CHAPTER 32 Nursing Care of Clients with Cardiac Disorders

LEARNING OUTCOMES

- Compare and contrast the etiology, pathophysiology, and manifestations of common cardiac disorders, including heart failure, structural disorders, and inflammatory disorders.

- Explain risk factors and preventive measures for cardiac disorders such as heart failure, inflammatory disorders, and valve disorders.

- Discuss indications for and management of clients undergoing hemodynamic monitoring.

- Discuss the effects and nursing implications for medications commonly prescribed for clients with cardiac disorders.

- Describe nursing care for the client undergoing cardiac surgery or cardiac transplant.

CLINICAL COMPETENCIES

- Apply knowledge of normal cardiac anatomy and physiology and assessment techniques in caring for clients with cardiac disorders.

- Assess functional health status of clients with cardiac disorders, documenting and reporting deviations for expected findings.

- Based on client assessment and knowledge of the disorder, determine priority nursing diagnoses.

- Plan, prioritize, and provide evidence-based, individualized care for clients with cardiac disorders.

- Safely and knowledgeably administer prescribed medications and treatments to clients with cardiac disorders.

- Actively participate in planning and coordinating interdisciplinary care for clients with cardiac disorders.

- Provide appropriate teaching and community-based care for clients with cardiac disorders and their families.

- Evaluate the effectiveness of nursing care, revising the plan of care as needed to promote, maintain, or restore functional health status of clients with cardiac disorders.

Cardiac disorders affect the structure and/or function of the heart. These disorders interfere with the heart's primary purpose: to pump enough blood to meet the body's demand for oxygen and nutrients. Disruptions in cardiac function affect the functioning of other organs and tissues, potentially leading to organ system failure and death.

Heart failure is the most common cardiac disorder. Other cardiac disorders discussed in this chapter include structural cardiac disorders, such as valve disorders and cardiomyopathy, and inflammatory cardiac disorders, such as endocarditis and pericarditis. Before continuing with this chapter, please review the heart's anatomy and physiology, nursing assessment, and diagnostic tests in Chapter 30 ∞.

HEART FAILURE

Heart failure is a complex syndrome resulting from cardiac disorders that impair the ventricles' ability to fill with and effectively pump blood (Hunt et al., 2005). In heart failure, the heart is unable to pump enough blood to meet the metabolic demands of the body. It is the end result of many conditions. Frequently, it is a long-term effect of coronary heart disease (CHD) and myocardial infarction (MI) when left ventricular damage is extensive enough to impair cardiac output (see Chapter 31 ∞). Other diseases of the heart also may cause heart failure, including structural and inflammatory disorders. In normal hearts, failure can result from excessive demands placed on the heart. Heart failure may be acute or chronic.

THE CLIENT WITH HEART FAILURE

As mentioned, heart failure develops when the heart cannot effectively fill or contract with adequate strength to function as a pump to meet the needs of the body. As a result, cardiac output falls, leading to decreased tissue perfusion. The body initially adjusts to reduced cardiac output by activating inherent compensatory mechanisms to restore tissue perfusion. These normal mechanisms may result in vascular congestion—hence, the commonly used term *congestive heart failure (CHF).* As these

mechanisms are exhausted, heart failure ensues, with increased morbidity and mortality.

Heart failure is a disorder of cardiac function. It frequently is due to *impaired myocardial contraction,* which may result from CHD and myocardial ischemia or infarct or from a primary cardiac muscle disorder such as cardiomyopathy or myocarditis. Structural cardiac disorders, such as valve disorders or congenital heart defects, and hypertension also can lead to heart failure when the heart muscle is damaged by the long-standing *excessive workload* associated with these conditions. Other clients without a primary abnormality of myocardial function may present with manifestations of heart failure due to *acute excess demands* placed on the myocardium, such as volume overload, hyperthyroidism, and massive pulmonary embolus (Table 32–1). Hypertension and coronary heart disease are the leading causes of heart failure in the United States. The high prevalence of hypertension in African Americans contributes significantly to their risk for and incidence of heart failure.

Incidence, Prevalence, and Risk Factors

Nearly 5 million people in the United States are currently living with heart failure; approximately 550,000 new cases of heart failure are diagnosed annually (American Heart Associa-

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TABLE 32–1 **Selected Causes of Heart Failure**

IMPAIRED MYOCARDIAL FUNCTION	INCREASED CARDIAC WORKLOAD	ACUTE NONCARDIAC CONDITIONS
■ Coronary heart disease	■ Hypertension	■ Volume overload
■ Cardiomyopathies	■ Valve disorders	■ Hyperthyroidism
■ Rheumatic fever	■ Anemias	■ Fever, infection
■ Infective endocarditis	■ Congenital heart defects	■ Massive pulmonary embolus

tion [AHA], 2005). Its incidence and prevalence increase with age: Less than 5% of people between ages 55 and 64 have heart failure, whereas 6% to 10% of people older than 65 are affected (see the box below) (AHA, 2005). The prevalence and mortality rate for heart failure is higher in African Americans than in whites. See the accompanying Cultural Diversity box.

Ischemic heart disease (coronary heart disease) is the leading risk factor for heart failure. Cardiomyopathies are the second leading cause of heart failure. Other, less common causes of heart failure are hypertension and congenital and valvular heart disease (Kasper et al., 2005).

The prognosis for a client with heart failure depends on its underlying cause and how effectively precipitating factors can be treated. Most clients with heart failure die within 8 years of the diagnosis. The risk for sudden cardiac death is dramatically increased, occurring at a rate six to nine times that of the general population (AHA, 2005).

Physiology Review

The mechanical pumping action of cardiac muscle propels the blood it receives to the pulmonary and systemic vascular systems for reoxygenation and delivery to the tissues. *Cardiac output (CO)* is the amount of blood pumped from the ventricles in 1 minute. Cardiac output is used to assess cardiac performance, especially left ventricular function. Effective cardiac output depends on adequate functional muscle mass and the ability of the ventricles to work together. Cardiac output normally is regulated by the oxygen needs of the body: As oxygen use increases, cardiac output increases to maintain cellular function. *Cardiac reserve* is the ability of the heart to increase CO to meet metabolic demand. Ventricular damage reduces the cardiac reserve.

Cardiac output is a product of heart rate and stroke volume. *Heart rate (HR)* affects cardiac output by controlling the num-

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FOCUS ON CULTURAL DIVERSITY

- Up to 5 million Americans have heart failure; of these, about 725,000 (15%) are African Americans.
- In African Americans:
    - Manifestations of heart failure develop at an earlier age.
    - The disease progresses more rapidly.
    - More hospital visits are attributed to heart failure.
    - The mortality rate is higher than in white men and women.

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ber of ventricular contractions per minute. It is influenced by the autonomic nervous system, catecholamines, and thyroid hormones. Activation of a stress response (e.g., hypovolemia or fear) stimulates the sympathetic nervous system, increasing the heart rate and its contractility. Elevated heart rates increase cardiac output. Very rapid heart rates, however, shorten ventricular filling time (diastole), reducing stroke volume and cardiac output. On the other hand, a slow heart rate reduces cardiac output simply because of fewer cardiac cycles.

Stroke volume, the volume of blood ejected with each heartbeat, is determined by preload, afterload, and myocardial contractility. *Preload* is the volume of blood in the ventricles at end diastole (just prior to contraction). The blood in the ventricles exerts pressure on the ventricle walls, stretching muscle fibers. The greater the blood volume, the greater the force with which the ventricle contracts to expel the blood. End diastolic volume depends on the amount of blood returning to the ventricles (*venous return*) and the distensibility or stiffness of the ventricles (*compliance*). See Box 32–1.

Afterload is the force needed to eject blood into the circulation. This force must be great enough to overcome arterial pressures

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NURSING CARE OF THE OLDER ADULT   Heart Failure

Heart failure is common in older adults, affecting nearly 10% of people over the age of 75 years.

Aging affects cardiac function. Diastolic filling is impaired by decreased ventricular compliance. With aging, the heart is less responsive to sympathetic nervous system stimulation. As a result, maximal heart rate, cardiac reserve, and exercise tolerance are reduced. Concurrent health problems such as arthritis that affect stamina or mobility often contribute to a more sedentary lifestyle, further decreasing the heart's ability to respond to increased stress.

Assessing for Home Care

The older adult with heart failure may not be dyspneic, instead presenting with weakness and fatigue, somnolence, confusion, disorientation, or worsening dementia. Dependent edema and respiratory crackles may or may not indicate heart failure in older adults.

Assess the diet of the older adult. Decreased taste may lead to increased use of salt to bring out food flavors. Limited mobility or visual acuity may cause the older adult to rely on prepared foods that are high in sodium such as canned soups and frozen

meals. Discuss normal daily activities and assess sleep and rest patterns. It is also important to assess the environment for:
- Safe roads or neighborhoods for walking
- Access to pharmacy, medical care, and assistive services such as a cardiac rehabilitation program or structured exercise programs designed for older adults.

Health Education for the Client and Family

Teaching for the older adult with heart failure focuses on maintaining function and promptly identifying and treating episodes of heart failure. Teach clients how to adapt to changes in cardiovascular function associated with aging, such as:
- Allowing longer warm-up and cool-down periods during exercise
- Engaging in regular exercise such as walking five or more times a week
- Resting with feet elevated (e.g., in a recliner) when fatigued
- Maintaining adequate fluid intake
- Preventing infection through pneumococcal and influenza immunizations.

BOX 32–1 Explaining Physiologic Terms Using Practical Examples

The concepts of preload, the Frank-Starling mechanism, compliance, and afterload can be difficult to understand and to explain to clients. Use common analogies to make these concepts easier to understand.

■ *Preload:* Think about a new rubber band. As you stretch the rubber band further, it snaps back into shape with greater force.
■ *Frank-Starling mechanism:* When you repeatedly stretch that rubber band beyond a certain limit, it loses some elasticity and fails to return to its original shape and size.
■ *Compliance:* Use a new rubber balloon to illustrate this concept. A new balloon is not very compliant—it takes a lot of work (force) to inflate it. As the balloon is repeatedly inflated and stretched, it becomes more compliant, expanding easily with less force.
■ *Afterload:* When a hose is crimped or plugged, more force is required to eject a stream of water out its end.

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within the pulmonary and systemic vascular systems. The right ventricle must generate enough force to open the pulmonary valve and eject its blood into the pulmonary artery. The left ventricle ejects its blood into the systemic circulation by overcoming the arterial resistance behind the aortic valve. Increased systemic vascular resistance (e.g., hypertension) increases afterload, impairing stroke volume and increasing myocardial work.

Contractility is the natural ability of cardiac muscle fibers to shorten during systole. Contractility is necessary to overcome arterial pressures and eject blood during systole. Impaired contractility affects cardiac output by reducing stroke volume. The *ejection fraction (EF)* is the percentage of blood in the ventricle that is ejected during systole. A normal ejection fraction is approximately 60%.

Pathophysiology

When the heart begins to fail, mechanisms are activated to compensate for the impaired function and maintain the cardiac output. The primary compensatory mechanisms are (1) the Frank-Starling mechanism, (2) neuroendocrine responses including activation of the sympathetic nervous system and the renin–angiotensin system, and (3) myocardial hypertrophy. These mechanisms and their effects are summarized in Table 32–2.

Decreased cardiac output initially stimulates aortic baroreceptors, which in turn stimulate the sympathetic nervous system (SNS). SNS stimulation produces both cardiac and vascular responses through the release of norepinephrine. Norepinephrine increases heart rate and contractility by stimulating cardiac beta-receptors. Cardiac output improves as both heart rate and stroke volume increase. Norepinephrine also causes arterial and venous vasoconstriction, increasing venous return to

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TABLE 32–2 Compensatory Mechanisms Activated in Heart Failure

MECHANISM	PHYSIOLOGY	EFFECT ON BODY SYSTEMS	COMPLICATIONS
Frank-Starling mechanism	The greater the stretch of cardiac muscle fibers, the greater the force of contraction.	■ Increased contractile force leading to increased CO	■ Increased myocardial oxygen demand ■ Limited by overstretching
Neuroendocrine response	Decreased CO stimulates the sympathetic nervous system and catecholamine release.	■ Increased HR, BP, and contractility ■ Increased vascular resistance ■ Increased venous return	■ Tachycardia with decreased filling time and decreased CO ■ Increased vascular resistance ■ Increased myocardial work and oxygen demand
	Decreased CO and decreased renal perfusion stimulate renin–angiotensin system.	■ Vasoconstriction and increased BP	■ Increased myocardial work ■ Renal vasoconstriction and decreased renal perfusion
	Angiotensin stimulates aldosterone release from adrenal cortex.	■ Salt and water retention by the kidneys ■ Increased vascular volume	■ Increased preload and afterload ■ Pulmonary congestion
	ADH is released from posterior pituitary.	■ Water excretion inhibited	■ Fluid retention and increased preload and afterload ■ Pulmonary congestion
	Atrial natriuretic peptide and brain natriuretic peptide are released.	■ Increased sodium excretion ■ Diuresis ■ Vasodilation	
	Blood flow is redistributed to vital organs (heart and brain).	■ Decreased perfusion of other organ systems ■ Decreased perfusion of skin and muscles	■ Renal failure ■ Anaerobic metabolism and lactic acidosis
Ventricular hypertrophy	Increased cardiac workload causes myocardial muscle to hypertrophy and ventricles to dilate.	■ Increased contractile force to maintain CO	■ Increased myocardial oxygen demand ■ Cellular enlargement

the heart. Increased venous return increases ventricular filling and myocardial stretch, increasing the force of contraction (the Frank-Starling mechanism). Overstretching the muscle fibers past their physiologic limit results in an ineffective contraction.

Blood flow is redistributed to the brain and the heart to maintain perfusion of these vital organs. Decreased renal perfusion causes renin to be released from the kidneys. Activation of the renin–angiotensin system produces additional vasoconstriction and stimulates the adrenal cortex to produce aldosterone and the posterior pituitary to release antidiuretic hormone (ADH). Aldosterone stimulates sodium reabsorption in renal tubules, promoting water retention. ADH acts on the distal tubule to inhibit water excretion and also causes vasoconstriction. The effect of these hormones is significant vasoconstriction and salt and water retention, with a resulting increase in vascular volume. Increased ventricular filling increases the force of contraction, improving cardiac output. The effects of the renin–angiotensin–aldosterone system and ADH release are counterbalanced to a certain extent by two additional hormones. The increased vascular volume and venous return prompted by vasoconstriction and sodium and water retention increase the volume and pressures in the heart. Stimulation of stretch receptors in the atria and ventricles lead to the release of *atrial natriuretic peptide (ANP)* and *brain natriuretic peptide (BNP)* from stores in the atria (ANP and BNP) and ventricles (BNP). These hormones promote sodium and water excretion and inhibit the release of norepinephrine, renin, and ADH, with resulting vasodilation. Although beneficial, the effects of these hormones are too weak to completely counteract the vasoconstriction and sodium and water retention that occurs in heart failure.

Ventricular remodeling occurs as the heart chambers and myocardium adapt to fluid volume and pressure increases. The chambers dilate to accommodate excess fluid resulting from increased vascular volume and incomplete emptying. Initially, this additional stretch causes more effective contractions. *Ventricular hypertrophy* occurs as existing cardiac muscle cells enlarge, increasing their contractile elements (actin and myosin) and force of contraction.

Although these responses may help in the short-term regulation of cardiac output, it is now recognized that they hasten the deterioration of cardiac function. The onset of heart failure is heralded by *decompensation,* the loss of effective compensation. Heart failure progresses due to the very mechanisms that initially maintained circulatory stability.

The rapid heart rate shortens diastolic filling time, compromises coronary artery perfusion, and increases myocardial oxygen demand. Resulting ischemia further impairs cardiac output. Beta-receptors in the heart become less sensitive to continued SNS stimulation, decreasing heart rate and contractility. As the beta-receptors become less sensitive, norepinephrine stores in the cardiac muscle become depleted. In contrast, alpha-receptors on peripheral blood vessels become increasingly sensitive to persistent stimulation, promoting vasoconstriction and increasing afterload and cardiac work.

Initially, ventricular hypertrophy and dilation increase cardiac output, but chronic distention causes the ventricular wall eventu-

ally to thin and degenerate. The purpose of hypertrophy is thus defeated. In addition, chronic overloading of the dilated ventricle eventually stretches the fibers beyond the optimal point for effective contraction. The ventricles continue to dilate to accommodate the excess fluid, but the heart loses the ability to contract forcefully. The heart muscle may eventually become so large that the coronary blood supply is inadequate, causing ischemia.

Chronic distention exhausts stores of ANP and BNP. The effects of norepinephrine, renin, and ADH prevail, and the renin–angiotensin pathway is continually stimulated. This mechanism ultimately raises the hemodynamic stress on the heart by increasing both preload and afterload. As heart function deteriorates, less blood is delivered to the tissues and to the heart itself. Ischemia and necrosis of the myocardium further weaken the already failing heart, and the cycle repeats.

In normal hearts, the cardiac reserve allows the heart to adjust its output to meet metabolic needs of the body, increasing the cardiac output by up to five times the basal level during exercise. Clients with heart failure have minimal to no cardiac reserve. At rest, they may be unaffected; however, any stressor (e.g., exercise, illness) taxes their ability to meet the demand for oxygen and nutrients. Manifestations of activity intolerance when the person is at rest indicate a critical level of cardiac decompensation.

Classifications and Manifestations of Heart Failure

Heart failure is commonly classified in several different ways, depending on the underlying pathology. Classifications include systolic versus diastolic failure, left-sided versus right-sided failure, high-output versus low-output failure, and acute versus chronic failure.

FAST FACTS

Terms used to describe or classify heart failure:
- Systolic or diastolic failure
- Left ventricular (or sided) or right ventricular (or sided) failure
- Low-output or high-output failure
- Acute or chronic failure
- Forward or backward effects.

Systolic versus Diastolic Failure

Systolic failure occurs when the ventricle fails to contract adequately to eject a sufficient blood volume into the arterial system. Systolic function is affected by loss of myocardial cells due to ischemia and infarction, cardiomyopathy, or inflammation. The manifestations of systolic failure are those of decreased cardiac output: weakness, fatigue, and decreased exercise tolerance.

Diastolic failure results when the heart cannot completely relax in diastole, disrupting normal filling. Passive diastolic filling decreases, increasing the importance of atrial contraction to preload. Diastolic dysfunction results from decreased ventricular compliance due to hypertrophic and cellular changes and impaired relaxation of the heart muscle. Its manifestations result from increased pressure and congestion behind the ventricle:

shortness of breath, tachypnea, and respiratory crackles if the left ventricle is affected; distended neck veins, liver enlargement, anorexia, and nausea if the right ventricle is affected. Many clients have components of both systolic and diastolic failure.

Left-Sided versus Right-Sided Failure

Depending on the pathophysiology involved, either the left or the right ventricle may be primarily affected. In chronic heart failure, however, both ventricles typically are impaired to some degree. Coronary heart disease and hypertension are common causes of *left-sided heart failure,* whereas *right-sided heart failure* often is caused by conditions that restrict blood flow to the lungs, such as acute or chronic pulmonary disease. Left-sided heart failure also can lead to right-sided failure as pressures in the pulmonary vascular system increase with congestion behind the failing left ventricle.

