greater than 500 pg/mL is highly specific and indicates increased mortality risk short-term. Patients are at increased risk of readmission and death if the BNP concentration remains persistently elevated at the time of discharge.[11] BNP is not a good indicator of heart failure for patients with chronic renal insufficiency.
- *Liver function studies.* The liver often becomes enlarged with tenderness because of hepatic congestion. Serum transaminase and bilirubin levels are elevated with diminished liver function. Function usually returns once the patient is treated and euvolemic.
- *ECG.* Intraventricular conduction delays are common. Left bundle branch blocks are often associated with structural abnormalities. Patients frequently have premature ventricular contractions, premature atrial contractions, and atrial dysrhythmias. Sinus tachycardia at rest implies substantive cardiac decompensation, and detection of this occurrence is essential.

Nursing Diagnoses

Many nursing diagnoses are associated with HF, such as decreased cardiac output, fluid volume excess, and activity intolerance. See the "Nursing Care Plan for the Patient with Heart Failure" for nursing diagnoses, outcomes, interventions, and rationale.

NURSING CARE PLAN

for the Patient with Heart Failure

NURSING DIAGNOSIS

Decreased Cardiac Output related to increased preload or afterload, decreased cardiac contractility, dysrhythmias, impaired diastolic function

PATIENT OUTCOMES

Adequate cardiac output

- Clear lung sounds
- No shortness of breath
- Absence of or reduced edema

NURSING INTERVENTIONS	RATIONALES
• Assess rate and quality of apical and peripheral pulses	• Assess for compensatory tachycardia
• Assess BP for orthostatic changes	• Low CO as well as vasodilating medications may alter adequate perfusion
• Assess for presence of S_3 and S_4 heart sounds	• Assess left ventricular ejection or reduced compliance
• Assess lung sounds	• Crackles reflect fluid accumulation
• Assess for complaints of fatigue or altered activity tolerance	• Common in low CO states
• Assess urine output	• Assess renal perfusion
• Determine mental status changes, restlessness, irritability	• Assess for alteration in cerebral perfusion
• Assess oxygen saturation; administer supplemental oxygen to maintain saturation above 90%	• Ensure adequate oxygenation
• Monitor serum electrolytes	• Assess risk factors for dysrhythmias
• Assess BNP	• Elevated with increased left ventricular filling pressures
• Monitor for signs/symptoms of digitalis toxicity	• Therapeutic and toxic margin is narrow
• Weigh and evaluate trends	• Assess for fluid volume status
• Administer medications	• Many medications needed to improve CO; assess response

• Optimize preload
  • Increased preload—restrict fluids and sodium
  • Decreased preload—increase fluids
	• Promote adequate CO

• Consider invasive hemodynamic monitoring	• Provide data to guide treatment

NURSING DIAGNOSIS

Excess Fluid Volume related to impaired cardiac contractility and decreased cardiac output

PATIENT OUTCOMES

Optimal fluid balance

- Stable weight
- Absence of or reduction in edema
- Clear lung sounds

NURSING INTERVENTIONS	RATIONALES
• Monitor and trend daily weight	• Weight gain of 2-3 lb in a day or 5 lb in 1 week indicates excess fluid volume
• Assess for presence of edema over ankles, feet, sacrum, and dependent areas	• Symmetrical dependent edema is characteristic in HF
• Auscultate for adventitous lung sounds and assess for labored breathing	• Elevation of pulmonary pressure shifts fluid to interstitial and alveolar spaces
• Assess for JVD, ascites, nausea, and vomiting	• Right-sided HF increases venous pressure and fluid congestion
• Assess electrolyte imbalances—low potassium, low sodium, low magnesium, and elevated creatinine levels	• Monitor for side effects of diuretics
• Consider hemofiltration or ultrafiltration for excess fluid volume	• Remove excess fluid volume
• Position patient comfortably with head of bed elevated	• Decrease orthopnea

Continued

NURSING CARE PLAN

for the Patient with Heart Failure—cont'd

NURSING DIAGNOSIS

Risk for Electrolyte Imbalance related to changes in volume status, decreased renal perfusion, diuretics, low-sodium diet

PATIENT OUTCOMES

Electrolytes within normal range

NURSING INTERVENTIONS	RATIONALES
• Monitor serum electrolyte levels	
• Hyponatremia	• Hyponatremia may be dilutional
• Hypokalemia	• Require higher safety range for normal potassium
• Hypomagnesemia	• Dysrhythmias increase risk of sudden death
• Hypernatremia	• Hypernatremia is caused by large loss of water
• Hyperkalemia	• Coadministration of ACE inhibitors, ARBs, or aldosterone blockers can cause potassium retention, especially if decreased renal function worsens as well
• Place on cardiac monitor	• Assess for dysrhythmias associated with electrolyte imbalances
• Administer diuretics	• Restore water and sodium balance
• Administer electrolyte supplements by mouth or intravenously: provide appropriate diet with foods that contain supplements	• Prevent electrolyte imbalances via medication and/or diet

NURSING DIAGNOSIS

Activity Intolerance related to decreased cardiac output, deconditioning, sedentary lifestyle, imbalance between oxygen supply and demand, insufficient sleep and rest, lack of motivation, depressions

PATIENT OUTCOMES

Improved activity tolerance

- Able to perform required activities of daily living
- Verbalizes and uses energy conservation techniques

NURSING INTERVENTIONS	RATIONALES
• Assess patient's current level of activity	• Assess baseline activity
• Observe and document response to activity	• HR increases >20 beats/min; BP drop of >20 mm Hg, dyspnea, light-headedness, and fatigue signify abnormal responses to activity
• Monitor sleep pattern and amount of sleep during night and day	• Provide adequate rest to facilitate progression of activity
• Evaluate need for oxygen with activity	• Compensate for increased oxygen demand
• Teach energy conservation techniques	• Reduce oxygen consumption
• Sit for tasks	
• Push rather than pull	
• Slide rather than lift	
• Store frequently used items within reach	
• Organize a work-rest-work schedule	
• Provide emotional support and encouragement	• Promote positive reinforcement to guide activity progression

NURSING CARE PLAN

for the Patient with Heart Failure—cont'd

NURSING DIAGNOSIS

Disturbed Sleep Pattern related to anxiety or fear, physical discomfort, shortness of breath, medication schedule and effects or side effects

PATIENT OUTCOMES

Adequate sleep and rest

- Verbalizes improvement in hours and quality of sleep
- Appears rested and more alert
- Need for daytime napping decreases

NURSING INTERVENTIONS	RATIONALES
• Assess sleep patterns	• Provide baseline assessment
• Assess for nocturia, dyspnea, orthopnea, PND, and fear of PND	• Assess for common issues associated with sleep disturbances
• Plan medication schedules to allow uninterrupted period and avoid waking up to use the bathroom	• Promote periods of uninterrupted sleep
• Avoid caffeine, smoking, and eating 2 hours before sleep	• Promote relaxation and sleep
• Encourage patient to elevate HOB	• Reduce pulmonary congestion and nighttime dyspnea
• Review how to summon for help during the night	• Reduce anxiety and fear that may disrupt sleep patterns

NURSING DIAGNOSIS

Deficient Knowledge related to unfamiliarity with pathology, treatment, and medications; lack of information literacy, ineffective teaching-learning in past hospitalizations, cognitive limitations, depression

PATIENT OUTCOME

Adequate knowledge of disease and treatment

- Patient or significant others and verbalize causes, treatment, and care related to HF

NURSING INTERVENTIONS	RATIONALE
• Assess knowledge of causes, treatment, and care related to HF as well as best learning style (i.e., reading, listening, demonstration, etc.)	• Provide a base for educational planning
• Identify misconceptions regarding care	• Identify baseline knowledge and misperceptions that need to be corrected
• Educate about normal heart and circulation, HF disease process, symptoms, dietary modifications, activity guidelines, medications, psychological aspects of illness, goals of therapy, and community resources	• Reduce symptoms and readmission for exacerbation
• Use teach-back methods and encourage questions	• Verify understanding of information

ACE, Angiotensin converting enzyme; *ARB,* angiotensin II receptor blocker; *BP,* blood pressure; *CO,* cardiac output; *HF,* heart failure; *HOB,* head of bed; *HR,* heart rate; *JVD,* jugular venous distention; *PND,* paroxysmal nocturnal dyspnea.

Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes,* 7th ed. St. Louis, Mosby; 2011.

Interventions

Medical and nursing interventions for the patient with HF consist of a threefold approach: (1) treatment of the existing symptoms, (2) prevention of complications, and (3) treatment of the underlying cause. For example, some patients with HF can be treated by controlling hypertension or by repairing or replacing abnormal heart valves.

Treatment of existing symptoms includes the following:

1. *Improve pump function, fluid removal, and enhanced tissue perfusion* (Tables 12-12 and 12-13)[11]
   a. First-line medications include ACE inhibitors, angiotensin receptor blockers, and diuretics. Once symptoms and volume status are stable, a beta-blocker (metoprolol, carvedilol, bisoprolol) should be added. An ACE inhibitor and beta-blocker form the cornerstone of the treatment for HF.[11] Angiotensin receptor blockers (candesartan and valsartan) are also indicated even before trying an ACE inhibitor.
   b. Additional drug therapies include digoxin, spironolactone, eplerenone, hydralazine, and nitrates.
   c. Inotropes—dobutamine, dopamine, and milrinone—have failed to demonstrate improved mortality in the treatment of severe decompensated HF although they may improve symptoms at end of life.[11]
   d. Nesiritide (Natrecor) is administered IV to patients with acutely decompensated HF who have dyspnea at rest or with minimal activity. The best candidates for therapy are those who have clinical evidence of fluid overload, increased central venous pressure, or both.[12]
2. *Reduce cardiac workload and oxygen consumption*
   a. The intraaortic balloon pump is an invasive strategy to preserve coronary flow in the presence of severe, acute decompensated HF. It is used to stabilize patients with marked hemodynamic instability to allow time for insertion of a left ventricular assist device (LVAD).[27]
   b. LVADs are capable of partial to complete circulatory support for short- to long-term use. They assist the failing heart and maintain adequate circulatory pressure. LVADs attach to the patient's own heart and leave the patient's heart intact, and they have the potential for removal. At present, the LVAD is therapy for patients with terminal HF and has been used in patients who are not eligible for heart transplant.[12]
   c. Biventricular pacing. Patients with chronic HF may exhibit dyssynchronous contraction of the left ventricle, resulting from abnormal electrical conduction pathways. The abnormality leads to increased symptoms of heart failure. Cardiac resynchronization therapy through biventricular pacing involves placing a ventricular lead in the right ventricle and another lead down the coronary sinus to the left ventricle. Both ventricles are stimulated simultaneously, resulting in a synchronized contraction that improves cardiac performance and exercise tolerance as well as decreasing hospitalizations and mortality.[8]
   d. Nursing measures that reduce cardiac workload and oxygen consumption are to schedule rest periods and to encourage patients to modify their activities of daily living. Activity is advanced as tolerated. Patients with HF derive tremendous benefit from formal cardiac rehabilitation to improve activity tolerance and endurance.
3. *Optimize gas exchange through supplemental oxygen and diuresis*
   a. Evaluate the airway, the degree of respiratory distress, and the need for supplemental oxygenation by pulse oximetry, arterial blood gas measurement, or both. Patients are more comfortable in semi-Fowler position. Adjust oxygen delivery. Consider noninvasive ventilatory support such as continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP). CPAP and BiPAP have demonstrated effectiveness in the management of HF and often reduce the need for intubation.[11]
   b. Diurese aggressively. Administer IV diuretics; furosemide and bumetanide are the preferred diuretics. Ethacrynic acid is useful if the patient has a serious sulfa allergy. Torsemide is also used. These agents are characterized by quick onset; diuresis is expected 15 to 30 minutes after administration. Intravenous loop diuretic administration is preferred over intravenous thiazide diuretic administration. The goal is to achieve euvolemia, which may take days. When the patient is euvolemic, oral medications are restarted.
   c. Patients with severe HF are often considered at high risk for thromboembolic events due to stasis of blood in the atria and ventricles as well as venous stasis due to poor circulation. Anticoagulation is often used in these patients for those reasons, however, the research is lacking in support of this[11,17]
   d. Control of sodium and fluid retention involves fluid restriction of 2 L/day and sodium restriction of 2 g/day. Sodium restriction alone may provide substantial benefits for patients with HF.[11] Dietary counseling includes a discussion about fluid balance management and the importance of avoiding excess sodium or water intake, or both. Referral to a dietician should be considered for all patients.
   e. Daily weights are a priority in these patients.

Nurses make a tremendous impact by teaching and enforcing these concepts throughout the hospital stay. Patients may find it easier to continue these habits at discharge if their importance is stressed throughout hospitalization (see box, "QSEN Exemplar").

TABLE 12-12 MEDICATION SUBSETS FOR HEART FAILURE

MEDICATION	MANAGEMENT OF HEART FAILURE
ACE inhibitors	Slow disease progression, improve exercise capacity, and decrease hospitalization and mortality
Angiotensin II receptor antagonists	Reduce afterload and improve cardiac output. Can be used for patients with ACE-inhibitor cough
Hydralazine/Isosorbide dinitrate	Vasodilator effect; useful in patients intolerant to ACE inhibitors
Diuretics	Manage fluid overload
Aldosterone antagonists	Manage HF associated with LV systolic dysfunction (<35%) while receiving standard therapy, including diuretics
Digoxin	Improve symptoms, exercise tolerance, and quality of life; no effect on mortality
Beta-blockers	Manage HF associated with LV systolic dysfunction (<40%); well tolerated in most patients, including those with comorbidities such as diabetes mellitus, chronic obstructive lung disease, and peripheral vascular disease

ACE, Angiotensin-converting enzyme; *HF*, heart failure; *LV*, left ventricular.