As left ventricular function fails, cardiac output falls. Pressures in the left ventricle and atrium increase as the amount of blood remaining in the ventricle after systole increases. These increased pressures impair filling, causing congestion and increased pressures in the pulmonary vascular system. Increased pressures in this normally low-pressure system increase fluid movement from the blood vessels into interstitial tissues and the alveoli (Figure 32–1 ■).
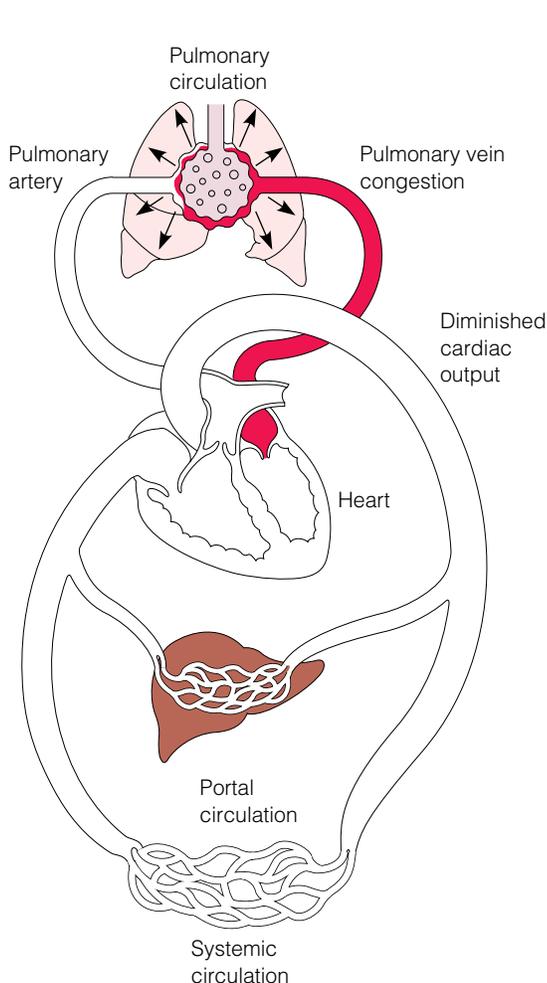
The manifestations of left-sided heart failure result from pulmonary congestion (*backward effects*) and decreased cardiac output (*forward effects*). Fatigue and activity intolerance are common early manifestations. Dizziness and syncope also may result from decreased cardiac output. Pulmonary congestion causes dyspnea, shortness of breath, and cough. The client may develop **orthopnea** (difficulty breathing while lying down), prompting use of two or three pillows or a recliner for sleeping. Cyanosis from impaired gas exchange may be noted. On auscultation of the lungs, inspiratory crackles (rales) and wheezes may be heard in lung bases. An S_3 gallop may be present, reflecting the heart's attempts to fill an already distended ventricle.

In right-sided heart failure, increased pressures in the pulmonary vasculature or right ventricular muscle damage impair the right ventricle's ability to pump blood into the pulmonary circulation. The right ventricle and atrium become distended, and blood accumulates in the systemic venous system. Increased venous pressures cause abdominal organs to become congested and peripheral tissue edema to develop (Figure 32–2 ■).

Dependent tissues tend to be affected because of the effects of gravity; edema develops in the feet and legs, or if the client
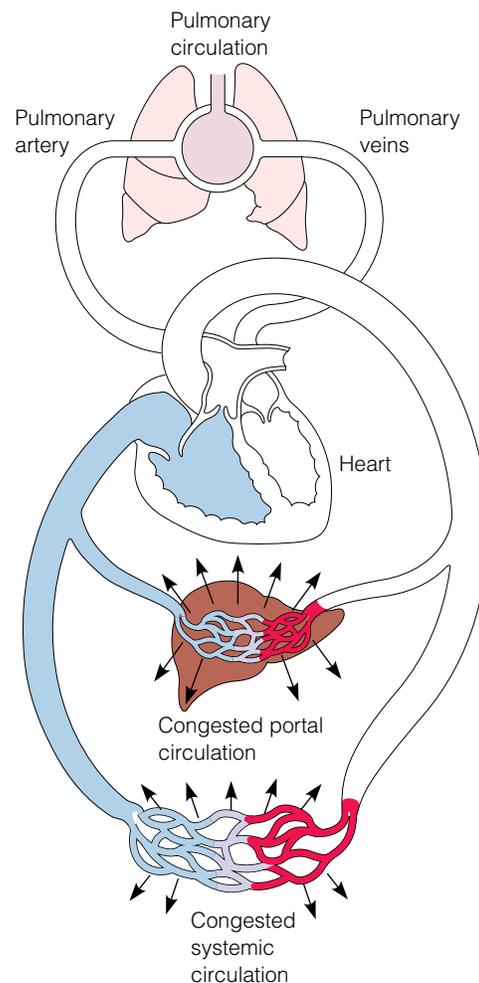

Figure 32–1 ■ The hemodynamic effects of left-sided heart failure.

Figure 32–2 ■ The hemodynamic effects of right-sided heart failure.

is bedridden, in the sacrum. Congestion of gastrointestinal tract vessels causes anorexia and nausea. Right upper quadrant pain may result from liver engorgement. Neck veins distend and become visible even when the client is upright, due to increased venous pressure.

Low-Output versus High-Output Failure

Clients with heart failure due to coronary heart disease, hypertension, cardiomyopathy, and other primary cardiac disorders develop *low-output failure* and manifestations such as those previously described. Clients in hypermetabolic states (e.g., hyperthyroidism, infection, anemia, or pregnancy) require increased cardiac output to maintain blood flow and oxygen to the tissues. If the increased blood flow cannot meet the oxygen demands of the tissues, compensatory mechanisms are activated to further increase cardiac output, which in turn further increases oxygen demand. Thus, even though cardiac output is high, the heart is unable to meet increased oxygen demands. This condition is known as *high-output failure.*

Acute versus Chronic Failure

Acute failure is the abrupt onset of a myocardial injury (such as a massive MI) resulting in suddenly decreased cardiac function and signs of decreased cardiac output. *Chronic failure* is a progressive deterioration of the heart muscle due to cardiomyopathies, valvular disease, or CHD.

Other Manifestations

In addition to the previous manifestations for the various classifications of heart failure, other signs and symptoms commonly are seen.

A fall in cardiac output activates mechanisms that cause increased salt and water retention. This causes weight gain and further increases pressures in the capillaries, resulting in edema. *Nocturia,* voiding more than one time at night, develops as edema fluid from dependent tissues is reabsorbed while the client is supine. **Paroxysmal nocturnal dyspnea (PND)**, a frightening condition in which the client awakens at night acutely short of breath, also may develop. Paroxysmal nocturnal dyspnea occurs when edema fluid that has accumulated during the day is reabsorbed into the circulation at night, causing fluid overload and pulmonary congestion. Severe heart failure may cause dyspnea at rest as well as with activity, signifying little or no cardiac reserve. Both an S_3 and an S_4 gallop may be heard on auscultation.

See page 1028 for the *Multisystem Effects of Heart Failure.*

Complications

The compensatory mechanisms initiated in heart failure can lead to complications in other body systems. Congestive hepatomegaly and splenomegaly caused by engorgement of the portal venous system result in increased abdominal pressure, ascites, and gastrointestinal problems. With prolonged right-sided heart failure, liver function may be impaired. Myocardial distention can precipitate dysrhythmias, further impairing cardiac output. Pleural effusions and other pulmonary problems may develop. Major complications of severe heart failure are cardiogenic shock (described in Chapter 11 ∞) and acute

pulmonary edema, a medical emergency described in the next section of this chapter.

INTERDISCIPLINARY CARE

The main goals for care of heart failure are to slow its progression, reduce cardiac workload, improve cardiac function, and control fluid retention. Treatment strategies are based on the evolution and progression of heart failure (Table 32–3).

Diagnosis

Diagnosis of heart failure is based on the history, physical examination, and diagnostic findings.

- *Atrial natriuretic peptide (ANP),* also called *atrial natriuretic hormone (ANH),* and *brain natriuretic peptide (BNP)* are hormones released by the heart muscle in response to changes in blood volume. Blood levels of these hormones increase in heart failure. BNP levels, in particular, have been shown to positively correlate with pressures in the left ventricle and the pulmonary vascular system. As the severity of left ventricular failure increases, BNP levels increase (White, 2005). It is important to remember, however, that BNP levels may be elevated in women and in people over age 60 who do not have heart failure. Therefore, an elevated BNP cannot be used alone to diagnosis heart failure (Hunt et al., 2005).
- *Serum electrolytes* are measured to evaluate fluid and electrolyte status. Serum osmolarity may be low due to fluid retention. Sodium, potassium, and chloride levels provide a baseline for evaluating the effects of treatment; serum calcium and magnesium are measured as well.
- *Urinalysis, blood urea nitrogen (BUN),* and *serum creatinine* are obtained to evaluate renal function.
- *Liver function tests* including ALT, AST, LDH, serum bilirubin, and total protein and albumin levels are obtained to evaluate possible effects of heart failure on liver function.
- Thyroid function tests, including TSH and TH levels, are obtained because both hyperthyroidism and hypothyroidism can be either a primary or a contributing cause of heart failure (Hunt et al., 2005).
- In acute heart failure, *arterial blood gases (ABGs)* are drawn to evaluate gas exchange in the lungs and tissues.
- *Chest x-ray* may show pulmonary vascular congestion and cardiomegaly in heart failure.
- *Electrocardiography* is used to identify ECG changes associated with ventricular enlargement and to detect dysrhythmias, myocardial ischemia, or infarction.
- *Echocardiography with Doppler flow studies* are performed to evaluate left ventricular function. Either *transthoracic echocardiography* or *transesophageal echocardiography* may be used. See Chapter 30 ∞ for more information and the nursing implications of these tests.
- *Radionuclide imaging* is used to evaluate ventricular function and size (see Chapter 30 ∞).

Hemodynamic Monitoring

Hemodynamics is the study of forces involved in blood circulation. Hemodynamic monitoring is used to assess cardiovascular function in the critically ill or unstable client. The main

Neurologic
- Confusion
- Impaired memory
- Anxiety, restlessness
- Insomnia

Respiratory
- Dyspnea on exertion
- Shortness of breath
- Tachypnea
- Orthopnea
- Dry cough
- Crackles (rales) in lung bases

Potential Complications
- Pulmonary edema
- Pneumonia
- Cardiac asthma
- Pleural effusion
- Cheyne-Stokes respirations
- Respiratory acidosis

Cardiovascular
- Activity intolerance
- Tachycardia
- Palpitations
- S_3, S_4 heart sounds
- Elevated central venous pressure
- Neck vein distention
- Hepatojugular reflux
- Splenomegaly

Potential Complications
- Angina
- Dysrhythmias
- Sudden cardiac death
- Cardiogenic shock

Gastrointestinal
- Anorexia, nausea
- Abdominal distention
- Liver enlargement
- Right upper quadrant pain

Potential Complications
- Malnutrition
- Ascites
- Liver dysfunction

Genitourinary
- Decreased urine output
- Nocturia

Integumentary
- Pallor or cyanosis
- Cool, clammy skin
- Diaphoresis

Potential Complications
- Increased risk for tissue breakdown

Musculoskeletal
- Fatigue
- Weakness

Metabolic Processes
- Peripheral edema
- Weight gain

Potential Complication
- Metabolic acidosis

TABLE 32–3 Stages of Heart Failure

STAGE	DESCRIPTION	RECOMMENDED TREATMENT MEASURES
A	Clients at high risk for developing heart failure, but without structural heart disease or symptoms of heart failure (clients with hypertension, CHD, diabetes, obesity, metabolic syndrome, or who have a family history of cardiomyopathy, or who are taking cardiotoxic drugs)	Treat underlying risk factors (e.g., hypertension) including lipid disorders ACE inhibitor or angiotensin-receptor blocker (ARB) therapy as appropriate Exercise Salt restriction Smoking cessation Discourage alcohol, illicit drug use Control blood glucose in clients with metabolic syndrome
B	Clients with structural heart disease but no manifestations of heart failure (clients with previous MI, asymptomatic valve disease, or left ventricular dysfunction)	As for stage A ACE inhibitor or ARB therapy as appropriate Beta-blocker therapy if indicated
C	Clients with structural heart disease and current or prior symptoms of heart failure (shortness of breath, fatigue, decreased exercise tolerance)	As for stages A and B Drug therapy with a diuretic, ACE inhibitor, and/or beta-blocker Additional drugs as indicated, such as an aldosterone antagonist, ARB, digitalis, hydralazine, nitrates Ventricular pacing or an implantable defibrillator (ICD) as indicated
D	Refractory heart failure (clients with manifestations of heart failure at rest despite aggressive treatment)	As for stages A, B, and C as appropriate Hospice care Hemodynamic monitoring Continual infusion of positive inotropic agents Valve replacement, cardiac transplant as indicated Permanent mechanical support; experimental surgery or drug therapy

Source: Adapted from "ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure)" by S. A. Hunt, W. T. Abraham, M. H. Chin, A. M. Feldman, G. S. Francis, T. G. Ganiats, M. Jessup, M. A. Konstam, D. M. Mancini, K. Michl, M. A. Silver, L. W. Stevenson, and C. W. Yancy, 2005, American College of Cardiology Web Site. Available at http://www.acc.org/clinical/guidlines/failure/index/pdf

goals of invasive hemodynamic monitoring are to evaluate cardiac and circulatory function and the response to interventions.

Hemodynamic parameters include heart rate, arterial blood pressure, central venous or right atrial pressure, pulmonary pressures, and cardiac output. *Direct* hemodynamic parameters are obtained straight from the monitoring device (e.g., heart rate, arterial and venous pressures). *Indirect* or *derived* measurements are calculated using the direct data (e.g., the cardiac index, mean arterial blood pressure, and stroke volume). Invasive hemodynamic monitoring is routinely used in critical care units.

Hemodynamic monitoring systems measure the pressure within a vessel and convert this signal into an electrical waveform that is amplified and displayed. The electrical signal may be graphically recorded on graph paper and displayed digitally on the monitor. System components include an invasive catheter threaded into an artery or vein connected to a transducer by stiff, high-pressure tubing. The pressure transducer translates pressures into an electrical signal that is relayed to the monitor. Additional components of the system include stopcocks and a continuous flush system with normal saline or heparinized saline and an infusion pressure bag to prevent clots from forming in the catheter. Figure 32–3 ■ illustrates a pressure transducer and typical hemodynamic monitoring system.

Hemodynamic pressure monitoring may be used to measure peripheral arterial pressures, or central pressures, such as central venous pressure or right atrial pressure and pulmonary artery pressure. Although the information obtained from invasive monitoring is valuable, the procedure is not without risk. Nursing care of the client undergoing hemodynamic monitoring is outlined on page 1031. Box 32–2 lists potential complications of central pressure monitoring.

INTRA-ARTERIAL PRESSURE MONITORING Intra-arterial pressure monitoring is commonly used in intensive and coronary care units. An indwelling arterial line, commonly called an *art line* or an *A line,* allows direct and continuous monitoring of systolic,

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BOX 32–2 Potential Complications of Central Catheters

- Bleeding
- Hematoma
- Pneumothorax
- Hemothorax
- Arterial puncture
- Dysrhythmias
- Venospasm
- Infection
- Air embolism
- Thromboembolism
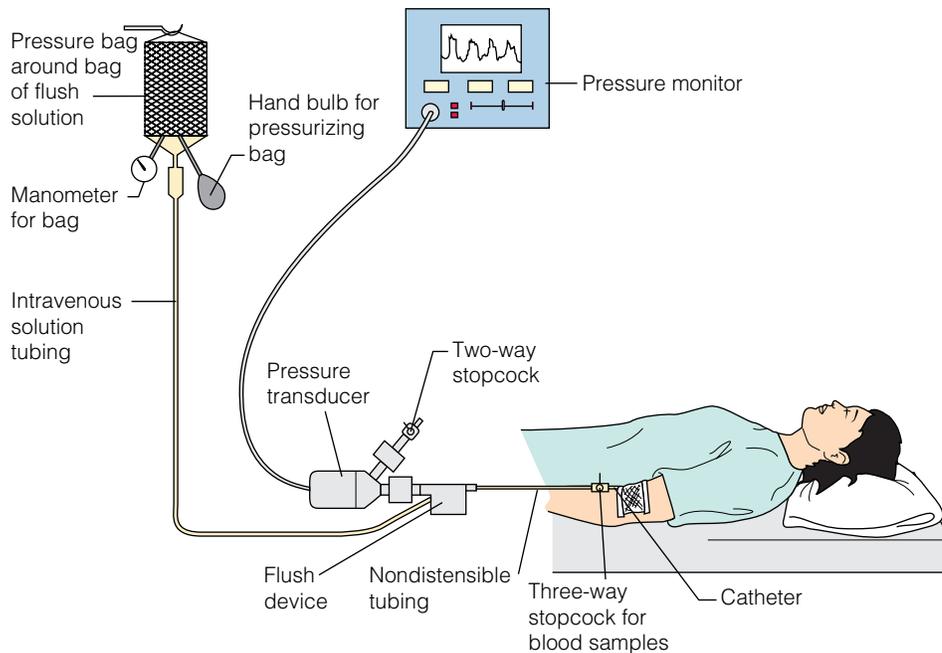- Brachial nerve injury
- Thoracic duct injury

Figure 32–3 ■ A hemodynamic monitoring setup.

diastolic, and mean arterial blood pressure and provides easy access for arterial blood sampling. Arterial lines are used to assess blood volume, to monitor the effects of vasoactive drugs, and to obtain frequent ABG determinations. Because the invasive catheter is inserted directly into the artery, it offers immediate access for blood gas measurements and blood testing.

The arterial blood pressure reflects the cardiac output and the resistance to blood flow created by the elastic arterial walls (*systemic vascular resistance, SVR*). Cardiac output is determined by the blood volume and the ability of the ventricles to fill and effectively pump that blood. Systemic vascular resistance is primarily determined by vessel diameter and distensibility (compliance). Factors such as SNS input, circulating hormones (e.g., epinephrine, norepinephrine, ANP, and vasopressin), and the renin–angiotensin system affect SVR.

The systolic blood pressure, normally about 120 mmHg in healthy adults, reflects the pressure generated during ventricular systole. During diastole, elastic arterial walls keep a minimum pressure within the vessel (diastolic blood pressure) to maintain blood flow through the capillary beds. The average diastolic pressure in a healthy adult is 80 mmHg. The **mean arterial pressure (MAP)** is the average pressure in the arterial circulation throughout the cardiac cycle. It reflects the driving pressure, or perfusion pressure, an indicator of tissue perfusion. The formula MAP = CO × SVR often is used to show the relationships between factors determining the blood pressure. Mean arterial pressure can be calculated by adding one-third of the pulse pressure (PP) to the diastolic pressure (MAP = DBP + PP/3). For example, a blood pressure of 120/80 results in a MAP of 93. Mean arterial pressures of 70 to 90 mmHg are desirable. Perfusion to vital organs is severely jeopardized at MAPs of 50 or less; MAPs greater than 105 mmHg may indicate hypertension or vasoconstriction.

VENOUS PRESSURE MONITORING *Central venous pressure (CVP)* and *right atrial pressure (RAP)* are measures of blood volume and venous return. They also reflect right heart filling pressures. Pressures are elevated in right-sided heart failure. CVP and RAP are primarily used to monitor fluid volume status. To measure venous and atrial pressures, a catheter is inserted in the internal jugular or subclavian vein. The distal tip of the catheter is positioned in the superior vena cava just above or just inside the right atrium. CVP may be measured in either centimeters of water (cm H_2O) or in millimeters of mercury (mmHg). A water manometer is a clear tube with calibrated markings that is attached between a central catheter and the intravenous fluid bag. Pressure in the venous system causes fluid in the manometer to rise or fall. The CVP is recorded by noting the fluid level in the manometer. If the central line is connected to a pressure transducer, venous pressure is displayed digitally in millimeters of mercury.

The normal range for CVP is 2 to 8 cm H_2O or 2 to 6 mmHg, but CVP varies in individual clients. Hypovolemia and shock decrease the CVP; fluid overload, vasoconstriction, and cardiac tamponade increase CVP.

PULMONARY ARTERY PRESSURE MONITORING The pulmonary artery (PA) catheter is a flow-directed, balloon-tipped catheter first used in the early 1970s. The PA catheter is often called a *Swan-Ganz catheter,* after the physicians who developed it. The PA catheter is used to evaluate left ventricular and overall cardiac function. The PA catheter is inserted into a central vein, usually the internal jugular or subclavian vein, and threaded into the right atrium. A small balloon at the tip of the catheter allows the catheter to be drawn into the right ventricle and from there into the pulmonary artery (Figure 32–4 ■). The inflated balloon carries the catheter forward until the balloon wedges in a small branch of pulmonary vasculature. Once in
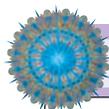
NURSING CARE OF THE CLIENT UNDERGOING   Hemodynamic Monitoring

- Calibrate and level the system at least once a shift using the right atrium as a constant reference level. Relevel the transducer after a change in position. Mark the right atrial position (at the fourth intercostal space, midaxillary line) on the chest wall, and use this as a reference point for all readings. *Calibration and leveling ensure that accurate pressures are recorded. Marking the right atrial level provides a consistent reference point for all caregivers.*
- Measure all pressures between breaths. *This ensures that intrathoracic pressure does not influence pressure readings.*
- Maintain 300 mmHg of pressure on the flush solution at all times. *This ensures a continuous flow of flush solution through the pressure tubing and catheter to prevent clot formation and catheter occlusion.*
- Monitor pressure trends rather than individual readings. *Individual readings may not reflect the client's true status. Trends in pressure readings along with clinical observations provide a better overall picture of the client's status.*
- Obtain a chest x-ray before infusing intravenous fluid into any newly placed central line. *Chest x-ray verifies the location of the catheter and helps prevent pulmonary complications of incorrect catheter placement such as pneumothorax.*
- Set alarm limits for monitored hemodynamic variables. Turn alarms on. *Alarms warn of hemodynamic instability. Always investigate alarms. They may be temporarily silenced to change tubing or draw blood but should never be turned off.*
- Use aseptic technique during catheter insertion and site care. *Aseptic technique is important to prevent infection.*
- Assess and document appearance of the insertion site at least every shift; observe for signs of infiltration, infection, or phlebitis. *Frequent assessment allows early detection and prompt treatment of complications.*
- Change intravenous solutions every 24 hours, site dressing every 48 hours, and tubing to the insertion site every 72 hours. Label solution, tubing, and dressing with date and time of change. *These measures help prevent infection.*
- Thoroughly flush stopcock ports after drawing blood samples from the pressure line. *Flushing prevents colonization of bacteria and occlusion of the catheter.*
- Assess pulse and perfusion distal to the monitoring site. *Frequent assessment is vital to ensure perfusion of the distal extremity.*
- When discontinuing the pressure line, apply manual pressure to the insertion site as soon as the catheter tip is out. Hold pressure for 5 to 15 minutes or until the bleeding stops. *This is particularly important for arterial lines to prevent bleeding and hematoma formation.*
- Secure all connections and stopcocks. *This is done to prevent disconnection of the invasive line and potential hemorrhage.*
- Ensure that electrical equipment is grounded, intact, and operating as expected. *This helps prevent electrical injury.*
- Loosely restrain the affected extremity if the client pulls on the catheter or connections. *Restraints may be necessary to prevent injury from accidental or intentional disconnection or discontinuation of invasive lines (i.e., if the client has dementia or is agitated).*
- Keep tubing free of kinks and tension. *This prevents the catheter from becoming clotted or inadvertently dislodged.*

place, the balloon is deflated, and multiple lumens of the catheter allow measurement of pressures in the right atrium, pulmonary artery, and left ventricle. The normal PA pressure is around 25/10 mmHg; normal mean pulmonary artery pressure is about 15 mmHg (Figure 32–5A ■). Pulmonary artery pressure is increased in left-sided heart failure.
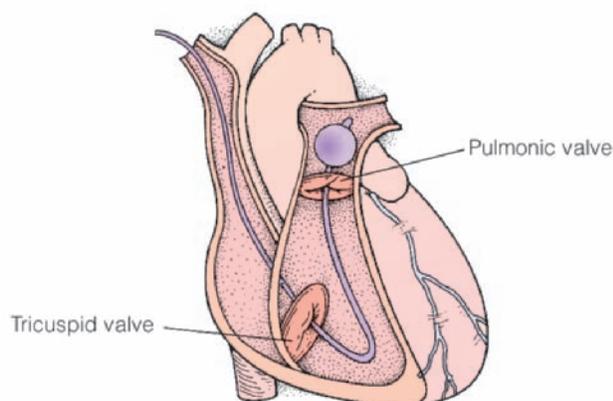

Figure 32–4 ■ Inflation of the balloon on the flow-directed catheter allows it to be carried through the pulmonic valve into the pulmonary artery.

Inflation of the balloon effectively blocks pressure from behind the balloon and allows measurement of pressures generated by the left ventricle. This is known as pulmonary artery wedge pressure (PAWP or PWP) and is used to assess left ventricular function. The normal PAWP is 8 to 12 mmHg (Figure 32–5B). PAWP is increased in left ventricular failure and pericardial tamponade, and decreased in hypovolemia.

Cardiac output also can be measured with the PA catheter using a technique called *thermodilution*. Cardiac output and the cardiac index are used to assess the heart's ability to meet the body's oxygen demands. Because body size affects overall cardiac output, the cardiac index is a more precise measure of heart function. The *cardiac index* is a calculation of cardiac output per square meter of body surface area. The normal cardiac index is 2.8 to 4.2 L/min/m².

Medications

Clients with heart failure often receive multiple medications to reduce cardiac work and improve cardiac function. The main drug classes used to treat heart failure are the angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), beta-blockers, diuretics, inotropic medications (including digitalis, sympathomimetic agents, and phosphodiesterase inhibitors), direct vasodilators, and antidysrhythmic drugs. Nursing implications for ACE inhibitors and ARBs,
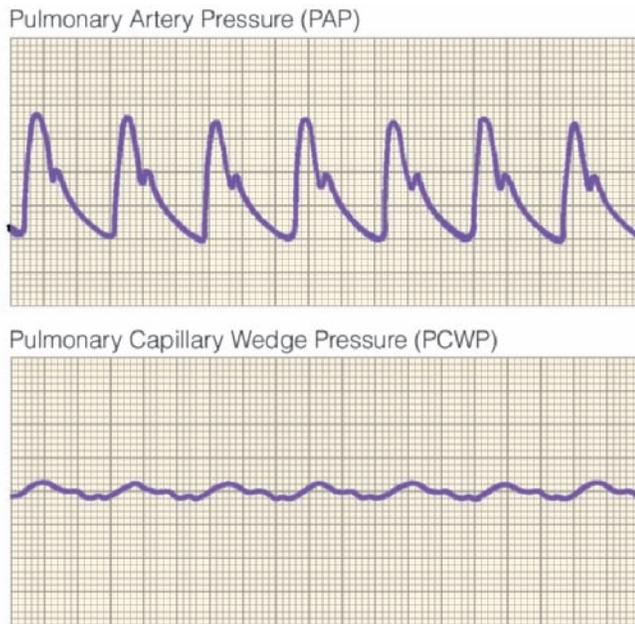
Pulmonary Artery Pressure (PAP)

Pulmonary Capillary Wedge Pressure (PCWP)

Figure 32–5 ■ Typical waveforms seen when measuring (*A*) pulmonary artery pressure and (*B*) pulmonary wedge pressure.

diuretics, and inotropic medications are found in the Medication Administration box on the following page.

ACE inhibitors, ARBs, and beta-blockers interfere with the neurohormonal mechanisms of sympathetic activation and the renin–angiotensin system. ACE inhibitors interrupt the conversion of angiotensin I to angiotensin II by inhibiting the enzyme that mediates the conversion (angiotensin-converting enzyme). Angiotensin II causes intense vasoconstriction, increasing afterload and ventricular wall stress and increasing preload and ventricular dilation. It also stimulates aldosterone and ADH production, causing fluid retention. ACE inhibitors block this renin–angiotensin system activity, decreasing cardiac work and increasing cardiac output. They reduce the progression and manifestations of heart failure, thus reducing the number and frequency of hospital admissions, decreasing mortality rates, and preventing cardiac complications (Kasper et al., 2005).

In contrast to ACE inhibitors, ARBs do not block the production of angiotensin II; instead, they block its action. The pharmacologic effect is similar, and they also are used in heart failure to slow its progression, reduce manifestations, and prevent cardiac complications.

Beta-blockers improve cardiac function in heart failure by inhibiting SNS activity. This prevents the long-term deleterious effects of sympathetic stimulation. Because beta-blockers reduce the force of myocardial contraction and may actually worsen symptoms, they are used in low doses. The combination of ACE inhibitors and beta-blockers improves client outcomes. Beta-blockers are discussed on pages 973–974.

Clients with symptomatic heart failure often are treated with diuretics as well. Diuretics relieve symptoms related to fluid retention. They may, however, cause significant electrolyte imbal-

ances and rapid fluid loss. Clients with severe heart failure are often treated with a loop, or high-ceiling, diuretic such as furosemide (Lasix), bumetanide (Bumex), torsemide (Demadex), or ethacrynic acid (Edecrin). These drugs have a rapid onset of action, inhibiting chloride reabsorption in the ascending loop of Henle, prompting sodium and water excretion. Their major drawback is their efficacy in promoting diuresis; loss of vascular volume can stimulate the SNS. Thiazide diuretics may be used for clients with less severe manifestations of heart failure. These agents promote fluid excretion by blocking sodium reabsorption in the terminal loop of Henle and the distal tubule.

Vasodilators relax smooth muscle in blood vessels, causing dilation. Arterial dilation reduces peripheral vascular resistance and afterload, reducing myocardial work. Venous dilation reduces venous return and preload. Pulmonary vascular relaxation reduces pulmonary capillary pressure, allowing reabsorption of fluid from interstitial tissues and the alveoli. Vasodilators include nitrates, hydralazine, and prazosin, an alpha-adrenergic blocker. See Chapter 35 ∞ for more information about vasodilators.

Nitrates produce both arterial and venous vasodilation. They may be given by nasal spray or the sublingual, oral, or intravenous route. Sodium nitroprusside is a potent vasodilator that may be used to treat acute heart failure. It can cause excessive hypotension, so it is often given along with dopamine or dobutamine to maintain the blood pressure. Isosorbide or nitroglycerin ointment may be used in long-term management of heart failure. See pages 973–974.

In 2005, the U.S. Food and Drug Administration (FDA) approved a new drug for treatment of heart failure in African Americans. This drug, known as BiDil, is a combination of two vasodilators, hydralazine and isosorbide, in fixed doses. In a study of African Americans with severe heart failure, BiDil improved symptoms and significantly reduced the number of hospitalizations and deaths attributed to heart failure (U.S. FDA, 2005). The Cultural Diversity box on page 1035 discusses the nursing implications for BiDil.

Digitalis glycosides are used judiciously in symptomatic heart failure. Digitalis has a *positive inotropic effect* on the heart, increasing the strength of myocardial contraction by increasing the intracellular calcium concentrations. Digitalis also decreases sinoatrial (SA) node automaticity and slows conduction through the atrioventricular (AV) node, increasing ventricular filling time.

Digitalis has a narrow therapeutic index; in other words, therapeutic levels are very close to toxic levels. Early manifestations of digitalis toxicity include anorexia, nausea and vomiting, headache, altered vision, and confusion. A number of cardiac dysrhythmias are also associated with digitalis toxicity, including sinus arrest, supraventricular and ventricular tachycardias, and high levels of AV block. Low serum potassium levels increase the risk of digitalis toxicity, as do low magnesium and high calcium levels. Older adults are at particular risk for digitalis toxicity.

Digitalis levels may be affected by a number of other drugs; check for potential interactions.

MEDICATION ADMINISTRATION **Heart Failure**

ANGIOTENSIN-CONVERTING ENZYME (ACE) INHIBITORS

Enalapril (Vasotec)	**Lisinopril (Prinivil, Zestril)**
Captopril (Capoten)	**Fosinopril (Monopril)**
Moexipril (Univasc)	**Quinapril (Accupril)**
Ramipril (Altace)	**Trandolapril (Mavik)**

ANGIOTENSIN II RECEPTOR BLOCKERS (ARBS)

Candesartan (Atacand)	**Irbesartan (Avapro)**
Losartan (Cozaar)	**Telmisartan (Micardis)**
Valsartan (Diovan)	

ACE inhibitors and ARBs prevent acute coronary events and reduce mortality in heart failure. ACE inhibitors interfere with production of angiotensin II, resulting in vasodilation, reduced blood volume, and prevention of its effects in the heart and blood vessels. In heart failure, ACE inhibitors reduce afterload and improve cardiac output and renal blood flow. They also reduce pulmonary congestion and peripheral edema. ACE inhibitors suppress myocyte growth and reduce ventricular remodeling in heart failure. While the pharmacologic effect of ARBs is similar, they block the action of angiotensin II at the receptor rather than interfering with its production.

Nursing Responsibilities

- Do not give these drugs to women in the second and third trimesters of pregnancy.
- Carefully monitor clients who are volume depleted or who have impaired renal function.
- Use an infusion pump when administering ACE inhibitors intravenously.
- Monitor blood pressure closely for 2 hours following first dose and as indicated thereafter.
- Monitor serum potassium levels; ACE inhibitors can cause hyperkalemia (this is less of a concern with ARBs).
- Monitor white blood cell (WBC) count for potential neutropenia. Report to the physician.

Health Education for the Client and Family

- Take the drug at the same time every day to ensure a stable blood level.
- Monitor your blood pressure and weight weekly. Report significant changes to your doctor.
- Avoid making sudden position changes; for example, rise from bed slowly. Lie down if you become dizzy or light-headed, particularly after the first dose.
- Report any signs of easy bruising and bleeding, sore throat or fever, edema, or skin rash. Immediately report swelling of the face, lips, or eyelids, and itching or breathing problems.
- A persistent, dry cough may develop if you are taking an ACE inhibitor. Contact your doctor if this becomes a problem.
- Take captopril or moexipril 1 hour before meals.

DIURETICS

Chlorothiazide (Diuril)	**Spironolactone (Aldactone)**
Furosemide (Lasix)	**Triamterene (Dyrenium)**
Ethacrynic acid (Edecrin)	**Amiloride (Midamor)**
Bumetanide (Bumex)	**Acetazolamide (Diamox)**
Hydrochlorothiazide (HydroDIURIL)	

Diuretics act on different portions of the kidney tubule to inhibit the reabsorption of sodium and water and promote their excretion. With the exception of the potassium-sparing diuretics—spironolactone, triamterene, and amiloride—diuretics also promote potassium excretion, increasing the risk of hypokalemia. Spironolactone, an al-

dosterone receptor blocker, reduces symptoms and slows progression of heart failure. Aldosterone receptors in the heart and blood vessels promote myocardial remodeling and fibrosis, activate the sympathetic nervous system, and promote vascular fibrosis (which decreases compliance) and baroreceptor dysfunction.

Nursing Responsibilities

- Obtain baseline weight and vital signs.
- Monitor blood pressure, intake and output, weight, skin turgor, and edema as indicators of fluid volume status.
- Assess for volume depletion, particularly with loop diuretics (furosemide, ethacrynic acid, and bumetanide): dizziness, orthostatic hypotension, tachycardia, muscle cramping.
- Report abnormal serum electrolyte levels to the physician. Replace electrolytes as indicated.
- Do not administer potassium replacements to clients receiving a potassium-sparing diuretic.
- Evaluate renal function by assessing urine output, BUN, and serum creatinine.
- Administer intravenous furosemide slowly, no faster than 20 mg/minute. Evaluate for signs of ototoxicity. Do not administer this drug or ethacrynic acid concurrently with aminoglycoside antibiotics (e.g., gentamicin), which are also ototoxic.

Health Education for the Client and Family

- Drink at least 6 to 8 glasses of water per day.
- Take your diuretic at times that will be the least disruptive to your lifestyle, usually in the morning and early afternoon if a second dose is ordered. Take with meals to decrease gastric upset.
- Monitor your blood pressure, pulse, and weight weekly. Report significant weight changes to your doctor.
- Report any of the following to your doctor: severe abdominal pain, jaundice, dark urine, abnormal bleeding or bruising, flu-like symptoms, signs of hypokalemia, hyponatremia, and dehydration (thirst, salt craving, dizziness, weakness, rapid pulse). See Chapter 10 ∞ for manifestations of electrolyte imbalances.
- Avoid sudden position changes. You may experience dizziness, light-headedness, or feelings of faintness.
- Unless you are taking a potassium-sparing diuretic, integrate foods rich in potassium into your diet (see Chapter 10 ∞). Limit sodium use.

POSITIVE INOTROPIC AGENTS

Digitalis Glycosides

Digoxin (Lanoxin)

Digitalis improves myocardial contractility by interfering with ATPase in the myocardial cell membrane and increasing the amount of calcium available for contraction. The increased force of contraction causes the heart to empty more completely, increasing stroke volume and cardiac output. Improved cardiac output improves renal perfusion, decreasing renin secretion. This decreases preload and afterload, reducing cardiac work. Digitalis also has electrophysiologic effects, slowing conduction through the AV node. This decreases the heart rate and reduces oxygen consumption.

Nursing Responsibilities

- Assess apical pulse before administering. Withhold digitalis and notify the physician if heart rate is below 60 bpm and/or
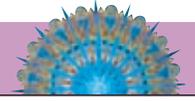
MEDICATION ADMINISTRATION Heart Failure (continued)

manifestations of decreased cardiac output are noted. Record apical rate on medication record.