TABLE 12-13 PHARMACOLOGY

Specific Medications for Heart Failure

Angiotensin-Converting Enzyme Inhibitors (ACE-Is)

Indications: used to treat hypertension, heart failure, and patients after myocardial infarction

Mechanism of action: prevent the conversion of angiotensin I to angiotensin II resulting in lower levels of angiotensin II, thus causing an increase in plasma renin activity and a reduction of aldosterone secretion; also inhibit the remodeling process after myocardial injury

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Enalapril (Vasotec)	*PO:* 2.5-20 mg BID	Hypotension, bradycardia, renal impairment, cough, and orthostatic hypotension. Do not give IV enalapril to patients with unstable heart failure or acute myocardial infarction. Monitor urine output and potassium levels. Avoid use of NSAIDs. Instruct patient to avoid rapid change in position such as from lying to standing. Contraindicated in pregnancy.
Fosinopril (Monopril)	*PO:* 10-40 mg daily	Same
Captopril (Capoten)	*PO:* 6.25-100 mg TID	Same

Diuretics

Indication: for the management of edema or fluid volume overload associated with heart failure and hepatic or renal disease

Mechanism of action: inhibit reabsorption of sodium and chloride in the ascending loop of Henle and distal renal tubule, interfering with the chloride-binding cotransport system, causing increased excretion of water, sodium, chloride, magnesium, and calcium

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Furosemide (Lasix)	*PO/IV:* 20-600 mg BID	Orthostatic hypotension, vertigo, dizziness, gout, hypokalemia, cramping, diarrhea or constipation, hearing impairment, tinnitus (rapid IV administration). Monitor laboratory results, especially potassium levels. Monitor cardiovascular and hydration status regularly. In decompensated patients, use IV route until euvolemic status is reached. Administer first dose early in the day and second dose late in afternoon, to prevent sleep disturbance.
Bumetanide (Bumex)	*PO/IV/IM:* 0.5-10 mg daily	Same
Torsemide (Demadex)	*PO/IV:* 10-200 mg daily Maximum 200 mg daily	Same
Metolazone (Zaroxolyn)	*PO:* 5-20 mg daily	Increased diuretic effect occurs when it is given with furosemide and other loop diuretics. Administer 30 minutes before IV loop diuretic.
Ethacrynic acid (Edecrin)	*PO:* 50-200 mg daily	Same Used when patient has a sulfa allergy.

Continued

TABLE 12-13 PHARMACOLOGY

Specific Medications for Heart Failure—cont'd

Beta-Blockers

Indications: used to treat angina, AMI, and heart failure

Mechanism of action: block beta-adrenergic receptors, with resulting decreased sympathetic nervous system responses such as decreases in heart rate, blood pressure, and cardiac contractility in heart failure may improve systolic function over time

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Metoprolol (Lopressor)	*PO:* 50-450 mg daily *IV:* 5 mg IVP *PO Toprol XL:* 25-200 mg daily	Bradycardia, hypotension, atrioventricular blocks, asthma attacks, fatigue, impotence, may mask hypoglycemic episodes. Teach patient to take pulse and blood pressure on regular basis. Patient should not abruptly stop taking these drugs. Close glucose monitoring if the patient is diabetic. Patients should be started on the lowest dose and slowly titrated to the maximum dose over 4-6 weeks to relieve symptoms.
Carvedilol (Coreg)	*PO:* 12.5-50 mg daily	Same Better tolerated on a full stomach.
Bisoprolol (Concor)	*PO:* 2.5-20 mg daily	Same

Aldosterone Receptor Antagonist

Indication: management of edema associated with excessive aldosterone secretion

Mechanism of action: competes with aldosterone for receptor sites in distal renal tubules, increasing sodium chloride and water excretion while conserving potassium and hydrogen ions; may block the effect of aldosterone on arterial smooth muscle

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Spironolactone (Aldactone)	*PO:* 25-200 mg daily	Monitor serum potassium and renal function; drug is potassium sparing.
Eplerenone (Inspra)	*PO:* 50 mg daily; increase to 50 mg BID if inadequate response after 4 weeks	Monitor blood pressure closely, especially at 2 weeks. Monitor potassium and sodium levels.

Inotropes

Indication: treatment of cardiac decompensation from heart failure, shock, or renal failure

Mechanism of action: augment cardiac output by increasing contractility and enhancing tissue perfusion; agents listed use different mechanisms

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Digoxin (Lanoxin)	*PO/IV:* 0.125-0.5 mg daily	Heart block, asystole, visual disturbances (blurred or yellow vision), confusion/mental disturbances, nausea, vomiting, diarrhea. Monitor serum concentrations; digoxin possesses a narrow therapeutic range and toxicity can be life-threatening. Maintain serum potassium levels; hypokalemia increases risk of digoxin toxicity. Monitor heart rate and notify provider if rate is <50 beats/min. Treatment of digoxin toxicity is digoxin immune fab (DigiFab).
Dopamine (Intropin)	*IV infusion:* 1-50 mcg/kg/min titrated to desired response Always administer into large vein via infusion device	Frequent ventricular ectopy, tachycardia, anginal pain, vasoconstriction, headache, nausea, or vomiting. Extravasation into surrounding tissue can cause tissue necrosis and sloughing. Monitor heart rate/rhythm and blood pressure closely. Dopamine is frequently used to treat hypotension because of its peripheral vasoconstrictor action. It is often used with dobutamine. Thus blood pressure is maintained by increased cardiac output (dobutamine) and vasoconstriction (dopamine). Monitor the IV site frequently.
Dobutamine (Dobutrex)	*IV infusion:* 2.5-40 mcg/kg/min titrated to desired response Always administer into large vein via infusion device	Increased heart rate, ventricular ectopy, hypotension, angina, headache, nausea, and local inflammatory changes. Drug has been used in outpatient settings (continuous at home or intermittent infusions in office) in patients with end-stage heart failure to stabilize symptoms. Monitor heart rate/rhythm and blood pressure closely.

TABLE 12-13 PHARMACOLOGY

Specific Medications for Heart Failure—cont'd

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Milrinone (Primacor)	*IV:* Loading dose of 50 mcg/kg over 10 minutes, followed by continuous infusion 0.375-0.75 mcg/kg/min Always administer into large vein via infusion device	Same as dobutamine
Inamrinone (Inocor)	*IV:* Loading dose of 0.75 mg/kg over 2 to 3 minutes, followed by continuous infusion of 5 to 10 mcg/kg/min May give additional bolus of 0.75 mcg/kg/min 30 minutes after starting therapy Do not exceed total daily dose of 10 mg/kg	Same as dobutamine Do not administer furosemide and inamrinone through the same IV line because precipitation occurs

Brain Natriuretic Peptide

Indication: decompensated congestive heart failure

Mechanism of action: exogenous form of hormone produced by myocardial myocytes as a result of myocardial stress and stretching; vasodilates both veins and arteries and has a positive neurohormonal effect by decreasing aldosterone, and positive renal effects by increasing diuresis and natriuresis

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Nesiritide (Natrecor)	*IV:* Loading dose of 2mcg/kg followed by infusion of 0.01 mcg/kg/min	Hypotension, enhanced diuresis, electrolyte imbalances (hypokalemia) Patients will usually respond quickly to therapy. Infusions generally run for 24 hours but can continue for days in the severely decompensated patient

Nitrates

Indications: to reduce afterload, elevated systemic vascular resistance

Mechanism of action: directly relax smooth muscle, which causes vasodilation of the peripheral vascular bed; decrease myocardial oxygen demands

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Nitroglycerin (Tridil)	*IV infusion:* 5 mcg/min, titrated to a maximum of 200 mcg/min	Headache, dizziness, flushing, orthostatic hypotension Monitor blood pressure closely. Titrate to effect

Angiotensin Receptor Blockers

Indications: hypertension, heart failure; used in patients who cannot tolerate use of ACE-Is

Mechanism of action: selective and competitive angiotensin II receptor antagonists; block the vasoconstrictor and aldosterone-secreting effects of angiotensin II

MEDICATION	DOSE/ROUTE	SIDE EFFECTS AND NURSING CONSIDERATIONS
Valsartan (Diovan)	*PO:* 40 mg daily BID up to 320 mg total daily dose	Hypotension, diarrhea, dyspepsia, upper respiratory infection Avoid use of NSAIDs, such as indomethacin or naproxen, which may cause renal impairment.
Candesartan (Atacand)	*PO:* 4-32 mg daily or BID	Same

BID, Twice daily; *IM*, intramuscular; *IV*, intravenous; *IVP*, intravenous push; *NSAIDs*, nonsteroidal antiinflammatory drugs; *PO*, orally; *TID*, three times daily.