- Evaluate ECG for scooped (spoon-shaped) ST segment, AV block, bradycardia, and other dysrhythmias (especially PVCs and atrial tachycardias).
- Report manifestations of digitalis toxicity: anorexia, nausea, vomiting, abdominal pain, weakness, vision changes (diplopia, blurred vision, yellow-green or white halos seen around objects), and new-onset dysrhythmias.
- Assess potassium, magnesium, calcium, and serum digoxin levels before giving digitalis. Hypokalemia can precipitate toxicity even when the serum digitalis level is in the "normal" range.
- Monitor clients with renal insufficiency or renal failure and older adults carefully for digitalis toxicity.
- Prepare to administer digoxin immune fab (Digibind) for digoxin toxicity.

Health Education for the Client and Family

- Take your pulse daily before taking your digoxin. Do not take the digoxin if your pulse is below 60 bpm or if you are weak, fatigued, light-headed, dizzy, short of breath, or having chest pain. Notify your physician immediately.
- Contact your doctor if you develop manifestations of digitalis toxicity: palpitations, weakness, loss of appetite, nausea, vomiting, abdominal pain, blurred or colored vision, double vision.
- Avoid using antacids and laxatives; they decrease digoxin absorption.
- Notify your physician immediately if you develop manifestations of potassium deficiency: weakness, lethargy, thirst, depression, muscle cramps, or vomiting.
- Incorporate foods high in potassium into your diet: fresh orange or tomato juice, bananas, raisins, dates, figs, prunes, apricots, spinach, cauliflower, and potatoes.

Sympathomimetic Agents
Dopamine (Inotropin)
Dobutamine (Dobutrex)

Sympathomimetic agents stimulate the heart, improving the force of contraction. Dobutamine is preferred in managing heart failure because it does not increase the heart rate as much as dopamine, and it has a mild vasodilatory effect. These drugs are given by intravenous infusion and may be titrated to obtain their optimal effects.

Phosphodiesterase Inhibitors
Amrinone (Inocor)
Milrinone (Primacor)

Phosphodiesterase inhibitors are used in treating acute heart failure to increase myocardial contractility and cause vasodilation. The net effects are an increase in cardiac output and a decrease in afterload.

Nursing Responsibilities

- Use an infusion pump to administer these agents. Monitor hemodynamic parameters carefully.
- Avoid discontinuing these drugs abruptly.
- Change solutions and tubing every 24 hours.
- Amrinone is given as an intravenous bolus over 2 to 3 minutes, followed by an infusion of 5 to 10 mg/kg/min.
- Amrinone may be infused full strength or diluted in normal saline or half-strength saline. Do not mix this drug with dextrose solutions. After dilution, amrinone can be piggybacked into a line containing a dextrose solution.
- Monitor liver function and platelet counts; amrinone may cause hepatotoxicity and thrombocytopenia.

Health Education for the Client and Family

- Notify the nursing staff if you experience abdominal pain or notice a skin rash or bruising.

Dysrhythmias are common in clients with heart failure. Although premature ventricular contractions (PVCs) may be frequent, they are often not associated with an increased risk of ventricular tachycardia and fibrillation. Because many antidysrhythmic medications depress left ventricular function, PVCs are frequently left untreated in heart failure. Amiodarone is the drug of choice to treat nonsustained ventricular tachycardia, which is associated with a poor prognosis. See page 1006.

Nutrition and Activity

A sodium-restricted diet is recommended to minimize sodium and water retention. Intake is generally limited to 1.5 to 2 g of sodium per day, a moderate restriction. Box 10–4 lists high-sodium foods to avoid; Box 10–5 includes client teaching regarding sodium-restricted diet.

Exercise intolerance, decreased ability to participate in activities using large skeletal muscles due to fatigue or dyspnea, is a common early manifestation of heart failure. Activity may be restricted to bed rest during acute episodes of heart failure to reduce cardiac workload and allow the heart to recompen-sate. Prolonged bed rest and continued activity limitations, however, are not recommended. A moderate, progressive activity program is prescribed to improve myocardial function. Exercise should be performed 3 to 5 days per week, and each session should include a 10- to 15-minute warm-up period, 20 to 30 minutes of exercise at the recommended intensity, and a cool-down period. Walking is encouraged on nontraining days (Piña et al., 2003).

Other Treatments

In end-stage heart failure, devices to provide circulatory assistance or surgery may be required. Surgery may be used to treat the underlying cause of failure (e.g., replacement of diseased valves) or to improve quality of life. Valve replacement is discussed later in this chapter. Heart transplant is currently the only clearly effective surgical treatment for end-stage heart failure; its use is limited by the availability of donor hearts.

CIRCULATORY ASSISTANCE Devices such as the intra-aortic balloon pump or a left-ventricular assist device may be used when the client is expected to recover or as a bridge to trans-

FOCUS ON CULTURAL DIVERSITY   BiDil for Treating Heart Failure in African Americans

BiDil, a fixed-dose combination of two vasodilators (hydralazine and isosorbide), is indicated as an adjunctive treatment in African Americans with heart failure. It has been shown to reduce symptoms, decrease the number of hospitalizations, and prolong life in blacks. The recommended dose is 1 to 2 tablets three times per day, although the dose may be as low as 1/2 tablet three times a day if side effects are intolerable.

Nursing Implications
- Assess vital signs and fluid volume status before administering this drug, because hypotension (orthostatic hypotension in particular) is a common effect.
- Notify the physician if manifestations of systemic lupus erythematosus, glomerulonephritis, or peripheral neuropathy develop.
- Use caution when administering concurrently with monoamine oxidase inhibitors.
- Closely monitor for hypotension when administered concurrently with any potent parenteral antihypertensive agent.

Health Education for the Client and Family
- Take this drug as prescribed.
- Headache is a common adverse effect of this drug, particularly when first starting therapy. Headaches tend to subside with continued treatment.
- Notify your doctor if headaches continue after the first few weeks of therapy, or if you develop chest pain or palpitations while taking this drug.
- This drug can cause a drop in blood pressure, particularly when changing positions from lying to sitting or sitting to standing. Change positions slowly and use caution to prevent falls.
- Do not use drugs such as sildenafil (Viagra, Revatio), vardenafil (Levitra), or tadalafil (Cialis) while taking this medication because the combination may cause an extreme drop in blood pressure leading to fainting, chest pain, or a heart attack.

plant. (See Chapter 31 ∞.) Newer devices that will allow longer term support outside the hospital are in the developmental stages. These devices will serve either as a bridge to transplant or allow the myocardium to heal over an extended period of time.

CARDIAC TRANSPLANTATION   Heart transplant is the treatment of choice for end-stage heart disease. Survival rates are good: 83% at 1 year and 76% at 3 years. More than 90% of clients return to normal, unrestricted functional abilities following transplant (Kasper et al., 2005). The most frequently used transplant procedure leaves posterior walls of the atria, the superior and inferior venae cavae, and pulmonary veins of the recipient intact (Figure 32–6A ■). The atrial walls of the donor heart are then anastomosed to the recipient's atria (Figure 32–6B). The donor pulmonary artery and aorta are anastomosed to the recipient vessels (Figure 32–6C). Care is taken to avoid damaging the sinus node of the donor heart and to ensure integrity of the suture line to prevent postoperative bleeding. Donor organs typically are obtained from young accident victims with no evidence of cardiac trauma.

Nursing care of the heart transplant client is similar to care of any cardiac surgery client (see pages 980–982). Bleeding is a major concern in the early postoperative period. Chest tube drainage is frequently monitored (initially every 15 minutes), as are the cardiac output, pulmonary artery pressures, and CVP. Cardiac tamponade (compression of the heart) can develop, presenting as either a sudden event or a gradual process. Chest tubes are gently milked (not stripped) as needed to maintain patency. Atrial dysrhythmias are relatively common following cardiac transplant. Temporary pacing wires are placed during surgery as the conduction system may be disrupted by surgical manipulation or postoperative swelling. Hypothermia is induced during surgery; postoperatively, the client is gradually rewarmed over a 1- to 2-hour period. Prevention of rapid rewarming and shivering are important to maintain hemodynamic stability and reduce oxygen consumption. Cardiac function is impaired in up to 50% of transplanted hearts during
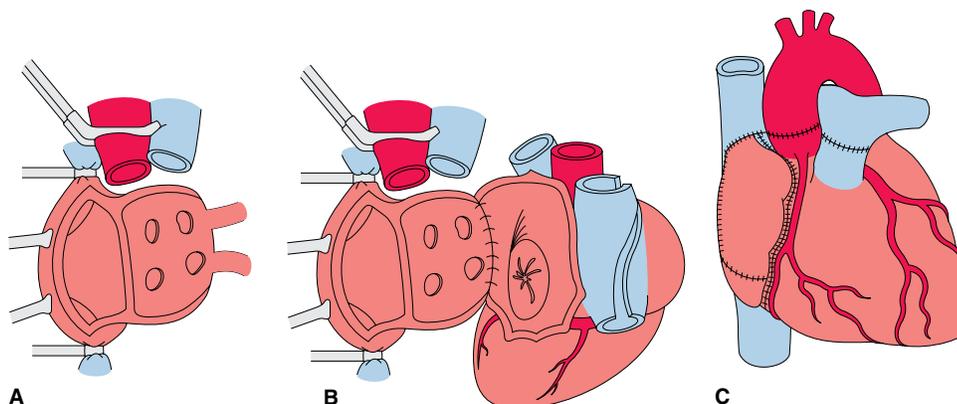

Figure 32–6 ■ Cardiac transplantation. *A,* The heart is removed, leaving the posterior walls of the atria intact. The donor heart is anastomosed to the atria, *B,* and the great vessels, *C.*

the early postoperative period. Inotropic agents such as low-dose dopamine, dobutamine, or milrinone may be required to support cardiac function and circulation (Wade et al., 2004).

Infection and rejection are major postoperative concerns; these are the chief causes of mortality in transplant clients. Rejection may develop immediately after transplant (a rare occurence), within weeks to months, or years after the transplant. Acute rejection usually presents within weeks of the transplant, developing when the transplanted organ is recognized by the immune system as foreign. Lymphocytes infiltrate the organ, and myocardial cell necrosis can be detected on biopsy. Acute rejection often can be treated using immunosuppressive drugs (Wade et al., 2004). These drugs also are given to prevent rejection of the transplanted organ, even when the tissue match is good (see Chapter 13 ∞). Although immunosuppressive medications help prevent organ rejection, they impair the client's defenses against infection. Early postoperative infections commonly are bacterial or fungal (*candida*). Multiple invasive lines, prolonged ventilator support, and immunosuppressive therapy contribute to the transplant recipient's risk for infection. Aggressive nursing care directed at prevention of infection is vital: limiting visitors with communicable diseases, pulmonary hygiene measures, early ambulation, and strict aseptic technique (Wade et al., 2004).

The donor heart is denervated during the transplant procedure. Lack of innervation by the autonomic nervous system affects the heart rate (usually between 90 and 110 bpm in transplanted hearts), its response to position changes, stress, exercise, and certain drugs.

OTHER PROCEDURES Other surgical procedures such as cardiomyoplasty and ventricular reduction surgery do not improve the prognosis or quality of life in clients with end-stage heart failure. *Cardiomyoplasty* involves wrapping the latissimus dorsi muscle around the heart to support the failing myocardium. The muscle is stimulated in synchrony with the heart, providing a more forceful contraction and increasing cardiac output. In ventricular reduction surgery (or *partial ventriculectomy*), a portion of the anterolateral left ventricular wall is resected to improve cardiac function (Tierney et al., 2005).

Complementary Therapies

Strong evidence supports the use of several complementary therapies for heart failure. Hawthorn, a shrubby tree, contains natural cardiotonic ingredients in its blossoms, leaves, and fruit. It increases the force of myocardial contraction, dilates blood vessels, and has a natural ACE inhibitor. Hawthorn should never be used without consulting an experienced herb practitioner and advising the physician (Fontaine, 2005). Nutritional supplements of coenzyme Q10, magnesium, and thiamine may be used in conjunction with other treatments. Coenzyme Q10 improves mitochondria function and energy production.

End-of-Life Care

Unless a cardiac transplant is performed, chronic heart failure is ultimately a terminal disease. The client and family need honest discussions about the anticipated course of the disease and treatment options. It is important to discuss advance directives such as the living will and medical power of attorney, differentiating potential acute events from which recovery would be anticipated (e.g., reversible exacerbation of heart failure, sudden cardiac arrest) from prolonged life support without reasonable expectation of functional recovery. Hospice services are available for clients with heart failure, and should be offered when appropriate. Severe dyspnea is common in the final stages of the disease. It may be managed with narcotic analgesics or with frequent intravenous diuretics and continuous infusion of a positive inotropic agent (Hunt et al., 2005).

NURSING CARE

Health Promotion

Health promotion activities to reduce the risk for and incidence of heart failure are directed at the risk factors. Teach clients about coronary heart disease, the primary underlying cause of heart failure. Discuss CHD risk factors and ways to reduce those risk factors (see Chapter 31 ∞).

Hypertension also is a major cause of heart failure. Routinely screen clients for elevated blood pressure, and refer clients to a primary care provider as indicated. Discuss the importance of effectively managing hypertension to reduce the future risk for heart failure. Likewise, stress the relationship between effective diabetes management and reduced risk of heart failure.

Assessment

Obtain both subjective and objective data when assessing the client with heart failure:

- *Health history:* Complaints of increasing shortness of breath, dyspnea with exertion, decreasing activity tolerance, or paroxysmal nocturnal dyspnea; number of pillows used for sleeping; recent weight gain; presence of a cough; chest or abdominal pain; anorexia or nausea; history of cardiac disease, previous episodes of heart failure; other risk factors such as hypertension or diabetes; current medications; usual diet and activity and recent changes.
- *Physical examination:* General appearance; ease of breathing, conversing, changing positions; apparent anxiety; vital signs including apical pulse; color of skin and mucous membranes; neck vein distention, peripheral pulses, capillary refill, presence and degree of edema; heart and breath sounds; abdominal contour, bowel sounds, tenderness; right upper abdominal tenderness, liver enlargement.
- *Diagnostic tests:* BNP, serum electrolyte, BUN, creatinine, and digitalis levels; ABG results; ECG, echocardiogram, and chest x-ray reports.

Nursing Diagnoses and Interventions

Heart failure impacts quality of life, interfering with such daily activities as self-care and role performance. Reducing the oxygen demand of the heart is a major nursing care goal for the client in acute heart failure. This includes providing rest and carrying out prescribed treatment measures to reduce cardiac work, improve contractility, and manage symptoms. See also the accompanying Nursing Care Plan for additional nursing diagnoses and interventions for the client with heart failure.
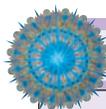
NURSING CARE PLAN A Client with Heart Failure

One year ago, Arthur Jackson, 67 years old, had a large anterior wall MI and underwent subsequent coronary artery bypass surgery. On discharge, he was started on a regimen of enalapril (Vasotec), digoxin, furosemide (Lasix), warfarin (Coumadin), and a potassium chloride supplement. He is now in the cardiac unit complaining of severe shortness of breath, hemoptysis, and poor appetite for 1 week. He is diagnosed with acute heart failure.

ASSESSMENT

Mr. Jackson refuses to settle in bed, preferring to sit in the bed-side recliner in high Fowler's position. He states, "Lately, this is the only way I can breathe." Mr. Jackson states that he has not been able to work in his garden without getting short of breath. He complains of his shoes and belt being too tight.

When Ms. Takashi, RN, Mr. Jackson's nurse, obtains his nursing history, Mr. Jackson insists that he takes his medications regularly. He states that he normally works in his garden for light exercise. In his diet history, Mr. Jackson admits fondness for bacon and Chinese food and sheepishly admits to snacking between meals "even though I need to lose weight."

Mr. Jackson's vital signs are: BP 95/72 mmHg, HR 124 and irregular, R 28 and labored, and T 97.5°F (36.5°C). The cardiac monitor shows atrial fibrillation. An S_3 is noted on auscultation; the cardiac impulse is left of the midclavicular line. He has crackles and diminished breath sounds in the bases of both lungs. Significant jugular venous distention, 3+ pitting edema of feet and ankles, and abdominal distention are noted. Liver size is within normal limits by percussion. Skin cool and diaphoretic. Chest x-ray shows cardiomegaly and pulmonary infiltrates.

DIAGNOSES

- *Excess Fluid Volume* related to impaired cardiac pump and salt and water retention
- *Activity Intolerance* related to impaired cardiac output
- *Impaired Health Maintenance* related to lack of knowledge about diet restrictions

EXPECTED OUTCOMES

- Demonstrate loss of excess fluid by weight loss and decreases in edema, jugular venous distention, and abdominal distention.
- Demonstrate improved activity tolerance.
- Verbalize understanding of diet restrictions.

PLANNING AND IMPLEMENTATION

- Hourly vital signs and hemodynamic pressure measurements.
- Administer and monitor effects of prescribed diuretics and vasodilators.
- Weigh daily; strict intake and output.
- Enforce fluid restriction of 1500 mL/24 hours: 600 mL day shift, 600 mL evening shift, 300 mL at night.

- Auscultate heart and breath sounds every 4 hours and as indicated.
- Administer oxygen per nasal cannula at 2 L/min. Monitor oxygen saturation continuously. Notify physician if less than 94%.
- High Fowler's or position of comfort.
- Notify physician of significant changes in laboratory values.
- Teach about all medications and how to take and record pulse. Provide information about anticoagulant therapy and signs of bleeding.
- Design an activity plan with Mr. Jackson that incorporates preferred activities and scheduled rest periods.
- Instruct about sodium-restricted diet. Allow meal choices within allowed limits.
- Consult dietitian for planning and teaching Mr. and Mrs. Jackson about low-sodium diet.

EVALUATION

Mr. Jackson is discharged after 3 days in the cardiac unit. He has lost 8 pounds during his stay and states it is much easier to breathe and his shoes fit better. He is able to sleep in semi-Fowler's position with only one pillow. His peripheral edema has resolved. Mr. and Mrs. Jackson met with the dietitian, who helped them develop a realistic eating plan to limit sodium, sugar, and fats. The dietitian also provided a list of high-sodium foods to avoid. Mr. Jackson is relieved to know that he can still enjoy Chinese food prepared without monosodium glutamate (MSG) or added salt. Ms. Takashi and the physical therapist designed a progressive activity plan with Mr. Jackson that he will continue at home. He remains in atrial fibrillation, a chronic condition. His knowledge of digoxin and Coumadin has been assessed and reinforced. Ms. Takashi confirms that he is able to accurately check his pulse and can list signs of digoxin toxicity and excessive bleeding.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mr. Jackson's medication regimen remains the same after discharge. What specific teaching does he need related to potential interactions of these drugs?
2. Mr. Jackson tells you, "Talk to my wife about my medications—she's Tarzan and I'm Jane now." How would you respond?
3. Design an exercise plan for Mr. Jackson to prevent deconditioning and conserve energy.
4. Mr. Jackson tells you, "Sometimes I forget whether I have taken my aspirin, so I'll take another just to be sure. After all, they are only baby aspirin. One or two extra a day shouldn't hurt, right?" What is your response?
5. Mr. Jackson is admitted to the neuro unit 6 months later with a cerebral vascular accident (CVA). What is the probable cause of his stroke?

See Evaluating Your Response in Appendix C.

Decreased Cardiac Output

As the heart fails as a pump, stroke volume and tissue perfusion decrease.

- Monitor vital signs and oxygen saturation as indicated. *Decreased cardiac output stimulates the SNS to increase the heart rate in an attempt to restore CO. Tachycardia at rest is common. Diastolic blood pressure may initially be elevated because of vasoconstriction; in late stages, compensatory mechanisms fail, and BP falls. Oxygen saturation levels provide a measure of gas exchange and tissue perfusion.*
- Monitor BNP levels, reporting trends. *BNP levels indicate the severity of heart failure: as the cardiac index decreases*

and left ventricular pressures increase, BNP levels increase. Noting trends provides additional information about cardiac output and effectiveness of the cardiac pump.

- Auscultate heart and breath sounds regularly. *S_1 and S_2 may be diminished if cardiac function is poor. A ventricular gallop (S_3) is an early sign of heart failure; atrial gallop (S_4) may also be present. Crackles are often heard in the lung bases; increasing crackles, dyspnea, and shortness of breath indicate worsening failure.*

PRACTICE ALERT
Report manifestations of decreased cardiac output and tissue perfusion: changes in mentation; decreased urine output; cool, clammy skin; diminished pulses; pallor or cyanosis; dysrhythmias. These are manifestations of decreased tissue perfusion to organ systems.

- Administer supplemental oxygen as needed. *This improves oxygenation of the blood, decreasing the effects of hypoxia and ischemia.*
- Administer prescribed medications as ordered. *Drugs are used to decrease the cardiac workload and increase the effectiveness of contractions.*
- Encourage rest, explaining the rationale. Elevate the head of the bed to reduce the work of breathing. Provide a bedside commode, and assist with activities of daily living (ADLs). Instruct to avoid the Valsalva maneuver. *These measures reduce cardiac workload.*

PRACTICE ALERT
Promote psychologic rest and decrease anxiety. Maintain a quiet environment, and encourage expression of fears and feelings. Explain care measures and their purpose. Psychologic rest decreases oxygen consumption and improves cardiac function.

Excess Fluid Volume
As cardiac output falls, compensatory mechanisms cause salt and water retention, increasing blood volume. This increased fluid volume places additional stress on the already failing ventricles, making them work harder to move the fluid load.

- Assess respiratory status and auscultate lung sounds at least every 4 hours. Notify the physician of significant changes in condition. *Declining respiratory status indicates worsening left heart failure.*

PRACTICE ALERT
Immediately notify the physician if the client develops air hunger, an overwhelming sense of impending doom or panic, tachypnea, severe orthopnea, or a cough productive of large amounts of pink, frothy sputum. Acute pulmonary edema, a medical emergency, can develop rapidly, necessitating immediate intervention to preserve life.

- Monitor intake and output. Notify the physician if urine output is less than 30 mL/h. Weigh daily. *Careful monitoring of fluid volume is important during treatment of heart failure. Diuretics may reduce circulating volume, producing hypo-*

volemia despite persistent peripheral edema. A fall in urine output may indicate significantly reduced cardiac output and renal ischemia. Weight is an objective measure of fluid status: 1 L of fluid is equal to 2.2 lb of weight.

- Record abdominal girth every shift. Note complaints of a loss of appetite, abdominal discomfort, or nausea. *Venous congestion can lead to ascites and may affect gastrointestinal function and nutritional status.*
- Monitor and record hemodynamic measurements. Report significant changes and negative trends. *Hemodynamic measurements provide a means of monitoring condition and response to treatment.*
- Restrict fluids as ordered. Allow choices of fluid type and timing of intake, scheduling most fluid intake during morning and afternoon hours. Offer ice chips and frequent mouth care; provide hard candies if allowed. *Providing choices increases the client's sense of control. Ice chips, hard candies, and mouth care relieve dry mouth and thirst and promote comfort.*

Activity Intolerance
Clients with heart failure have little or no cardiac reserve to meet increased oxygen demands. As the disease progresses and cardiac function is further compromised, activity intolerance increases. The low cardiac output and inability to participate in activities may hinder self-care.

PRACTICE ALERT
Monitor vital signs and cardiac rhythm during and after activities. Tachycardia, dysrhythmias, increasing dyspnea, changes in blood pressure, diaphoresis, pallor, complaints of chest pain, excessive fatigue, or palpitations indicate activity intolerance. Instruct to rest if manifestations are noted. The failing heart is unable to increase cardiac output to meet increased oxygen demands associated with activity. Assessing response to activities helps evaluate cardiac function. Decreasing activity tolerance may signal deterioration of cardiac function, not overexertion.

- Organize nursing care to allow rest periods. *Grouping activities together allows adequate time to "recharge."*
- Assist with ADLs as needed. Encourage independence within prescribed limits. *Assisting with ADLs helps ensure that care needs are met while reducing cardiac workload. Involving the client promotes a sense of control and reduces helplessness.*
- Plan and implement progressive activities. Use passive and active range-of-motion (ROM) exercises as appropriate. Consult with physical therapist on activity plan. *Progressive activity slowly increases exercise capacity by strengthening and improving cardiac function without strain. Activity also helps prevent skeletal muscle atrophy. ROM exercises prevent complications of immobility in severely compromised clients.*
- Provide written and verbal information about activity after discharge. *Written information provides a reference for important information. Verbal information allows clarification and validation of the material.*

Deficient Knowledge: Low-Sodium Diet
Diet is an important part of long-term management of heart failure to manage fluid retention.

- Discuss the rationale for sodium restrictions. *Understanding fosters compliance with the prescribed diet.*
- Consult with dietitian to plan and teach a low-sodium and, if necessary for weight control, low-kilocalorie diet. Provide a list of high-sodium, high-fat, high-cholesterol foods to avoid. Provide American Heart Association materials. *Dietary planning and teaching increase the client's sense of control and participation in disease management. Food lists are useful memory aids.*

> **PRACTICE ALERT**
> Teach how to read food labels for nutritional information. Many processed foods contain "hidden" sodium, which can be identified by careful label reading.

- Assist the client to construct a 2-day meal plan choosing foods low in sodium. *This allows learning assessment, clarification of misunderstandings, and reinforcement of teaching.*
- Encourage small, frequent meals rather than three heavy meals per day. *Small, frequent meals provide continuing energy resources and decrease the work required to digest a large meal.*

NANDA, NIC, and NOC Linkages

Chart 32–1 shows links between NANDA nursing diagnoses, NIC, and NOC for the client with heart failure.

Community-Based Care

Heart failure is a chronic condition requiring active participation by the client and family for effective management. In teaching for home care, include the following topics:
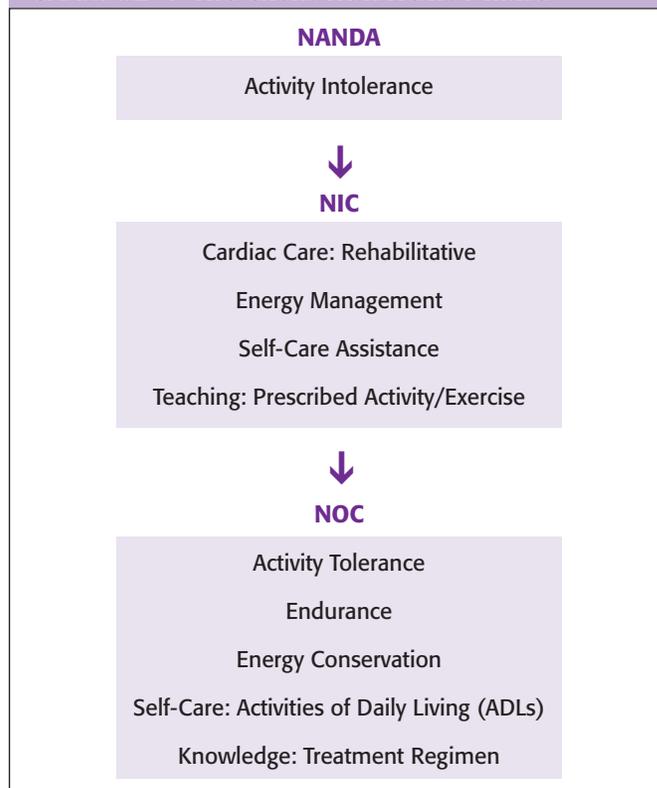
- The disease process and its effects on the client's life
- Warning signals of cardiac decompensation that require treatment
- Desired and adverse effects of prescribed drugs; monitoring for effects; importance of compliance with drug regimen to prevent acute and long-term complications of heart failure
- Prescribed diet and sodium restriction; practical suggestions for reducing salt intake; recommend American Heart Association materials and recipes
- Exercise recommendations to strengthen the heart muscle and improve aerobic capacity (Box 32–3)
- The importance of keeping scheduled follow-up appointments to monitor disease progression and effects of therapy.
  Provide referrals for home health care and household assistance (shopping, transportation, personal needs, and housekeeping) as indicated. Referrals to community agencies, such as local cardiac rehabilitation programs, heart support groups, or the AHA, can provide the client and family with additional materials and psychosocial support.

THE CLIENT WITH PULMONARY EDEMA

Pulmonary edema is an abnormal accumulation of fluid in the interstitial tissue and alveoli of the lung. Both cardiac and noncardiac disorders can cause pulmonary edema. Cardiac causes

> **NANDA, NIC, AND NOC LINKAGES**
> **CHART 32–1 The Client with Heart Failure**
>
> **NANDA**
>
> Activity Intolerance
>
> ↓
>
> **NIC**
>
> Cardiac Care: Rehabilitative
>
> Energy Management
>
> Self-Care Assistance
>
> Teaching: Prescribed Activity/Exercise
>
> ↓
>
> **NOC**
>
> Activity Tolerance
>
> Endurance
>
> Energy Conservation
>
> Self-Care: Activities of Daily Living (ADLs)
>
> Knowledge: Treatment Regimen

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

include acute myocardial infarction, acute heart failure, and valvular disease. *Cardiogenic pulmonary edema,* the focus of this section, is a sign of severe cardiac decompensation. Noncardiac causes of pulmonary edema include primary pulmonary disorders, such as acute respiratory distress syndrome (ARDS), trauma, sepsis, drug overdose, or neurologic sequelae. Pulmonary edema due to ARDS is discussed in Chapter 39 ∞.

> **FAST FACTS**
> - Cardiogenic pulmonary edema is a severe form of heart failure. Risk factors are those associated with heart failure, and treatment focuses on maintaining oxygenation and improving cardiac function.
> - Noncardiogenic pulmonary edema is a primary or secondary lung disorder. It usually occurs secondarily to a critical event such as major trauma, shock, or disseminated intravascular coagulation (DIC). Treatment focuses on maintaining oxygenation and the primary, underlying disorder.

Pulmonary edema is a medical emergency: The client is literally drowning in the fluid in the alveolar and interstitial pulmonary spaces. Its onset may be acute or gradual, progressing to severe respiratory distress. Immediate treatment is necessary.

BOX 32–3 Home Activity Guidelines for the Client with Heart Failure

- Perform as many activities as independently as you can.
- Space your meals and activities.
  - a. Eat six small meals a day.
  - b. Allow time during the day for periods of rest and relaxation.
- Perform all activities at a comfortable pace.
  - a. If you get tired during any activity, stop what you are doing and rest for 15 minutes.
  - b. Resume activity only if you feel up to it.
- Stop any activity that causes chest pain, shortness of breath, dizziness, faintness, excessive weakness, or sweating. Rest. Notify your physician if your activity tolerance changes and if symptoms continue after rest.
- Avoid straining. Do not lift heavy objects. Eat a high-fiber diet and drink plenty of water to prevent constipation. Use laxatives or stool softeners, as approved by your physician, to avoid constipation and straining during bowel movements.
- Begin a graded exercise program. Walking is good exercise that does not require any special equipment (except a good pair of

walking shoes). Plan to walk twice a day at a comfortable, slow pace for the first couple of weeks at home, and then gradually increase the distance and pace. Below is a suggested schedule— but progress at your own speed. Take your time. Aim for walking at least 3 times per week (every other day).

Week 1	200 to 400 ft	Twice a day, slow leisurely pace
Week 2	1/4 mile	15 min, minimum of 3 times per week
Weeks 2 to 3	1/2 mile	30 min, minimum of 3 times per week
Weeks 3 to 4	1 mile	30 min, minimum of 3 times per week
Weeks 4 to 5	1 1/2 mile	30 min, minimum of 3 times per week
Weeks 5 to 6	2 miles	40 min, minimum of 3 times per week

Pathophysiology

In cardiogenic pulmonary edema, the contractility of the left ventricle is severely impaired. The ejection fraction falls because the ventricle is unable to eject the blood that enters it, causing a sharp rise in end-diastolic volume and pressure. Pulmonary hydrostatic pressures rise, ultimately exceeding the osmotic pressure of the blood. As a result, fluid leaking from the pulmonary capillaries congests interstitial tissues, decreasing lung compliance and interfering with gas exchange. As capillary and interstitial pressures increase further, the tight junctions of the alveolar walls are disrupted, and the fluid enters the alveoli, along with large red blood cells (RBCs) and protein molecules. Ventilation and gas exchange are severely disrupted, and hypoxia worsens.

Manifestations

The client with acute pulmonary edema presents with classic manifestations (see box below). Dyspnea, shortness of breath, and labored respirations are acute and severe, accompanied by orthopnea (inability to breathe when lying down). Cyanosis is present, and the skin is cool, clammy, and diaphoretic. A productive cough with pink, frothy sputum develops due to fluid, RBCs, and plasma proteins in the alveoli and airways. Crackles are heard throughout the lung fields on auscultation. As the condition worsens, lung sounds become harsher. The client often is restless and highly anxious, although severe hypoxia may cause confusion or lethargy.

As noted earlier, pulmonary edema is a medical emergency. Without rapid and effective intervention, severe tissue hypoxia and acidosis will lead to organ system failure and death.

INTERDISCIPLINARY CARE

Immediate treatment for acute pulmonary edema focuses on restoring effective gas exchange and reducing fluid and pressure in the pulmonary vascular system. The client is placed in an upright sitting position with the legs dangling to reduce venous return by trapping some excess fluid in the lower extremities. This position also facilitates breathing.

Diagnostic testing is limited to assessment of the acute situation. *ABGs* are drawn to assess gas exchange and acid–base balance. Oxygen tension (PaO_2) is usually low. Initially, carbon dioxide levels ($PaCO_2$) may also be reduced because of rapid respirations. As the condition progresses, the $PaCO_2$ rises and respiratory acidosis develops (see Chapter 10 ∞). *Oxygen saturation* levels also are continuously monitored. The *chest x-ray* shows pulmonary vascular congestion and alveolar edema. Provided the client's condition allows, *hemodynamic monitoring* is instituted. In cardiogenic pulmonary edema, the PAWP is elevated, usually over 25 mmHg. Cardiac output may be decreased.

Morphine is administered intravenously to relieve anxiety and improve the efficacy of breathing. It also is a vasodilator that reduces venous return and lowers left atrial pressure. Although morphine is very effective for clients with cardiogenic

MANIFESTATIONS of Pulmonary Edema

RESPIRATORY
- Tachypnea
- Labored respirations
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Cough productive of frothy, pink sputum
- Crackles, wheezes

CARDIOVASCULAR
- Tachycardia
- Hypotension
- Cyanosis
- Cool, clammy skin
- Hypoxemia
- Ventricular gallop

NEUROLOGIC
- Restlessness
- Anxiety
- Feeling of impending doom

pulmonary edema, naloxone, its antidote, is kept readily available in case respiratory depression occurs.

Oxygen is administered using a positive pressure system that can achieve a 100% oxygen concentration. A continuous positive airway pressure (CPAP) mask system may be used, or the client may be intubated and mechanical ventilation employed (see Chapter 39 ∞). Positive pressure increases alveolar pressures and gas exchange while decreasing fluid diffusion into the alveoli.

Potent loop diuretics such as furosemide, ethacrynic acid, or bumetanide are administered intravenously to promote rapid diuresis. Furosemide is also a venous dilator, reducing venous return to the heart. Vasodilators such as intravenous nitroprusside are given to improve cardiac output by reducing afterload. Dopamine or dobutamine and possibly digoxin are administered to improve the myocardial contractility and cardiac output. Intravenous aminophylline may be used cautiously to reduce bronchospasm and decrease wheezing.

When the client's condition has stabilized, further diagnostic tests may be done to determine the underlying cause of pulmonary edema, and specific treatment measures directed at the cause instituted.

NURSING CARE

Nursing care of the client with acute pulmonary edema focuses on relieving the pulmonary effects of the disorder. Interventions are directed toward improving oxygenation, reducing fluid volume, and providing emotional support.

The nurse often is instrumental in recognizing early manifestations of pulmonary edema and initiating treatment. As with many critical conditions, emergent care is directed toward the ABCs: airway, breathing, and circulation.

Nursing Diagnoses and Interventions

Promoting effective gas exchange and restoring an effective cardiac output are the priorities for nursing and interdisciplinary care of the client with cardiogenic pulmonary edema. The experience of acute dyspnea and shortness of breath is terrifying for the client; the nurse is instrumental in providing emotional support and reassurance.

Impaired Gas Exchange

Accumulated fluid in the alveoli and airways interferes with ventilation of the lungs. As a result, alveolar oxygen levels fall and carbon dioxide levels may rise. Reduced alveolar oxygen decreases diffusion of the gas into pulmonary capillaries. In addition, pulmonary edema increases the distance over which gases must diffuse to cross the alveolar–capillary membrane, further reducing oxygen levels in the blood and oxygen delivery to the tissues.

- Ensure airway patency. *A patent airway is absolutely vital for pulmonary function, including ventilation and gas exchange.*

PRACTICE ALERT
Assess the effectiveness of respiratory efforts and airway clearance. Pulmonary edema increases the work of breathing. This increased effort can lead to fatigue and decreased respiratory effort.

- Assess respiratory status frequently, including rate, effort, use of accessory muscles, sputum characteristics, lung sounds, and skin color. *The status of a client in acute pulmonary edema can change rapidly for the better or worse.*
- Place in high-Fowler's position with the legs dangling. *The upright position facilitates breathing and decreases venous return.*
- Administer oxygen as ordered by mask, CPAP mask, or ventilator. *Supplemental oxygen promotes gas exchange; positive pressure increases the pressure within the alveoli, airways, and thoracic cavity, decreasing venous return, pulmonary capillary pressure, and fluid leak into the alveoli.*
- Encourage to cough up secretions; provide nasotracheal suctioning if necessary. *Coughing moves secretions from smaller airways into larger airways where they can be suctioned out if necessary.*

PRACTICE ALERT
Have emergency equipment readily available in case of respiratory arrest. Be prepared to assist with intubation and initiation of mechanical ventilation. Fatigue, impaired gas exchange, and respiratory acidosis can lead to respiratory and cardiac arrest.

Decreased Cardiac Output

Cardiogenic pulmonary edema usually is caused by either an acute decrease in myocardial contractility or increased workload that exceeds the ability of the left ventricle. The significant decrease in cardiac output increases pressure within the pulmonary vascular system and triggers compensatory mechanisms that increase the heart rate and blood volume. These compensatory mechanisms further increase the workload of the failing heart.