QSEN EXEMPLAR

Patient-Centered Care

Clinical Practice Exemplar

Heart failure is associated with a high readmission rate. Assisting patients to make decisions to treat symptoms based on objective criteria may assist in reducing readmissions. The Agency for Healthcare Research and Quality (AHRQ) has recommended that discharge be reengineered and has developed a training module called "Project RED" (Re-Engineered Discharge). As part of the training, it is recommended that nurses use the Teach-Back technique when educating patients. A group of clinical nurse specialists incorporated the Teach-Back technique into a training program designed to improve nurses' ability to educate patients about heart failure. Using role play and practice, nurses' skills in using the Teach-Back improved after the training and they are now using the technique in their discharge teaching for heart failure patients. The technique should be incorporated into all education, because it is designed as a patient-centered, standardized approach to discharge planning.

The Teach-Back technique emphasizes the following points:

- Do not ask a patient, "Do you understand?" or yes/no questions
- Ask patients to explain or demonstrate how they will undertake a recommended treatment or intervention
- Ask open-ended questions
- Assume that you have not provided adequate teaching if the patient does not explain correctly.
- Re-teach in a different way.

Reference

Project RED (Re-Engineered Discharge) Training Program. August 2011, Agency for Healthcare Research and Quality, Rockville, MD. http://www.ahrq.gov/qual/projectred/

Cardiac transplantation is a therapeutic option of last resort for patients with end-stage HF. Patients who have severe cardiac disability refractory to expert management and who have a poor prognosis for 6-month survival are optimal candidates. For many patients with symptomatic HF and ominous objective findings (ejection fraction <20%, stroke volume <40 mL, severe ventricular dysrhythmias), timing of the surgery is difficult. A further consideration may be the quality of life, which is a judgment made between the patient and physicians.

Once the crisis stage has passed and the patient is stabilized, the precipitating factors for the complications must be addressed and treated. Treatment consists of surgical or catheter-based interventions as addressed for a patient with a MI, such as CABG, PTCA or stent, and pharmacological therapy (ACE inhibitors, beta-blocker); valve replacement or repair for valvular heart disease; restoration of sinus rhythm if atrial fibrillation or flutter and tachydysrhythmias are present; and management of risk factors such as hypertension, hyperlipidemia, diabetes, and obesity. Compliance with medications and sodium restriction is continually and vigilantly readdressed.

Complications

Complications of HF can be devastating. Interventions must be provided to avoid extending the existing conditions or allowing the development of new, life-threatening complications. Two specific complications for which the patients are monitored are pulmonary edema and cardiogenic shock.

Pulmonary Edema

The failing heart is sensitive to increases in afterload. In some patients with HF, when systolic blood pressure is 150 mm Hg or higher, pulmonary edema will ensue. The pulmonary vascular system becomes full and engorged. The results are increasing volume and pressure of blood in pulmonary vessels, increasing pressure in pulmonary capillaries, and leaking of fluid into the interstitial spaces of lung tissue.

Pulmonary edema greatly reduces the amount of lung tissue space available for gas exchange and results in clinical symptoms of extreme dyspnea, cyanosis, severe anxiety, diaphoresis, pallor, and blood-tinged, frothy sputum. Arterial blood gas results indicate severe respiratory acidosis and hypoxemia.

Patients with persistent volume overload may be candidates for continuous IV diuretics, ultrafiltration, or hemodialysis.[11] Loop diuretics given as an IV bolus are considered along with an IV infusion. Furosemide is the most commonly used loop diuretic, with the dose adjusted upward if the patient is currently on oral doses. The diuretic effect occurs in 30 minutes, with the peak effect in 1 to 2 hours.[28] IV torsemide or bumetanide are alternative loop diuretics.

The pharmacological characteristics of loop diuretics are similar. Continuous infusion of loop diuretics is considered if the patient does not respond to intermittent dosing. In addition, combinations of diuretics with different mechanisms of action are considered. Thiazide diuretics such as metolazone are often added. Monitoring hourly urinary output assists in determining the effectiveness of the diuretic therapy.

Although diuretic therapy is important, it is also critical to lower the blood pressure and cardiac filling pressures. Intravenous NTG is administered and titrated until the blood pressure is controlled, resulting in a reduction in both preload and afterload.[11] Patients who do not demonstrate improvement in symptoms require more aggressive treatment. A NTG infusion is initiated at 10 to 20 mcg/min, and initial titration is in increments of 10 mcg/min at intervals of 3 to 5 minutes, guided by patient response. The maximum dose is 200 mcg/min. Other care requirements for the administration of NTG include the use of non–polyvinyl chloride tubing. If this tubing is not available and traditional polyvinyl chloride tubing must be used, then the initial dose for NTG starts at 25 mcg/min IV. Patients who do not respond to aggressive diuretics and nitroglycerin may be a candidate for nesitiride (Natrecor), a natriuretic peptide.[11]

Cardiogenic Shock

Cardiogenic shock is the most acute and ominous form of pump failure. Cardiogenic shock can be seen after a severe MI, with dysrhythmias, decompensated HF, pulmonary

embolus, cardiac tamponade, and ruptured abdominal aortic aneurysm. Often, the outcome of cardiogenic shock is death. Cardiogenic shock and its treatment are discussed in depth in Chapter 11. Outcomes for the patient with HF are included in the nursing care plan.

PERICARDITIS

Pericarditis is acute or chronic inflammation of the pericardium. It may occur as a consequence of AMI or secondary to kidney injury (uremic pericarditis), infection, radiation therapy, connective tissue diseases, or cancer.[25] The pericardium has an inner and outer layer with a small amount of lubricating fluid between the layers. When the pericardium becomes inflamed, the amount of fluid between the two layers increases (pericardial effusion). This squeezes the heart and restricts its action and may result in cardiac tamponade. Chronic inflammation can result in constrictive pericarditis, which leads to scarring. The epicardium may thicken and calcify (see Figure 12-1).

The patient with pericarditis usually has precordial pain; this pain frequently radiates to the shoulder, neck, back, and arm and is intensified during deep inspiration, movement, coughing, and even swallowing. Other signs and symptoms may include a pericardial friction rub, dyspnea, weakness, fatigue, a persistent temperature elevation, an increased white blood cell count and sedimentation rate, and an increased anxiety level.[25] Pulsus paradoxus may be noted while auscultating the blood pressure. Pain due to pericarditis is usually positional and pleuritic (worse with inspiration and cough).

Detection of a pericardial friction rub is the most common method of diagnosing pericarditis. The friction rub is usually heard best on inspiration with the diaphragm of the stethoscope placed over the second, third, or fourth intercostal spaces at the sternal border. It is best heard when the patient is leaning forward. Friction rubs have been described as grating, scraping, squeaking, or scratching sounds. This rubbing sound results from an increase in fibrous exudate between the two irritated pericardial layers.