- Monitor vital signs, hemodynamic status, and rhythm continuously. *Acute pulmonary edema is a critical condition, and cardiovascular status can change rapidly.*
- Assess heart sounds for possible S_3, S_4, or murmurs. *These abnormal heart sounds may be due to excess work or may indicate the cause of the acute pulmonary edema.*
- Initiate an intravenous line for medication administration. Administer morphine, diuretics, vasodilators, bronchodilators, and positive inotropic medications (e.g., digoxin) as ordered. *These drugs reduce cardiac work and improve contractility.*

PRACTICE ALERT
Insert an indwelling catheter; record output hourly. Urine output of less than 30 mL/h indicates impaired renal perfusion due to severely impaired cardiac output and a risk for renal failure or other complications.

- Keep accurate intake and output records. Restrict fluids as ordered. *Fluids may be restricted to reduce vascular volume and cardiac work.*

Fear

Acute pulmonary edema is a very frightening experience for everyone (including the nurse).

- Provide emotional support for the client and family members. *Fear and anxiety stimulate the sympathetic nervous system, which can lead to ineffective respiratory patterns and interfere with cooperation with care measures.*
- Explain all procedures and the reasons they are performed to the client and family members. Keep information brief and to the point. Use short sentences and a reassuring tone. *Anxiety and fear interfere with the ability to assimilate information; brief, factual information and reassurance reduce anxiety and fear.*
- Maintain close contact with the client and family, providing reassurance that recovery from acute pulmonary edema is often as dramatic as its onset.

- Answer questions, and provide accurate information in a caring manner. *Knowledge reduces anxiety and psychologic stress associated with this critical condition.*

Community-Based Care

During the acute period, teaching is limited to immediate care measures. Once the acute episode of pulmonary edema has resolved, teach the client and family about its underlying cause and prevention of future episodes. If pulmonary edema follows an acute MI, include information related to CHD and the acute AMI, as well as information related to heart failure. Review the teaching and home care for clients with these disorders for further information.

INFLAMMATORY HEART DISORDERS

Any layer of cardiac tissue—the endocardium, myocardium, or pericardium—can become inflamed, thus damaging the heart valves, heart muscle, or pericardial lining. Manifestations of inflammatory heart disorders range from very mild to life threatening. This section discusses the causes and management of rheumatic heart disease, endocarditis, myocarditis, and pericarditis.

THE CLIENT WITH RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Rheumatic fever is a systemic inflammatory disease caused by an abnormal immune response to pharyngeal infection by group A beta-hemolytic streptococci. Rheumatic fever usually is a self-limiting disorder, although it may become recurrent or chronic. Although the heart commonly is involved in the acute inflammatory process, only about 10% of people with rheumatic fever develop rheumatic heart disease (Porth, 2005). Rheumatic heart disease frequently damages the heart valves and is a major cause of mitral and aortic valve disorders discussed in the next section of this chapter.

Incidence, Prevalence, and Risk Factors

In the United States and other industrialized nations, rheumatic fever and its sequelae are rare. The peak incidence of rheumatic fever is between ages 5 and 15; although it is rare after age 40, it may affect people of any age. About 3% of people with untreated group A streptococcal pharyngitis develop rheumatic fever (Kasper et al., 2005). Rheumatic fever and rheumatic heart disease remain significant public health problems in many developing countries. Highly virulent strains of group A streptococci have caused scattered outbreaks in the United States in recent years (McCance & Huether, 2006).

Risk factors for streptococcal infections of the pharynx include environmental and economic factors such as crowded living conditions, malnutrition, immunodeficiency, and poor access to health care. Evidence also suggests an unknown genetic factor in susceptibility to rheumatic fever.

FAST FACTS
- The peak incidence of rheumatic fever is in children ages 5 to 15.
- Young adults (in late adolescence and the early 20s) are the primary adult population affected by rheumatic fever.
- People past age 40 rarely develop the disease, unless it is a case of recurrent rheumatic fever.
- Although crowded living conditions and lower socioeconomic status are risk factors, a relatively recent outbreak in the United States occurred in people with ready access to health care. Mucoid strains of *streptococcus pyogenes* have been associated with this outbreak (Veasy et al, 2004).

Pathophysiology

The pathophysiology of rheumatic fever is not yet totally understood. It is thought to result from an abnormal immune response to M proteins on group A beta-hemolytic streptococcal bacteria. These antigens can bind to cells in the heart, muscles, and brain. They also bind with receptors in synovial joints, provoking an autoimmune response (McCance & Huether, 2006). The resulting immune response to the bacteria also leads to inflammation in tissues containing these M proteins. Inflammatory lesions develop in connective tissues on the heart, joints, and skin. The antibodies may remain in the serum for up to 6 months following the initiating event. See Chapters 12 ∞ and 13 ∞ for more information about the immune system and inflammatory response.

Carditis, inflammation of the heart, develops in about 50% of people with rheumatic fever. The inflammatory process usually involves all three layers of the heart—the pericardium, myocardium, and endocardium. *Aschoff bodies,* localized areas of tissue necrosis surrounded by immune cells, develop in cardiac tissues. Pericardial and myocardial inflammation tends to be mild and self-limiting. Endocardial inflammation, however, causes swelling and erythema of valve structures and small vegetative lesions on valve leaflets. As the inflammatory process resolves, fibrous scarring occurs, causing deformity.

Rheumatic heart disease (RHD) is a slowly progressive valvular deformity that may follow acute or repeated attacks of

rheumatic fever. Valve leaflets become rigid and deformed; commissures (openings) fuse, and the chordae tendineae fibrose and shorten. This results in stenosis or regurgitation of the valve. In **stenosis**, a narrowed fused valve obstructs forward blood flow. **Regurgitation** occurs when the valve fails to close properly (an *incompetent* valve), allowing blood to flow back through it. Valves on the left side of the heart are usually affected; the mitral valve is most frequently involved.

Manifestations

Manifestations of rheumatic fever typically follow the initial streptococcal infection by about 2 to 3 weeks. Fever and migratory joint pain are often initial manifestations. The knees, ankles, hips, and elbows are common sites of swelling and inflammation. *Erythema marginatum* is a temporary nonpruritic skin rash characterized by red lesions with clear borders and blanched centers usually found on the trunk and proximal extremities. Neurologic symptoms of rheumatic fever, although rare in adults, may range from irritability and an inability to concentrate to clumsiness and involuntary muscle spasms.

Manifestations of carditis include chest pain, tachycardia, a pericardial friction rub, or evidence of heart failure. On auscultation, an S_3, S_4, or a heart murmur may be heard. Cardiomegaly or pericardial effusion may develop. Other manifestations of rheumatic fever are listed in the box below.

INTERDISCIPLINARY CARE

Management of the client with rheumatic heart disease (RHD) focuses on eradicating the streptococcal infection and managing the manifestations of the disease. Carditis and resulting heart failure are treated with measures to reduce the inflammatory process and manage the heart failure. Activities are limited, but bed rest is not generally ordered.

MANIFESTATIONS of Rheumatic Fever

CARDIAC
- Chest pain
- Friction rub
- Heart murmur

MUSCULOSKELETAL
- *Migratory polyarthritis:* redness, heat, swelling, pain, and tenderness of more than one joint
- Usually affects large joints of extremities

SKIN
- *Erythema marginatum:* transitory pink, nonpruritic, macular lesions on trunk or inner aspect of upper arms or thighs
- *Subcutaneous nodules* over extensors of wrist, elbow, ankle, and knee joints

NEUROLOGIC
- *Sydenham's chorea:* irritability, behavior changes; sudden, jerky, involuntary movements

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Diagnosis

In addition to the history and physical examination, a number of laboratory and diagnostic tests may be ordered for the client with suspected rheumatic fever. Table 32–4 identifies tests and values indicative of carditis associated with rheumatic fever.

- *Complete blood count (CBC)* and *erythrocyte sedimentation rate (ESR)* are indicators of the inflammatory process. The WBC count is elevated, and the number of RBCs may be low due to the inflammatory inhibition of erythropoiesis. The ESR, a general indicator of inflammation, is elevated.
- *C-reactive protein (CRP)* is positive in an active inflammatory process.
- *Antistreptolysin (ASO) titer* is a test for streptococcal antibodies. It rises within 2 months of the onset and is positive in most clients with rheumatic fever.
- Throat culture is positive for group A beta-hemolytic streptococcus in only 25% to 40% of clients with acute rheumatic fever (Kasper et al., 2005).

Medications

As soon as rheumatic fever is diagnosed, antibiotics are started to eliminate the streptococcal infection. Penicillin is the antibiotic of choice to treat group A streptococci. Antibiotics are prescribed for at least 10 days. Erythromycin or clindamycin is used if the client is allergic to penicillin. Prophylactic antibiotic therapy is continued for 5 to 10 years to prevent recurrences. Recurrences after 5 years or age 25 are rare (Tierney et al., 2005). Penicillin G, 1.2 million units injected intramuscularly every 3 to 4 weeks, is the prophylaxis of choice. Oral penicillin, amoxicillin, sulfadiazine, or erythromycin may also be used.

TABLE 32–4 Diagnostic Tests for Rheumatic Heart Disease

TEST	VALUES CHARACTERISTIC OF RHEUMATIC HEART DISEASE
White blood cell (WBC) count	Greater than 10,000/mm^3
Red blood cell (RBC) count	Less than 4 million/mm^3
Erythrocyte sedimentation rate (ESR)	More than 20 mm/h
C-reactive protein	Positive
Antistreptolysin (ASO) titer	Above 250 international unit/mL
Throat culture	Usually positive for group A beta-hemolytic streptococci
Cardiac enzymes	Elevated in severe carditis
ECG changes	Prolonged PR interval
Chest x-ray	May show cardiac enlargement
Echocardiogram	May show valvular damage, enlarged chambers, decreased ventricular function, or pericardial effusion

Joint pain and fever are treated with salicylates (e.g., aspirin), ibuprofen, or another nonsteroidal anti-inflammatory drug (NSAID); corticosteroids may be used for severe pain due to inflammation or carditis. See Chapter 13 ∞ for information about the use of these anti-inflammatory medications.

NURSING CARE

Health Promotion

Rheumatic fever is preventable. Prompt identification and treatment of streptococcal throat infections help decrease spread of the pathogen and the risk for rheumatic fever. Characteristics of streptococcal sore throat; include a red, fiery-looking throat; pain with swallowing; enlarged and tender cervical lymph nodes; fever range of 101° to 104°F (38.3° to 40.0°C); and headache. Emphasize the importance of finishing the complete course of medication to eradicate the pathogen.

Assessment

Assess clients at risk for rheumatic fever (prolonged, untreated, or recurrent pharyngitis) for possible manifestations.

- *Health history:* Complaints of recent sore throat with fever, difficulty swallowing, and general malaise; treatment measures; previous history of strep throat or rheumatic fever; history of heart murmur or other cardiac problems; current medications.
- *Physical examination:* Vital signs including temperature; skin color, presence of rash on trunk or proximal extremities; mental status; evidence of inflamed joints; heart and lung sounds.
- *Diagnostic tests:* WBC and differential, ESR, CRP results; ASO titer and throat culture results; ECG and echocardiogram reports.

Nursing Diagnoses and Interventions

The nursing care focus for the client with RHD is on providing supportive care and preventing complications. Teaching to prevent recurrence of rheumatic fever is extremely important. *Pain* and *Activity Intolerance* are priority nursing diagnoses for the client with rheumatic fever and RHD.

Acute Pain

Joint and chest pain due to acute inflammation is common in rheumatic fever. Pain and inflammation may interfere with rest and healing.

- Administer anti-inflammatory drugs as ordered. Promptly report manifestations of aspirin toxicity, including tinnitus, vomiting, and gastrointestinal bleeding. Give aspirin and other NSAIDs with food, milk, or antacids to minimize gastric irritation. *Joint pain and fever may be treated with anti-inflammatory agents such as aspirin and NSAIDs. When used for its anti-inflammatory effect, aspirin doses may be high, and it is given around the clock (e.g., every 4 hours). Steroids may be prescribed for severe carditis.*

- Provide warm, moist compresses for local pain relief of acutely inflamed joints. *Moist heat helps relieve pain associated with inflamed joints by reducing inflammation.*
- Auscultate heart sounds as indicated (every shift or each home visit). Notify the physician if a pericardial friction rub or a new murmur develops. *A friction rub is produced as inflamed pericardial surfaces rub against each other. This also stimulates pain receptors, and may increase discomfort.*

Activity Intolerance

The client with acute carditis or RHD may develop heart failure if the heart is unable to supply enough oxygen to meet the body's demand. Manifestations of fatigue, weakness, and dyspnea on exertion may result.

- Explain the importance of activity limitations and reinforce teaching as needed. *Activities are limited during the acute phase of carditis to reduce the workload of the heart. Understanding the rationale improves cooperation with the limitations.*
- Encourage social and diversional activities such as visits with friends and family, reading, playing cards or board games, watching television, and listening to music or talking books. *Diversional activities provide a focus for the client whose physical activities must be limited.*
- Encourage gradual increases in activity, monitoring for evidence of intolerance or heart failure. Consult a cardiac rehabilitation specialist to help design an activity progression schedule. *Gradual activity progression is encouraged as the client's condition improves. Activity tolerance is monitored and activities modified as needed.*

Community-Based Care

Most clients with rheumatic fever and carditis do not require hospitalization. Teaching for home care focuses on both acute care and preventing recurrences and further tissue damage. Include the following topics:

- The importance of completing the full course of antibiotic therapy and continuing antibiotic prophylaxis as prescribed. For the client with chronic RHD, include the importance of antibiotic prophylaxis for invasive procedures (e.g., dental care, endoscopy, or surgery) to prevent bacterial endocarditis. Pamphlets on endocarditis prevention are helpful reminders, and are available from the American Heart Association.
- Preventive dental care and good oral hygiene to maintain oral health and prevent gingival infections, which can lead to recurrence of the disease.
- Early recognition of streptococcal sore throat and appropriate treatment for both the client and family members.
- Early manifestations of heart failure to report to the physician.
- Prescribed medications, including their dosage, route, intended and potential adverse effects, and manifestations to report to the physician.
- Dietary sodium restriction if ordered or recommended. A high-carbohydrate, high-protein diet may be recommended to facilitate healing and combat fatigue.

Refer for home health services or household assistance as indicated.

THE CLIENT WITH INFECTIVE ENDOCARDITIS

Endocarditis, inflammation of the endocardium, can involve any portion of the endothelial lining of the heart. The valves usually are affected. Endocarditis is usually infectious in nature, characterized by colonization or invasion of the endocardium and heart valves by a pathogen.

> **FAST FACTS**
> - Subacute bacterial endocarditis develops more slowly and usually occurs in people with previous heart valve damage.
> - Acute bacterial endocarditis has an abrupt onset and typically affects people with no previous history of heart problems.

Incidence and Risk Factors

Endocarditis is relatively uncommon, with an incidence of 1.5 to 6.2 cases per 100,000 people in developed countries (Kasper et al., 2005). The greatest risk factor for endocarditis is previous heart damage. Lesions develop on deformed valves, on valve prostheses, or in areas of tissue damage due to congenital deformities or ischemic disease. The left side of the heart, the mitral valve in particular, is usually affected. Intravenous drug use also is a significant risk factor. The right side of the heart usually is affected in these clients. Other risk factors include invasive catheters (e.g., a central venous catheter, hemodynamic monitoring, or an indwelling urinary catheter), dental procedures or poor dental health, and recent heart surgery.

Prosthetic valve endocarditis (PVE) may occur in clients with a mechanical or tissue valve replacement. This infection may develop in the early postoperative period (within 2 months after surgery) or late. Prosthetic valve endocarditis accounts for 10% to 20% of endocarditis cases. It usually affects males over the age of 60, and is more frequently associated with aortic valve prostheses than with mitral valve replacements. Early PVE is usually due to prosthetic valve contamination during surgery or perioperative bacteremia. Its course often is rapid, and mortality is high. Late-onset PVE more closely resembles subacute endocarditis.

Pathophysiology

Entry of pathogens into the bloodstream is required for infective endocarditis to develop. Bacteria may enter through oral lesions, during dental work or invasive procedures, such as intravenous catheter insertion, surgery, or urinary catheterization; during intravenous drug use; or as a result of infectious processes such as urinary tract or upper respiratory infection.

The initial lesion is a sterile platelet-fibrin vegetation formed on damaged endothelium (Figure 32–7 ■). In acute infective endocarditis, these lesions develop on healthy valve structures, although the mechanism is unknown. In subacute endocarditis, they usually develop on already damaged valves or in endocardial tissue that has been damaged by abnormal pressures or blood flow within the heart.

Organisms that have invaded the blood colonize these vegetations. The vegetation enlarges as more platelets and fibrin are

Figure 32–7 ■ A vegetative lesion of bacterial endocarditis.
Source: M. English/Custom Medical Stock Photo, Inc.

attracted to the site and cover the infecting organism. This covering "protects" the bacteria from quick removal by immune defenses such as phagocytosis by neutrophils, antibodies, and complement. Vegetations may be singular or multiple. They expand while loosely attached to edges of the valve. Friable vegetations can break or shear off, embolizing and traveling through the bloodstream to other organ systems. When they lodge in small vessels, they may cause hemorrhages, infarcts, or abscesses. Ultimately, the vegetations scar and deform the valves and cause turbulence of blood flowing through the heart. Heart valve function is affected, either obstructing forward blood flow, or closing incompletely.

Endocarditis is classified by its acuity and disease course (Table 32–5). *Acute infective endocarditis* has an abrupt onset and is a rapidly progressive, severe disease. Although almost any organism can cause infective endocarditis, virulent organisms such as *Staphylococcus aureus* cause a more abrupt onset and destructive course. *S. aureus* is commonly the infective organism in acute endocarditis. In contrast, *subacute infective endocarditis* has a more gradual onset, with predominant systemic manifestations. It is more likely to occur in clients with preexisting heart disease. *Streptococcus viridans,* enterococci, other gram-negative and gram-positive bacilli, yeasts, and fungi tend to cause the subacute forms of endocarditis (Porth, 2005).

Manifestations

The manifestations of infective endocarditis often are nonspecific (see the box on page 1046). A temperature above 101.5°F (39.4°C) and flulike symptoms develop, accompanied by cough, shortness of breath, and joint pain. The presentation of acute staphylococcal endocarditis is more severe, with a sudden onset, chills, and a high fever. Heart murmurs are heard in 90% of persons with infective endocarditis. An existing murmur may worsen, or a new murmur may develop.