The ECG is useful in confirming the diagnosis of pericarditis because it is abnormal in 90% of patients with acute pericarditis. There are diffuse concave ST-segment elevation and PR-segment deviations opposite to P-wave polarity. T waves progressively flatten and invert, with generalized T-wave inversions present in most or all leads.[25] In echocardiogram is also useful in diagnosis to visualize the effusion.

The treatment of patients with pericarditis involves relief of pain (analgesic agents or antiinflammatory agents, such as colchicine and ibuprofen), antibiotics if the causative agent is bacterial, and treatment of other systemic symptoms.[25]

Approximately 15 to 50 mL of fluid is in the pericardial space. Excess fluid compresses the heart chambers, limits the filling capacity of the heart, and may result in tamponade. Treatment of cardiac tamponade includes inserting a needle into the pericardial space to remove the fluid (pericardiocentesis). In extreme cases, surgery may be required to remove part of the pericardium (pericardial window).

ENDOCARDITIS

Infective endocarditis occurs when microorganisms circulating in the bloodstream attach onto an endocardial surface. It is caused by various microbes, and frequently involves the heart valves. Endocarditis is classified as one of three types: native valve endocarditis (NVE), acute and subacute; prosthetic valve endocarditis (PVE), early and late; and intravenous drug abuse (IVDA) endocarditis.[7] *Staphylococcus aureus* is the most common causative pathogen in endocarditis. *Streptococcus* and *Enterococcus* organisms are often seen in subacute NVE.[7] Certain preexisting heart conditions increase the risk of developing endocarditis: implantation of an artificial (prosthetic) heart valve, a history of previous endocarditis, and heart valves damaged by conditions such as rheumatic fever, congenital heart defects, or valve defects.[6]

Infectious lesions, referred to as *vegetation,* form on the heart valves. These lesions have irregular edges, creating a cauliflower-like appearance. The mitral valve is the most common valve to be affected.[6] The vegetative process can grow to involve the chordae tendineae, papillary muscles, and conduction system. Therefore the patient may have dysrhythmias or acute HF. Patients with IVDA endocarditis usually do not have underlying structural disease.

The clinical presentation of patients with acute infectious endocarditis includes high fever and shaking chills. Other clinical manifestations of endocarditis include night sweats, cough, weight loss, general malaise, weakness, fatigue, headache, musculoskeletal complaints, new murmurs, and symptoms of HF. Skin abnormalities associated with septic emboli may also be seen: Janeway lesions (lesions that are often hemorrhagic and present on the palms and soles), Osler nodes (red-purple lesions on fingers or toes), splinter hemorrhages, and Roth spots (retinal hemorrhages). Skin lesions are referred to as the peripheral stigmata of endocarditis.[20]

Treatment of endocarditis involves diagnosing the infective agent and treating with the appropriate intravenous antibiotics for 4 to 6 weeks. Valve replacement surgery may be indicated in severe cases.[20] Prevention is important, and antibiotic prophylaxis is recommended for high-risk patients prior to procedures.

VASCULAR ALTERATIONS

The aorta is the largest blood vessel in the body both in length and diameter. Shaped like a walking cane, the aorta is an artery that carries blood from the heart. It extends from the aortic valve to the abdomen.[6] Its many branches supply blood to all other areas of the body. The aorta is divided into the thoracic and abdominal aorta (Figure 12-15).

The thoracic aorta is divided into the ascending aorta, the aortic arch, and the descending aorta. The thoracic aorta begins at the aortic root, which supports the bases of the three aortic valve leaflets.[5] The round segment, or cane handle, is

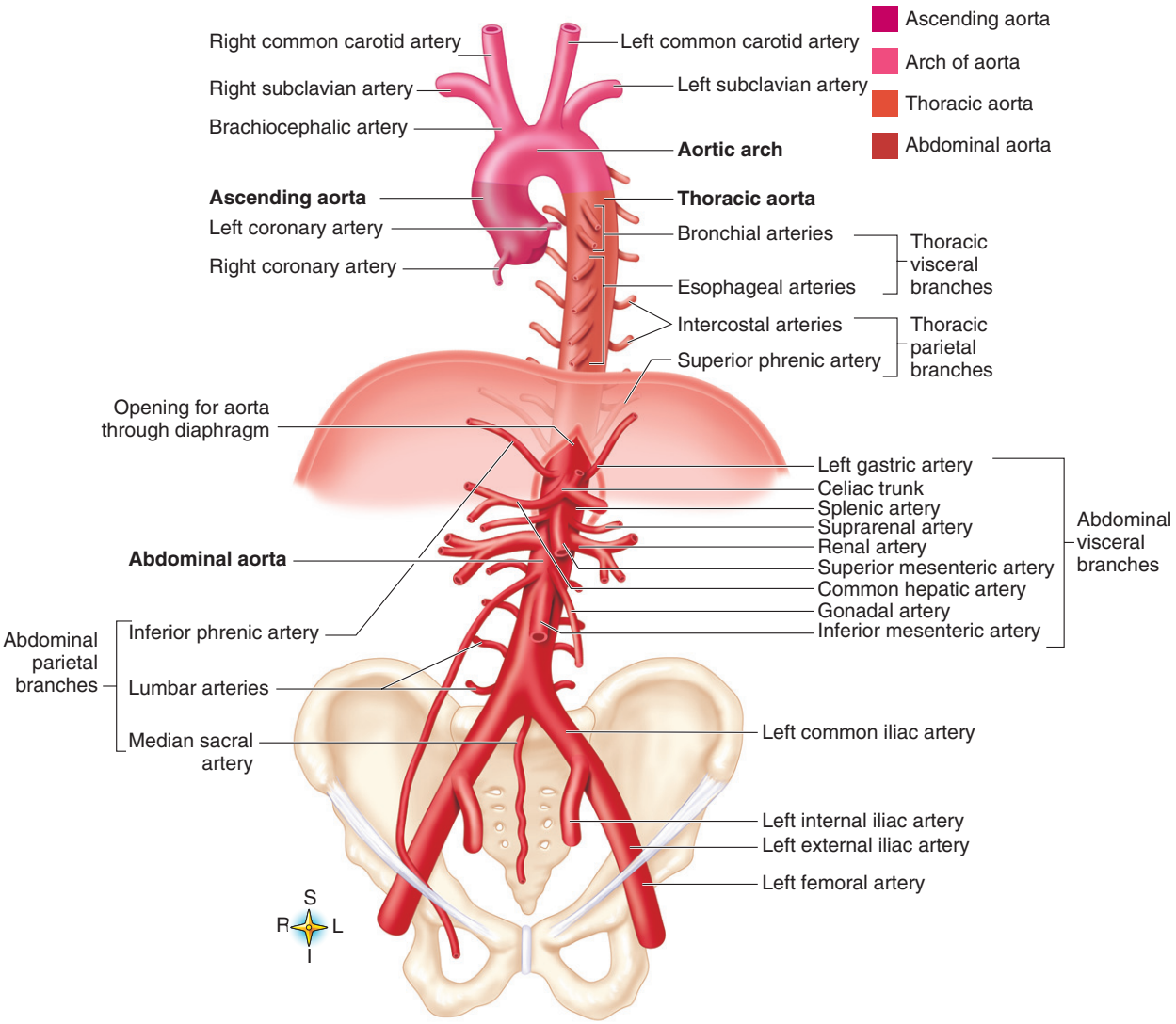

FIGURE 12-15 Anatomy of the aorta and its major branches. (From Patton KT, Thibodeau GA. *Anatomy and Physiology*. 8th ed. St. Louis: Mosby; 2013.)

the ascending aorta and the aortic arch. Branches of the ascending aorta include the right and left coronary arteries, which feed the myocardium. The arch vessels include the innominate artery, which branches into the right subclavian artery and right common carotid artery, and the left common carotid and left subclavian arteries. These branches send blood to the head and the upper extremities. The descending thoracic aorta, the long segment of the cane, is to the left of the midline of the chest. Branches of the descending aorta are the intercostal arteries. These arteries are the major blood supply to the distal spinal cord.

The abdominal aorta begins at the level of the diaphragm. At the umbilicus, it bifurcates into the iliac arteries. Abdominal branches include the celiac artery, the superior and inferior mesenteric arteries, and the renal arteries.

Aortic Aneurysms

The word *aneurysm* comes from the Greek *aneurysma,* which means "widening." An aneurysm is a diseased area of an artery causing dilatation and thinning of the wall. An aneurysm may be further classified as a false (pseudoaneurysm) or true aneurysm (Figure 12-16). A false aneurysm results from a complete tear in the arterial wall. Blood leaks from the artery to form a clot. Connective tissue is then laid down around this cavity. One example of a false aneurysm is an arterial wall tear resulting from an arterial puncture in the groin area. Anastomotic aneurysms are false aneurysms found at any graft-host artery anastomosis. True aneurysms include fusiform, saccular, and dissecting aneurysms. Fusiform or spindle-shaped aneurysms are generally found in the abdominal aorta and are the most common. A saccular aneurysm is a bulbous pouching of the artery usually found in the thoracic aorta.

Abdominal aortic aneurysms (AAA) are divided into thoracic aortic, thoracoabdominal aortic, and abdominal aortic types.

Atherosclerosis and degeneration of elastin and collagen are the underlying causes in most cases. They are also associated with certain connective tissue disorders such as Marfan syn-

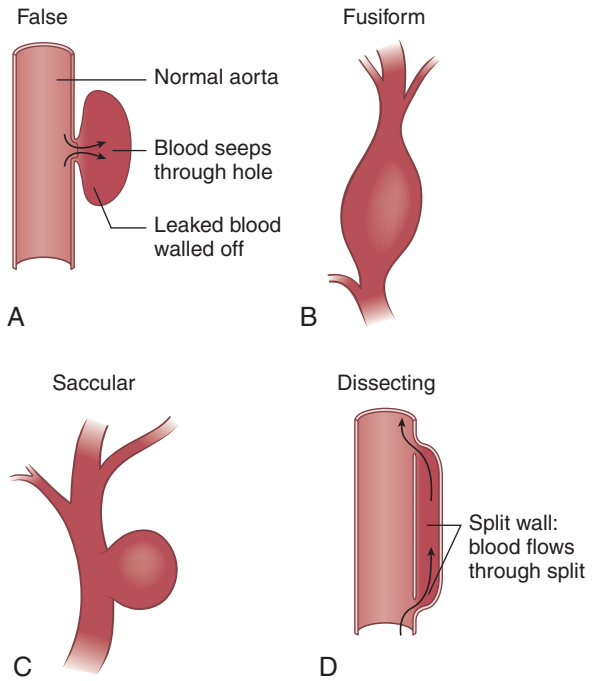

FIGURE 12-16 The four types of aneurysms. **A,** False. **B,** Fusiform. **C,** Saccular. **D,** Dissecting.

drome (see box, "Genetics: Marfan Syndrome"). Aneurysms are frequently hereditary, with a predominance in males. Risk factors of atherosclerosis such as age, smoking, hyperlipidemia, hypertension, and diabetes are also risk factors for aortic aneurysms.[18]

Most aneurysms are asymptomatic and are found on routine physical examination, or when testing for another disease entity. Back or abdominal pain may be noted with AAA. The goal of treatment is avoidance of rupture, which is dramatic and often fatal. Risk of rupture is related to the size of the aneurysm, with aneurysms larger than 6 cm carrying the greatest risk. Patients should be followed closely for changes in size of the aneurysm.

Treatment of an aneurysm is based on the symptoms of the patient and the size of the aneurysm. Thoracic aortic or thoracoabdominal aortic aneurysms larger than 5.5 to 6.0 cm, and abdominal aortic aneurysms 5.0 cm or larger are usually surgically repaired. Patients with smaller aneurysms are followed up diagnostically for any change in size. For

GENETICS

Marfan Syndrome

Advances in genetic disorders have resulted in new insights into the pathogenesis of disease, particularly in understanding the expression of proteins whose alteration or deficiency causes disease. One cardiovascular disease that illustrates this type of advance in the biologic basis of disease is Marfan syndrome (MFS). This single gene disorder demonstrates an autosomal dominant pattern of inheritance, which means that each child of an affected parent has a 50% chance of receiving a disease-causing gene variant. Further, it is highly penetrant, meaning that nearly all carriers develop the disease.[5] However, there is marked variation in phenotypes, even within families. New understanding of the molecular mechanisms underlying the pathogenesis of MFS helps explain the variety of abnormalities manifested.[2]

Most cases of MFS are caused by a mutation in the *FBN1* gene, located on chromosome 15. This gene codes for fibrillin-1, a glycoprotein that is found in both elastic and nonelastic tissues.[2] Fibrillin-1 helps form the complexes that anchor elastic fibers and regulates messengers associated with growth of long bones and cardiovascular tissue remodelling. Generally, it is missense mutations in the *FBN1* gene that alter a single amino acid out of the 2871 proteins that build fibrillin-1. The altered genetic code results in a changed structure, delayed secretion, or enhanced destruction of fibrillin-1. The reduction in effective fibrillin-1 results in connective-tissue weakness and explains the structural defects in the lens of the eyes, blood vessels, heart valves and skin. Altered regulation of growth factors explains bone overgrowth and skin and muscle hypoplasia in MFS.[4]

About 75% of MFS is inherited, but another 25% of individuals develop the disease from de novo mutations, meaning that the mutation occurred in the egg or sperm or in the early embryo.[2] Whether inherited or a new alteration, variation exists in the number and severity of symptoms, despite inheriting a similarly altered gene. Thus, despite high penetrance, the presence of a *FBN1* MFS variant gene does not predict the onset or severity of disease. The diagnosis of MFS is based on the presence of a positive family history and the number of organ systems with MFS-related defects. Genetic testing is available from a wide variety of laboratories[6] and mutations of the *FBN1* gene can be detected in more than 90% of patients with MFS.[7] While genetic testing is sensitive to MFS, it is not specific, in that *FBN1* variations are associated with other hereditary connective tissue disorders such as bicuspid aortic valve or Ehlers-Danlos syndrome.[2]

The phenotypes—clinical manifestations—that are most commonly associated with MFS are aortic aneurysm (especially thoracic); dilation of the root of the aorta where it is connected to the left ventricle; tall stature with especially long arms, legs, fingers, and toes; a protruding or indented sternum; enlargement of the dural membrane surrounding the lower spine or brainstem (i.e., dural ectasia); and discoloration of the lens of the eye. Individuals can also present with blebs or emphysema-like changes in the lung with possible spontaneous pneumothorax, inguinal or incisional hernias, skin stretch marks (i.e., striae), joint hypermobility, and visual disorders such as myopia or early cataracts.[1,5] Aortic aneurysm and dissection in MFS is associated with high morbidity and mortality.