Splenomegaly is common in chronic disease. Peripheral manifestations of infective endocarditis result from microemboli or circulating immune complexes. These manifestations include:

- *Petechiae:* small, purplish-red hemorrhagic spots on the trunk, conjunctiva, and mucous membranes

TABLE 32–5 Classifications of Infective Endocarditis

	ACUTE INFECTIVE ENDOCARDITIS	SUBACUTE INFECTIVE ENDOCARDITIS
Onset	Sudden	Gradual
Usual organism	*Staphylococcus aureus*	*Streptococcus viridans*, enterococci, gram-negative and gram-positive bacilli, fungi, yeasts
Risk factors	Usually occurs in previously normal heart; intravenous drug use, infected intravenous sites	Usually occurs in damaged or deformed hearts; dental work, invasive procedures, and infections
Pathologic process	Rapid valve destruction	Valve destruction leading to regurgitation; embolization of friable vegetations
Presentation	Abrupt onset with spiking fever and chills; manifestations of heart failure	Gradual onset of febrile illness with cough, dyspnea, arthralgias, abdominal pain

MANIFESTATIONS of Infective Endocarditis

- Chills and fever
- General malaise, fatigue
- Arthralgias
- Cough, dyspnea
- Heart murmur
- Anorexia, abdominal pain
- Petechiae, splinter hemorrhages
- Splenomegaly

- *Splinter hemorrhages:* hemorrhagic streaks under the fingernails or toenails
- *Osler's nodes:* small, reddened, painful raised growths on finger and toe pads
- *Janeway lesions:* small, nontender, purplish-red macular lesions on the palms of the hands and soles of the feet
- *Roth's spots:* small, whitish spots (cotton-wool spots) seen on the retina.

Complications

Embolization of vegetative fragments may affect any organ system, particularly the lungs, brain, kidneys, and the skin and mucous membranes, with resulting organ infarction. Other common complications of infective endocarditis include heart failure, abscess, and aneurysms due to infiltration of the arterial wall by organisms. Without treatment, endocarditis is almost universally fatal; fortunately, antibiotic therapy is usually effective to treat this disease.

INTERDISCIPLINARY CARE

Eradicating the infecting organism and minimizing valve damage and other adverse consequences of infective endocarditis are the priorities of care.

Diagnosis

There are no definitive tests for infective endocarditis, but diagnostic tests help establish the diagnosis.

- *Blood cultures* usually are positive for bacteria or other pathogens. Blood cultures are considered positive when a typical infecting organism is identified from two or more separate blood cultures (drawn from different sites and/or at different times, e.g., 12-hour intervals).
- *Echocardiography* (either transthoracic or transesophageal) to visualize vegetations can be diagnostic for infective endocarditis when combined with positive blood cultures. See Chapter 30 ∞ for more information about echocardiography.
- *Serologic immune testing* for circulating antigens to typical infective organisms may be done.

Other diagnostic tests may include the CBC, ESR, serum creatinine, chest x-ray, and an electrocardiogram.

Medications

Preventing endocarditis in clients at high risk is important. Antibiotics are commonly prescribed for clients with preexisting valve damage or heart disease prior to high-risk procedures (Table 32–6).

Antibiotic therapy effectively treats infective endocarditis in most cases. The goal of therapy is to eradicate the infecting organism from the blood and vegetative lesions in the heart. The fibrin covering that protects colonies of organisms from immune defenses also protects them from antibiotic therapy. Therefore, an extended course of multiple intravenous antibiotics is required.

Following blood cultures, antibiotic therapy is initiated with drugs known to be effective against the most common infecting organisms: staphylococci, streptococci, and enterococci. The initial regimen may include nafcillin or oxacillin, penicillin or ampicillin, and gentamicin. Once the organism has been identified, therapy is tailored to that organism. Streptococcal and enterococcal infections are treated with a combination of penicillin and gentamicin. If the client is allergic to penicillin, ceftriaxone, cefazolin, or vancomycin may be used. Staphylococcal infections are treated with nafcillin or oxacillin and gentamicin; cefazolin or vancomycin may be used if penicillin allergy is present. Intravenous drug therapy is continued for 2 to 8 weeks, depending on the infecting organism, the drugs used, and the results of repeat blood cultures. See Chapter 12 ∞ for the nursing implications for antibiotic therapy.

The client with prosthetic valve endocarditis requires extended treatment, usually 6 to 8 weeks. Combination therapy

TABLE 32–6 Antibiotic Prophylaxis for Infective Endocarditis

INDICATIONS FOR PROPHYLAXIS	SELECTED PROCEDURES FOR WHICH PROPHYLAXIS IS RECOMMENDED	SUGGESTED ANTIBIOTICS
Prosthetic valves	Dental procedures in which bleeding is likely, including cleaning	Amoxicillin
Previous episode(s) of infective endocarditis	Most surgeries	Erythromycin
Rheumatic heart disease	Bronchoscopy	Ampicillin
Hypertrophic cardiomyopathy	Cystoscopy	Clindamycin
Mitral valve prolapse with regurgitation and murmur	Urinary catheterization when infection is present	Vancomycin
Sclerotic aortic valve	Incision and drainage of infected tissue	(*Note:* Choice of antibiotic depends on procedure.)
Most congenital heart malformations	Vaginal delivery if infection is present	

using vancomycin, rifampin, and gentamicin is used to treat these resistant infections.

Surgery

Some clients with infective endocarditis require surgery to:
- Replace severely damaged valves.
- Remove large vegetations at risk for embolization.
- Remove a valve that is a continuing source of infection that does not respond to antibiotic therapy.

The most common indication for surgery is valvular regurgitation that causes heart failure and does not respond to medical therapy. When the infection has not responded to antibiotic therapy within 7 to 10 days, the infected valve may be replaced to facilitate eradication of the organism. Clients with fungal endocarditis usually require surgical intervention. More information on valve replacement surgery is provided in the section on valve disorders.

NURSING CARE

Health Promotion

Prevention of endocarditis is vital in susceptible people. Education is a key part of prevention. Use every opportunity to educate individuals and the public about the risks of intravenous drug use, including endocarditis. Discuss preventive measures with all clients with specific risk factors, such as a heart murmur or known heart disease.

Assessment

Assessment related to ineffective endocarditis includes identifying risk factors and manifestations of the disease.
- *Health history:* Complaints of persistent flulike symptoms, fatigue, shortness of breath, and activity intolerance; history of recent dental work or other invasive procedures; known heart murmur, valve or other heart disorder; recent intravenous drug use.
- *Physical examination:* Vital signs including temperature; apical pulse and heart sounds; rate and ease of respirations, lung sounds; skin color, temperature, and presence of petechiae or splinter hemorrhages.
- *Diagnostic tests:* WBC and differential, ESR; blood culture and sensitivity results; echocardiogram reports.

Nursing Diagnoses and Interventions

Nursing care focuses on managing the manifestations of endocarditis, administering antibiotics, and teaching the client and family members about the disorder. In addition to the diagnoses identified below, nursing diagnoses and interventions for heart failure also may be appropriate for clients with infective endocarditis.

Risk for Imbalanced Body Temperature

Fever is common in clients with infective endocarditis. It may be acutely elevated and accompanied by chills, particularly with acute infective endocarditis. The inflammatory process initiates a cycle of events that affects the regulation of temperature and causes discomfort.
- Record temperature every 2 to 4 hours. Report temperature above 101.5°F (39.4°C). Assess for complaints of discomfort. *Fever is usually low grade (below 101.5°F [39.4°C]) in infective endocarditis; higher temperatures may cause discomfort. The temperature usually returns to normal within 1 week after initiation of antibiotic therapy. Continued fever may indicate a need to modify the treatment regimen.*
- Obtain blood cultures as ordered, before initial antibiotic dose. *Initial blood cultures are obtained before antibiotic therapy is started to obtain adequate organisms to culture and identify. Follow-up cultures are used to assess the effectiveness of therapy.*
- Provide anti-inflammatory or antipyretic agents as prescribed. *Fever may be treated with anti-inflammatory or antipyretic agents such as aspirin, ibuprofen, or acetaminophen.*
- Administer antibiotics as ordered; obtain peak and trough drug levels as indicated. *Intravenous antibiotics are given to eradicate the pathogen. Peak and trough levels are used to evaluate the dose effectiveness in maintaining a therapeutic blood level.*

Risk for Ineffective Tissue Perfusion

Embolization of vegetative lesions can threaten tissue and organ perfusion. Vegetations from the left heart may lodge in arterioles or capillaries of the brain, kidneys, or peripheral tissues, causing infarction or abscess. A large embolism can cause manifestations of stroke or transient ischemic attack, renal failure, or tissue ischemia. Emboli from the right side of the heart become entrapped in pulmonary vasculature, causing manifestations of pulmonary embolism.

- Assess for, document, and report manifestations of decreased organ system perfusion:
  a. *Neurologic:* changes in level of consciousness, numbness or tingling in extremities, hemiplegia, visual disturbances, or manifestations of stroke
  b. *Renal:* decreased urine output, hematuria, elevated BUN or creatinine
  c. *Pulmonary:* dyspnea, hemoptysis, shortness of breath, diminished breath sounds, restlessness, sudden chest or shoulder pain
  d. *Cardiovascular:* chest pain radiating to jaw or arms, tachycardia, anxiety, tachypnea, hypotension.
  All major organs and tissues, and the microcirculation, may be affected by emboli when vegetations break off due to turbulent blood flow. Emboli may cause manifestations of organ dysfunction. The most devastating effects of emboli are in the brain and the myocardium, with resulting infarctions. Intravenous drug users have a high risk of pulmonary emboli as a result of right-sided endocardial fragments.
- Assess and document skin color and temperature, quality of peripheral pulses, and capillary refill. *Peripheral emboli affect tissue perfusion, with a risk for tissue necrosis and possible extremity loss.*

Ineffective Health Maintenance

The client with endocarditis often is treated in the community. Teaching about disease management and prevention of possible recurrences of endocarditis is vital.

- Demonstrate intravenous catheter site care and intermittent antibiotic administration if the client and family will manage therapy. Have the client and/or significant other redemonstrate appropriate techniques. *Intermittent antibiotic infusions may be managed by the client or family members, or the client may go to an outpatient facility to receive the infusions. Appropriate site care is necessary to reduce the risk of trauma and infection.*
- Explain the actions, doses, administration, and desired and adverse effects of prescribed drugs. Identify manifestations to be reported to the physician. Provide practical information about measures to reduce the risk of superinfection (e.g., consuming 8 oz of yogurt or buttermilk containing live bacterial cultures daily). *Careful compliance with prescribed drug therapy is vital to eradicate the infecting organism. Antibiotic therapy can, however, cause superinfections such as candidiasis due to elimination of normal body flora.*
- Teach about the function of heart valves and the effects of endocarditis on heart function. Include a simple definition of endocarditis, and explain the risk for its recurrence. *Information helps the client and family understand endocarditis, its treatment, and its effects. Understanding increases compliance.*
- Describe the manifestations of heart failure to be reported to the physician. *Evidence of heart failure may necessitate modification of the treatment regimen or replacement of infected valves.*

- Encourage good dental hygiene and mouth care and regular dental checkups. Teach how to prevent bleeding from the gums and avoid developing mouth ulcers (e.g., gentle toothbrushing, ensuring that dentures fit properly, and avoiding toothpicks, dental floss, and high-flow water devices). *The oropharynx harbors streptococci, which are common causes of endocarditis. Bleeding gums offer an opportunity for bacteria to enter the bloodstream.*
- Encourage the client to avoid people with upper respiratory infections. *Streptococci are normal pathogens in the upper respiratory tract; exposure to people with upper respiratory infections may increase the risk of infection.*
- If anticoagulant therapy is ordered, explain its actions, administration, and major side effects. Identify manifestations of bleeding to be promptly reported to the physician. *Clients with valve disease or a prosthetic valve following infective endocarditis may require continued anticoagulant therapy to prevent thrombi and emboli. Knowledge is vital for appropriate management of anticoagulant therapy and prevention of complications.*

Community-Based Care

When preparing the client with infective endocarditis for home care, provide teaching as outlined for the nursing diagnosis *Ineffective Health Maintenance.* In addition, discuss the following topics:

- Although serious and frightening, infective endocarditis can usually be treated effectively with intravenous antibiotics.
- The importance of promptly reporting any unusual manifestation, such as a change in vision, sudden pain, or weakness, so that interventions to control complications can be promptly implemented.
- The rationale for all treatments and procedures.
- Preventing recurrences of infective endocarditis.
- The importance of maintaining contact with the physician for follow-up care and monitoring for long-term effects such as progressive valve damage and dysfunction.
- If appropriate, explain the risks associated with intravenous drug use.

Provide educational materials on infective endocarditis from the American Heart Association. Refer as appropriate to home health or home intravenous therapy services. Refer the client and family members or significant others as appropriate to a drug or substance abuse treatment program or facility. Provide follow-up care to ensure compliance with the referral and treatment plan.

THE CLIENT WITH MYOCARDITIS

Myocarditis is inflammation of the heart muscle. It usually results from an infectious process, but also may occur as an im-

munologic response, or due to the effects of radiation, toxins, or drugs. In the United States, myocarditis is usually viral, caused by coxsackievirus B. Approximately 10% of people with HIV disease develop myocarditis due to infiltration of the myocardium by the virus. Bacterial myocarditis, much less common, may be associated with endocarditis caused by *S. aureus,* or with diphtheria. Parasitic infections caused by *Trypanosoma cruzi* (Chagas' disease) are common in Central and South America (Kasper et al., 2005).

Incidence and Risk Factors

Myocarditis may occur at any age, and it is more common in men than women. Factors that alter immune response (e.g., malnutrition, alcohol use, immunosuppressive drugs, exposure to radiation, stress, and advanced age) increase the risk for myocarditis. It also is a common complication of rheumatic fever and pericarditis.

Pathophysiology

In myocarditis, myocardial cells are damaged by an inflammatory process that causes local or diffuse swelling and damage. Infectious agents infiltrate interstitial tissues, forming abscesses. Autoimmune injury may occur when the immune system destroys not only the invading pathogen but also myocardial cells. The extent of damage to cardiac muscle ultimately determines the long-term outcome of the disease. Viral myocarditis usually is self-limited; it may progress, however, to become chronic, leading to dilated cardiomyopathy (see the section of this chapter that follows). Severe myocarditis may lead to heart failure.

Manifestations

The manifestations of myocarditis depend on the degree of myocardial damage. The client may be asymptomatic. Nonspecific manifestations of inflammation such as fever, fatigue, general malaise, dyspnea, palpitations, and arthralgias may be present. A nonspecific febrile illness or upper respiratory infection often precedes the onset of myocarditis symptoms. Abnormal heart sounds such as muffled S_1, an S_3, murmur, and pericardial friction rub may be heard. In some cases, manifestations of myocardial infarction, including chest pain, may occur.

INTERDISCIPLINARY CARE

Myocarditis treatment focuses on resolving the inflammatory process to prevent further damage to the myocardium.

Diagnostic studies may be ordered to help diagnose myocarditis:
- *Electrocardiography* may show transient ST-segment and T-wave changes, as well as dysrhythmias and possible heart block.
- *Cardiac markers,* such as the creatinine kinase, troponin T, and troponin I, may be elevated, indicating myocardial cell damage.
- *Endomyocardial biopsy* to examine myocardial cells is necessary to establish a definitive diagnosis; patchy cell necrosis and the inflammatory process can be identified.

If appropriate, antimicrobial therapy is used to eradicate the infecting organism. Antiviral therapy with interferon alpha may be instituted. Immunosuppressive therapy with corticosteroids or other immunosuppressive agents (see Chapter 13 ∞) may be used to minimize the inflammatory response. Heart failure is treated as needed, using ACE inhibitors and drugs. Clients with myocarditis often are particularly sensitive to the effects of digitalis, so it is used with caution. Other medications used in treating myocarditis include antidysrhythmic agents to control dysrhythmias and anticoagulants to prevent emboli.

Bed rest and activity restrictions are ordered during the acute inflammatory process to reduce myocardial work and prevent myocardial damage. Activities may be limited for as long as 6 months to a year (Porth, 2005).

NURSING CARE

Nursing care is directed at decreasing myocardial work and maintaining cardiac output. Both physical and emotional rest are indicated, because anxiety increases myocardial oxygen demand. Hemodynamic parameters and the ECG are monitored closely, especially during the acute phase of the illness. Activity tolerance, urine output, and heart and breath sounds are frequently assessed for manifestations of heart failure. Consider the following nursing diagnoses for the client with myocarditis:
- *Activity Intolerance* related to impaired cardiac muscle function
- *Decreased Cardiac Output* related to myocardial inflammation
- *Fatigue* related to inflammation and impaired cardiac output
- *Anxiety* related to possible long-term effects of the disorder
- *Excess Fluid Volume* related to compensatory mechanisms for decreased cardiac output.

Community-Based Care

Include the following topics when preparing the client with myocarditis for home care:
- Activity restrictions and other prescribed measures to reduce cardiac workload
- Early manifestations of heart failure to report to the physician
- The importance of following the prescribed treatment regimen
- Any recommended dietary modifications (such as a low-sodium diet for heart failure)
- Prescribed medications, their purpose, doses, and possible adverse effects
- The importance of adhering to the treatment plan and recommended follow-up appointments to reduce the risk of long-term consequences such as cardiomyopathy.

THE CLIENT WITH PERICARDITIS

The pericardium is the outermost layer of the heart. It is a two-layered membranous sac with a thin layer of serous fluid (normally no more than 30 to 50 mL) separating the layers. It protects and cushions the heart and the great vessels, provides a barrier to infectious processes in adjacent structures, prevents displacement of the myocardium and blood vessels, and prevents sudden distention of the heart.

Pericarditis is the inflammation of the pericardium. Pericarditis may be a primary disorder or develop secondarily to another cardiac or systemic disorder. Some possible causes of pericarditis are listed in Box 32–4. Acute pericarditis is usually viral and affects men (usually under the age of 50) more frequently than women (Tierney et al., 2005). Pericarditis affects 40% to 50% of clients with end-stage renal disease and uremia. Postmyocardial infarction pericarditis and postcardiotomy (following open-heart surgery) pericarditis also are common.

Pathophysiology

Pericardial tissue damage triggers an inflammatory response. Inflammatory mediators released from the injured tissue cause vasodilation, hyperemia, and edema. Capillary permeability increases, allowing plasma proteins, including fibrinogen, to escape into the pericardial space. White blood cells amass at the site of injury to destroy the causative agent. Exudate is formed, usually fibrinous or serofibrinous (a mixture of serous fluid and fibrinous exudate). In some cases, the exudate may contain red blood cells or, if infectious, purulent material. The inflammatory process may resolve without long-term effects, or scar tissue and adhesions may form between the pericardial layers.

Fibrosis and scarring of the pericardium may restrict cardiac function. Pericardial effusions may develop as serous or purulent exudate (depending on the causative agent) collects in the pericardial sac. Pericardial effusion may be recurrent. Chronic inflammation causes the pericardium to become rigid.

Manifestations

Classic manifestations of acute pericarditis include chest pain, a pericardial friction rub, and fever. Chest pain, the most common symptom, has an abrupt onset. It is caused by inflammation of nerve fibers in the lower parietal pericardium and pleura covering the diaphragm. The pain is usually sharp, may be steady or intermittent, and may radiate to the back or neck. The pain can mimic myocardial ischemia; careful assessment is important to rule out myocardial infarction. Pericardial pain is aggravated by respiratory movements (i.e., deep inspiration and/or coughing), changes in body position, or swallowing. Sitting upright and leaning forward reduces the discomfort by moving the heart away from the diaphragmatic side of the lung pleura.