Management of MFS is tailored to each individual's manifestation of the disease. For individuals who have cardiovascular manifestations of MFS, interventions typically include beta-blockers and angiotensin converting enzyme inhibitors or selective angiotensin-2 receptor blockers to control blood pressure and slow the progression of widening in the aorta.[3,4] Clinicians also advise individuals to restrict contact sports and weight lifting. Definitive treatment is surgical intervention on the aortic

Continued

GENETICS

Marfan Syndrome—cont'd

root and ascending aorta to prevent dissection. Patients with MFS and their family members may be referred by clinicians for genetic testing and genetic counseling, including psychosocial support and risk assessment.

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patients with small aortic abdominal aneurysms, smoking cessation is emphasized because there is evidence that smoking leads to faster expansion and rupture of an aneurysm.[5,14]

Aortic Dissection

Aortic dissection is a life-threatening emergency that requires immediate medical attention. Dissection is a tear in the intimal layer of the vessel creating a "false" lumen, causing blood flow diversion into the false lumen. Sudden, severe chest pain is the most common presenting symptom of aortic dissection. Dissections are divided into two categories: Stanford type A (proximal) and type B (distal). Type A is more concerning because it involves the ascending aorta and therefore dissection can extend into the coronary and arch vessels. It usually presents as severe anterior chest pain. Type B is confined to the descending thoracic and abdominal aorta and is often associated with pain between the scapulae. Ascending dissections are more common in younger patients, especially those with Marfan syndrome. Immediate treatment is directed at controlling blood pressure to 100 to 120 mm Hg, and decreasing the force of contraction of the heart. Therefore beta-blockers are the initial pharmacological treatment of choice. Emergency surgery is warranted to prevent death. Once rupture occurs, the overall 30-day survival rate is only 11%.[14,18]

Nursing Assessment

Knowledge of anatomy is the key factor in the treatment and care of patients with aortic aneurysms. Presentation of symptoms, intraoperative risk, and postoperative care are often location dependent. Blood flow to aortic branches may be hindered by the aneurysm itself, or embolization of thrombus may cause signs and symptoms such as chest pain, transient ischemic attacks, arm paresthesia with arch location, transient paralysis with descending aorta involvement, or abdominal or flank pain with AAA. In addition, systolic blood pressure may be different in each arm if the dissection occludes one of the subclavian arteries. A murmur may be auscultated if the dissection results in aortic regurgitation.[18]

Diagnostic Studies

1. *Physical examination.* Disparity in blood pressure measurements may be noted between the right and left arms or between the arms and legs, or a diminished pulse may be found in one of the limbs. Palpation reveals decreased or absent peripheral pulses. The patient may have a history of paresthesia, transient ischemic attacks, lower extremity or buttock claudication, and/or back or abdominal pain.
2. *Imaging studies.* Abdominal ultrasound, Computed tomography, angiography, TEE, and MRI are accurate diagnostic tools for abdominal aneurysms.

Treatment

Open surgical or endovascular repair is the treatment for large aortic aneurysms.[14] The open or conventional repair of aortic aneurysm is the endoaneurysmal repair (Figure 12-17). This surgery requires a midline, transverse anterior, or a retroperitoneal approach.

Endovascular aneurysm repair (EVAR) or thoracic endovascular aneurysm repair (TEVAR) is less invasive and refers to percutaneous stent placement in the descending thoracic or thoracoabdominal aorta. Both method and approach depend on the surgeon's preference and the patient's anatomy.[1] Through a small opening in the exposed femoral artery, an intraluminal sheathed stent is introduced, placed, and deployed with fluoroscopic guidance. The repair is usually successful, with less than 2% of patients who have to be converted to open repair.[1] Care of the vascular surgery patient is detailed in Box 12-13.

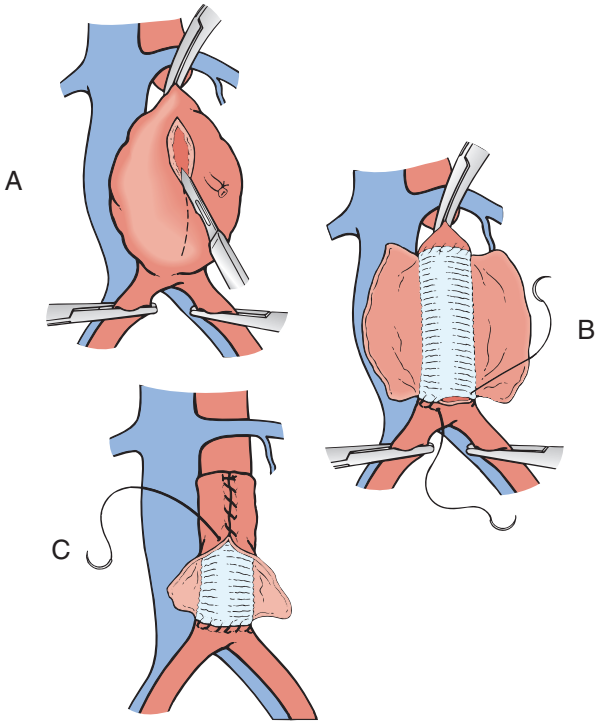

FIGURE 12-17 Surgical repair of an abdominal aortic aneurysm. **A,** The aneurysmal sac is incised. **B,** The synthetic graft is inserted. **C,** The native aortic wall is sutured over the synthetic graft (From Wipke-Tevis DD, Rich K, *Nursing Management Vascular Disorders.* In Lewis SL, et al. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby; 2011.)

BOX 12-13 NURSING INTERVENTIONS AFTER AORTIC SURGERY

- Monitor vital signs every 1 hour (postrecovery stage): pulse; assess for tachycardia and irregular rhythms.
- Blood pressure: keep the patient normotensive; hypertension causes bleeding; give vasodilators per protocol. Hypotension causes organ ischemia; give fluids and vasoconstrictors.
- Monitor hemodynamic pressures: SvO_2, stroke index, cardiac index; PAOP and RAP; and treat per protocols.
- Assess for hypovolemia: monitor output from chest tubes, drains, and urine output every 1 hour.
- Assess for hypothermia: rewarm the patient per protocol.
- Monitor fluid and electrolytes, hemoglobin, hematocrit, renal function, and coagulation studies.
- Monitor the radial, dorsalis pedis, and posterior tibial pulses every 1 hour; use Doppler studies as needed. Assess the ankle-brachial index every 2 hours or as ordered.
- Monitor for complications: intraoperative AMI, dysrhythmias, heart failure, cardiac tamponade, thromboembolism, impaired renal function, pneumonia, pneumothorax, pleural effusion, cerebral ischemia, or stroke.
- Implement ventilator bundle of care (see Chapter 9), wean from mechanical ventilation and extubate as soon as possible; promote pulmonary hygiene.
- Assess wounds and provide incisional care per protocol.
- Organize nursing care; control environmental stimuli.
- Gradually increase the patient's activity.
- Provide emotional support to the patient and family; assess the family's level of understanding; discuss the postoperative course.
- For abdominal aneurysm, assess for ischemic colitis

PAOP, Pulmonary artery occlusion pressure; *RAP*, right atrial pressure; *SvO_2*, mixed venous oxygen saturation.

GERIATRIC CONSIDERATIONS

The geriatric cardiac patient needs special considerations when planning and implementing care. Many older patients react differently and with more sensitivity to medications, procedures, and other modes of treatment. Some areas of special consideration include the following:

Medications

Great caution must be exercised when administering any medication to a geriatric patient, especially cardiac medications. Elderly persons may have greater sensitivity to these medications, they may not require the usual recommended dosage, or they may require more if they have been taking the medication in question for a long period of time. Monitor the patient closely for signs of drug effectiveness, adverse reactions, and possible interactions with other medications.

Procedures

The geriatric patient may need more information, support, and attendance during diagnostic or treatment procedures. Always having someone in attendance is a major consideration. It is also necessary to answer any and all questions to the extent needed for understanding and compliance. Frequent repetition and intentionality to reassessment in the elderly patient will emphasize the importance of teaching.

Surgery

Cardiac surgery is a major stress factor for anyone. The geriatric patient needs special attention to answer questions at the appropriate level of understanding and to provide the support for a very stressful, life-threatening procedure. Information and education are important, but be cautious of overwhelming the patient and causing greater stress.

Postoperative

The geriatric patient has special needs in the postoperative period. The aging patient has a natural physiological process of gradually diminished circulation. Anesthesia and a major surgical procedure add to this problem area and warrant careful monitoring and continuous assessment.

Family

It is imperative to have the involvement of family members or close friends. This can add a stabilizing factor that elderly patients need as they adjust to changes in treatment, activity, diet, medications, and ability to maintain activities of daily living.

Rehabilitation

Rehabilitation is important for any cardiac patient, whether after a myocardial infarction or after surgery. The geriatric patient needs extra encouragement to adhere to the set regimen to progress to maximum cardiac and vascular function.

CASE STUDY

Mr. S. was admitted to the emergency department (ED) by the emergency medical service with a complaint of sudden onset of substernal chest pain while he was mowing his lawn. The paramedics placed Mr. S. on oxygen at 2 L/min by nasal cannula. They started an 18-gauge intravenous (IV) line in his left antecubital area with normal saline at keep open rate. They gave Mr. S. aspirin and three sublingual nitroglycerin tablets every 5 minutes en route. Mr. S. states that his pain has gone from a 7, on a scale from 0 to 10, to a 3.

The ED nurse places Mr. S. on the cardiac monitor and notes that he is in sinus rhythm with frequent premature ventricular contractions (PVCs). The paramedic states that Mr. S. was diaphoretic, cool, and clammy on arrival of the emergency medical service at the scene. Mr. S. is warm and less clammy, although he is still quite pale. His blood pressure is 154/88 mm Hg, pulse is 95 beats per minute, and respiratory rate is 24 breaths per minute and nonlabored.

While awaiting the arrival of the ED physician to examine Mr. S., the nurse starts a second IV line, gives Mr. S. another nitroglycerin tablet, and proceeds to obtain a brief history from Mr. S.

Mr. S. is a 63-year-old white man, 220 lb, and he has been married for 41 years. He is hypertensive and diabetic, and he smokes 1½ packs of cigarettes per day. He is allergic to penicillin.

While the nurse is obtaining the history from Mr. S., the monitor alarms. She identifies the rhythm as ventricular fibrillation and begins cardiopulmonary resuscitation. The code team arrives, and Mr. S. is defibrillated with 200 J using the biphasic defibrillator. Following defibrillation, his rhythm is regular sinus with frequent PVCs. His blood pressure is 92/56 mm Hg, his pulse is thready, and he is diaphoretic. His pupils are 4 mm, equal and reactive. His respiratory rate is 16 breaths per minute and shallow, and his oxygen saturation is 92%. He has developed crackles in his lower and middle lung fields bilaterally. He is not fully awake at this time, but he is moving all his extremities. A 150-mg IV bolus of amiodarone is given over 10 minutes, and an infusion is started at 1 mg/min. Emergency laboratory tests and arterial blood gases are ordered, along with a 12-lead electrocardiogram (ECG). A request for an emergency consultation is placed to the cardiologist.

Mr. S.'s cardiac enzyme results return:

Creatine kinase (CK)	456 units/L
Creatine kinase–myocardial band (CK-MB)	52%
Troponin I	0.5 ng/mL
Troponin T	151 mcg/L

Electrolyte values are:

Sodium	143 mEq/L
Potassium	3.4 mEq/L
Chloride	109 mEq/L
Carbon dioxide	34 mEq/L
Glucose	354 mg/dL
Magnesium	1.5 mEq/L

Arterial blood gas values are:

pH	7.32
$PaCO_2$	49 mm Hg
PaO_2	77 mm Hg
Bicarbonate	24 mEq/L
SaO_2	92%

H&H:

Hemoglobin level	16.9 g/dL
Hematocrit	47.2%

A 12-lead ECG shows ST elevation in leads V_2, V_3, and V_4. Mr. S. is diagnosed with an acute anterior myocardial infarction. His oxygen is increased to 6 L/min by nasal cannula. Based on these assessment and study results, tissue plasminogen activator (t-PA) is administered.

Questions

1. What do Mr. S's cardiac enzyme values indicate about the time and extent of his myocardial infarction?
2. What would you expect his repeat troponin levels to be at the following times after his heart attack?
   a. At 8 hours
   b. At 12 hours
3. What complications may be anticipated for Mr. S. related to the infusion of t-PA? What parameters would the nurse need to monitor?
4. What assessments would indicate that the t-PA was effective?
5. What risk factors for CAD should be addressed before Mr. S.'s discharge to reduce his risk of another MI?

SUMMARY

This chapter focuses on the care of the patient with alterations in cardiovascular status. Geriatric patients are increasingly having medical and surgical interventions. They have even greater needs associated with the aging process (see box, "Geriatric Considerations"). The purpose of this chapter is to acquaint the critical care nurse with the problems and pathological conditions most commonly seen in the cardiovascular patient. This chapter is intended to provide a basic understanding of the cardiovascular patient that will facilitate sound clinical judgment in the planning of care that is holistic and incorporates a cooperative, interdisciplinary approach.

CRITICAL THINKING EXERCISES

1. You are taking care of a 58-year-old post-MI male patient readmitted to the unit because of recurrent chest pain, shortness of breath, and the need for intravenous nitroglycerin.
   a. Prioritize your actions at this time.
   b. What assessment findings regarding MI would concern you?
   c. What pertinent information from the patient's history would you want to obtain?
   d. What diagnostic tests do you anticipate?
2. Many patients now come into the hospital the same day that cardiac surgery is performed. Discuss methods for teaching patients effectively given this situation.
3. You are caring for a 63-year-old woman who has just returned to the cardiac care unit after PTCA and stent placement to the right coronary artery. Her proximal right coronary artery had a 90% occlusive lesion. She has her arterial sheath in place to the right femoral artery. She is receiving intravenous nitroglycerin and eptifibatide (Integrilin).
   a. What type of dysrhythmia would you anticipate if her right coronary artery were to reocclude?
   b. Prioritize your actions on her arrival.
   c. What type of assessment would you perform regarding the sheath?
4. A patient has been hospitalized three times in the past 2 months for chronic HF. What teaching and interventions can you implement to prevent rehospitalization after discharge?

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