Although not always present, a *pericardial friction rub* is the characteristic sign of pericarditis. A pericardial friction rub is a leathery, grating sound produced by the inflamed pericardial layers rubbing against the chest wall or pleura. It is heard most clearly at the left lower sternal border with the client sitting up or leaning forward. The rub is usually heard on expiration and may be constant or intermittent.

A low-grade fever (below 100°F [38.4°C]) often develops due to the inflammatory process. Dyspnea and tachycardia are common.

Complications

Pericardial effusion, cardiac tamponade, and constrictive pericarditis are possible complications of acute pericarditis.

Pericardial Effusion

A *pericardial effusion* is an abnormal collection of fluid between the pericardial layers that threatens normal cardiac function. The fluid may consist of pus, blood, serum, lymph, or a combination. The manifestations of a pericardial effusion depend on the rate at which the fluid collects. Although the pericardium normally contains about 30 to 50 mL of fluid, the sac can stretch to accommodate a gradual accumulation of fluid. Over time, the pericardial sac can accommodate up to 2 L of fluid without immediate adverse effects. Conversely, a rapid buildup of pericardial fluid (as little as 100 mL) does not allow the sac to stretch and can compress the heart, interfering with myocardial function. This compression of the heart is known as **cardiac tamponade**. Slowly developing pericardial effusion is often painless and has few manifestations. Heart sounds may be distant or muffled. The client may have a cough or mild dyspnea.

Cardiac Tamponade

Cardiac tamponade is a medical emergency that must be aggressively treated to preserve life. Cardiac tamponade may result from pericardial effusion, trauma, cardiac rupture, or hemorrhage. Rapid collection of fluid in the pericardial sac interferes with ventricular filling and pumping, critically reducing cardiac output.

Classic manifestations of cardiac tamponade result from rising intracardiac pressures, decreased diastolic filling, and decreased cardiac output. A hallmark of cardiac tamponade is a paradoxical pulse, or *pulsus paradoxus*. A paradoxical pulse markedly decreases in amplitude during inspiration. Intrathoracic pressure normally drops during inspiration, enhancing venous return to the right heart. This draws more blood into the right side of the heart than the left, causing the interventricular septum to bulge slightly into the left ventricle. When ventricular filling is impaired by excess fluid in the pericardial sac, this bulging of the interventricular septum decreases cardiac output during inspiration (see Figure 32–8). On palpation of the carotid or femoral artery, the pulse is diminished or absent during inspiration. A drop in systolic blood pressure of more than 10 mmHg during inspiration also indicates pulsus paradoxus.

Other manifestations of cardiac tamponade include muffled heart sounds, dyspnea and tachypnea, tachycardia, a nar-

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BOX 32–4 Selected Causes of Pericarditis

Infectious
- Viruses
- Bacteria
- Tuberculosis
- Fungi
- Syphilis
- Parasites

Noninfectious
- Myocardial and pericardial injury
- Uremia
- Neoplasms
- Radiation
- Trauma or surgery
- Myxedema
- Autoimmune disorders
- Rheumatic fever
- Connective tissue diseases
- Prescription and nonprescription drugs
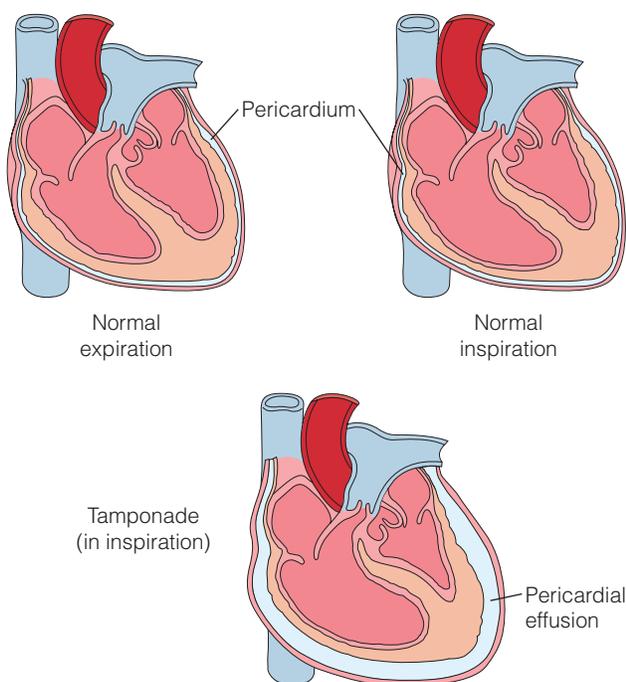- Postcardiac injury

Figure 32–8 ■ Cardiac tamponade. Note increased volume in the right ventricle during inspiration in both the normal heart and the heart affected by a pericardial effusion. In tamponade, fluid in the pericardial sac and the distended right ventricle restrict filling of the left ventricle and consequently, cardiac output.

Figure 32–9 ■ Constrictive pericarditis.
Source: Custom Medical Stock Photo, Inc.

rowed pulse pressure, and distended neck veins (see the box below).

Chronic Constrictive Pericarditis

Chronic pericardial inflammation can lead to scar tissue formation between the pericardial layers. This scar tissue eventually contracts, restricting diastolic filling and elevating venous pressure. Constrictive pericarditis may follow viral infection, radiation therapy, or heart surgery. Its manifestations include progressive dyspnea, fatigue, and weakness. Ascites is common; peripheral edema may develop. Neck veins are distended, and may be particularly noticeable during inspiration (*Kussmaul's sign*). This occurs because the right atrium is unable to dilate to accommodate increased venous return during inspiration. See Figure 32–9 ■.

MANIFESTATIONS of Cardiac Tamponade

- Paradoxical pulse
- Narrowed pulse pressure, hypotension
- Tachycardia
- Weak peripheral pulses
- Distant, muffled heart sounds
- Jugular venous distention
- High central venous pressure
- Decreased level of consciousness
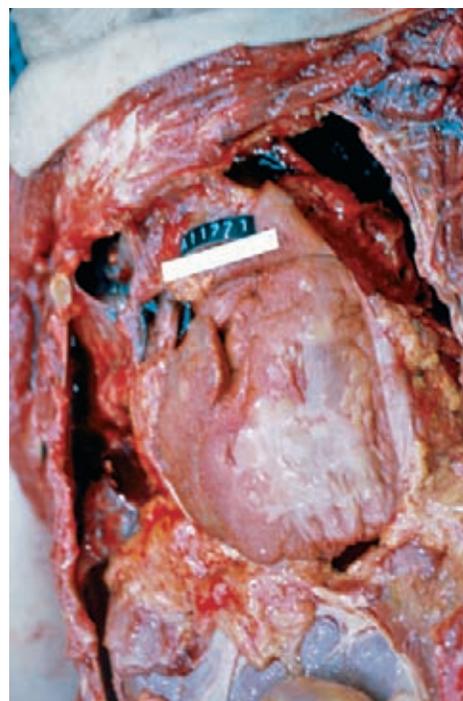- Low urine output
- Cool, mottled skin

INTERDISCIPLINARY CARE

Care for the client with pericarditis focuses on identifying its cause if possible, reducing inflammation, relieving symptoms, and preventing complications. The client is closely monitored for early manifestations of cardiac tamponade so that it can be treated promptly.

Diagnosis

There are no specific laboratory tests to diagnose pericarditis, but tests are often performed to differentiate pericarditis from myocardial infarction.

- *CBC* shows elevated WBCs and an ESR greater than 20 mm/h, indicating acute inflammation.
- *Cardiac enzymes* may be slightly elevated because the inflammatory process extends to involve the epicardial surface of the heart. Cardiac enzymes are typically much lower in pericarditis than in myocardial infarction.
- *Electrocardiography* shows typical changes associated with pericarditis, such as diffuse ST-segment elevation in all leads. This resolves more quickly than changes of acute MI and is not associated with the QRS-complex and T-wave changes typically seen in MI. With a large pericardial effusion, the QRS amplitude may be decreased. Atrial dysrhythmias may occur in acute pericarditis.
- *Echocardiography* is used to assess heart motion, for pericardial effusion, and the extent of restriction.
- *Hemodynamic monitoring* may be used in acute pericarditis or pericardial effusion to assess pressures and cardiac output. Elevated pulmonary artery pressures and venous pressures occur with impaired filling due to pericardial effusion or constrictive pericarditis.
- *Chest x-ray* may show cardiac enlargement if a pericardial effusion is present.

- *Computed tomography (CT) scan* or *magnetic resonance imaging (MRI)* may be used to identify pericardial effusion or constrictive pericarditis.

Medications

Drug treatment for pericarditis addresses its manifestations. Aspirin and acetaminophen may be used to reduce fever. NSAIDs are used to reduce inflammation and promote comfort. In severe cases or with recurrent pericarditis, corticosteroids may be given to suppress the inflammatory response.

Pericardiocentesis

Pericardiocentesis may be done to remove fluid from the pericardial sac for diagnostic or therapeutic purposes (see Figure 30–15 ∞). The physician inserts a large (16- to 18-gauge) needle into the pericardial sac and withdraws excess fluid. The needle is attached to an ECG monitoring lead to help determine if the needle is touching the epicardial surface, which helps prevent piercing the myocardium. Pericardiocentesis may be an emergency procedure for the client with cardiac tamponade. Nursing implications for pericardiocentesis are outlined in Chapter 30 ∞.

Surgery

For recurrent pericarditis or recurrent pericardial effusion, a rectangular piece of the pericardium, or "window," may be excised to allow collected fluid to drain into the pleural space. Constrictive pericarditis may necessitate a partial or total *pericardiectomy,* removal of part or all of the pericardium, to relieve the ventricular compression and allow adequate filling.

NURSING CARE

Health Promotion

While it may not yet be possible to identify many clients at risk for and to prevent acute pericarditis, early identification and treatment of the disorder can reduce the risk of complications. Promptly report a pericardial friction rub or other manifestations of pericarditis in clients with recent acute AMI, cardiac surgery, or systemic diseases associated with a risk for pericarditis.

Assessment

Assessment data to collect from the client with suspected pericarditis includes:

- *Health history:* Complaints of acute substernal or precordial chest pain, effect of movement and breathing on discomfort, pain radiation, associated symptoms; recent acute AMI, heart surgery, or other cardiac disorder; current medications; chronic conditions such as renal failure or a connective tissue or autoimmune disorder.
- *Physical examination:* Vital signs including temperature, variation in systolic BP with respirations; strength of peripheral pulses, variations with respiratory movement; apical pulse, clarity, changes with respiratory movement, presence of a friction rub; neck vein distention; level of consciousness, skin color, and other indicators of cardiac output.
- *Diagnostic tests:* WBC and differential, ESR; cardiac enzyme levels; ECG and echocardiogram reports.

Nursing Diagnoses and Interventions

Nursing care for the client with pericarditis may occur in the acute or community setting. Closely observe for early manifestations of increasing effusion or cardiac tamponade. Priority nursing diagnoses relate to comfort, the risk for tamponade, and effects of the acute inflammatory process.

Acute Pain

Inflamed pericardial layers rubbing against each other and the lung pleura stimulate phrenic nerve pain fibers in the lower portion of the parietal pericardium. Pain is usually acute and may be severe until inflammation resolves.

- Assess chest pain using a standard pain scale and noting the quality and radiation of the pain. Note nonverbal cues of pain (grimacing, guarding behaviors), and validate with the client. *Careful assessment helps identify the cause of pain. The pain of pericarditis may radiate to the neck or back and is aggravated by movement, coughing, or deep breathing. A pain scale allows evaluation of the effectiveness of interventions.*
- Auscultate heart sounds every 4 hours. *Presence of a pericardial friction rub often correlates with the location and severity of the pain.*
- Administer NSAIDs on a regular basis as prescribed with food. Document effectiveness. *NSAIDs reduce fever, inflammation, and pericardial pain. They are most effective when administered around the clock on a consistent basis. Administering the medications with food helps decrease gastric distress.*
- Maintain a quiet, calm environment, and position of comfort. Offer back rubs, heat/cold therapy, diversional activity, and emotional support. *Supportive interventions enhance the effects of the medication, may decrease pain perception, and convey a sense of caring.*

Ineffective Breathing Pattern

Respiratory movement intensifies pericardial pain. In an effort to decrease pain, the client often breathes shallowly, increasing the risk for pulmonary complications.

> **PRACTICE ALERT**
> Document respiratory rate, effort, and breath sounds every 2 to 4 hours. Report adventitious or diminished breath sounds. Shallow, guarded respirations may lead to increased respiratory rate and effort. Poor ventilation of peripheral alveoli may lead to congestion or atelectasis.

- Encourage deep breathing and use of the incentive spirometer. Provide pain medication before respiratory therapy, as needed. *Deep breathing and an incentive spirometer promote alveolar ventilation and prevent atelectasis. Administration of analgesia prior to respiratory treatments improves their effectiveness by decreasing guarding.*
- Administer oxygen as needed. *Supplementary oxygen promotes optimal gas exchange and tissue oxygenation.*
- Place in Fowler's or high-Fowler's position. Assist to a position of comfort. *Appropriate positioning reduces the work of breathing and decreases chest pain due to pericarditis.*

Risk for Decreased Cardiac Output

The acute inflammatory process of pericarditis can lead to significant pericardial effusion and cardiac tamponade. This potentially fatal complication can also occur with chronic pericardial effusion if the amount of fluid exceeds the ability of the pericardial sac to expand. Constrictive pericarditis increases the risk for decreased cardiac output because of restricted cardiac filling.

■ Document vital signs hourly during the acute inflammatory processes. *Frequent assessment allows early recognition of manifestations of decreased cardiac output, such as tachycardia, hypotension, or changes in pulse pressure.*

> **PRACTICE ALERT**
> Assess heart sounds and peripheral pulses, and observe for neck vein distention and paradoxical pulse hourly. Promptly report distant, muffled heart sounds, new murmurs or extra heart sounds, decreasing quality of peripheral pulses, and distended neck veins. Acute pericardial effusion interferes with normal cardiac filling and pumping, causing venous congestion and decreased cardiac output. As the amount of fluid increases in the pericardial sac, heart sounds are obscured. A drop in systolic blood pressure of more than 10 mmHg on inspiration signifies an abnormal response to changes in intrathoracic pressure.

■ Report significant changes or trends in hemodynamic parameters and dysrhythmias. *Compression of the heart interferes with venous return, increasing CVP and right atrial pressures; dysrhythmias may also occur.*

■ Promptly report other signs of decreased cardiac output: decreased level of consciousness; decreased urine output; cold, clammy, mottled skin; delayed capillary refill; and weak peripheral pulses. *These signs of decreased organ and tissue perfusion indicate a significant drop in cardiac output.*

■ Maintain at least one patent intravenous access site. *The client in cardiac tamponade may require rapid intravenous fluid infusion to restore blood volume and administration of emergency drugs to support the circulation.*

■ Prepare for emergency pericardiocentesis and/or surgery as necessary. Provide appropriate explanations and reassurance. Observe for adverse responses during pericardiocentesis. *Excess pericardial fluid must be rapidly evacuated to prevent further compromise of cardiac output and death. Emotional support and explanations reduce the client's and family's anxiety and promote a caring atmosphere.*

Activity Intolerance

In chronic constrictive pericarditis, pericardial adhesions and scarring restrict pericardial compliance, restricting heart filling and movement. Restricted filling and ineffective cardiac contraction decrease the cardiac output. The heart cannot compensate for increased metabolic demands by increasing cardiac output, and cardiac reserve falls significantly.

■ Document vital signs, cardiac rhythm, skin color, and temperature before and after activity. Note any subjective complaints of fatigue, shortness of breath, chest pain, palpitations, or other symptoms with activity. *These parameters help determine the response to increased cardiac work. Increased heart rate and respiratory rate and effort, decreased blood pressure, and dysrhythmias are indicators of activity intolerance. Pallor or cyanosis and cool, clammy, mottled skin are signs of decreased tissue perfusion. Complaints of weakness, shortness of breath, fatigue, dizziness, or palpitations are further evidence of activity intolerance.*

■ Work with the client and physical therapist to develop a realistic, progressive activity plan. Monitor response. Encourage independence, but provide assistance as needed. *Client involvement in planning increases the likelihood of success, as well as the client's self-esteem and sense of control. Promoting self-care provides additional control and independence and enhances self-image. Activity that significantly increases the heart rate (more than 20 beats/min over resting) should be stopped and reassessed for intensity.*

■ Plan interventions and care activities to allow uninterrupted rest and sleep. *This supports healing and restoration of physical and emotional health.*

Community-Based Care

Include the following topics when teaching the client and family in preparation for home care:

■ The importance of continuing anti-inflammatory medications as ordered. Advise to take NSAIDs with food, milk, or antacids to minimize gastric distress, and to notify the physician if unable to tolerate the drug. Instruct to avoid aspirin or preparations containing aspirin while taking NSAIDs because it may interfere with activity.

■ Prescribed medications, including dose, desired and possible adverse effects, and interactions with other drugs or food.

■ Monitoring weight twice weekly because NSAIDs may cause fluid retention.

■ Maintaining fluid intake of at least 2500 mL/day to minimize the risk of renal toxicity due to NSAID use.

■ Measures to maintain activity restriction if ordered. Activity will be gradually increased once the inflammatory process has resolved.

■ Manifestations of recurrent pericarditis, and the importance of reporting these manifestations promptly to the physician.

DISORDERS OF CARDIAC STRUCTURE

THE CLIENT WITH VALVULAR HEART DISEASE

Proper heart valve function ensures one-way blood flow through the heart and vascular system. **Valvular heart disease** interferes with blood flow to and from the heart. Acquired valvular disorders can result from acute conditions, such as infective endocarditis, or from chronic conditions, such as rheumatic heart disease. Rheumatic heart disease is the most common cause of valvular disease (McCance & Huether,

2006). Acute myocardial infarction also can damage heart valves, causing tearing, ischemia, or damage to the papillary muscles that affects valve leaflet function. Congenital heart defects may affect the heart valves, often with no manifestations until adulthood. Aging affects heart structure and function, and also increases the risk for valvular disease.

Physiology Review

The heart valves direct blood flow within and out of the heart. The valves are fibroelastic tissue supported by a ring of fibrous tissue (the annulus) that provides support.

The AV valves, the **mitral** (or *bicuspid*) **valve** on the left and the **tricuspid valve** on the right, separate the atria from the ventricles. These valves normally are fully open during diastole, allowing blood to flow freely from the atria into the ventricles. Rising pressure within the ventricles at the onset of systole (contraction) closes the AV valves, creating the S_1 heart sound ("lub"). The leaflets of the AV valves are connected to ventricular papillary muscles by fibrous *chordae tendineae*. The chordae tendineae prevent the valve leaflets from bulging back into the atria during systole.

The semilunar valves, the **aortic** and **pulmonic valves**, separate the ventricles from the great vessels. They open during systole, allowing blood to flow out of the heart with ventricular contraction. As the ventricle relaxes and intraventricular pressure falls at the beginning of diastole, the higher pressure within the great vessels (the aorta and pulmonary artery) closes these valves, creating the S_2 heart sound ("dup").

Pathophysiology

Valvular heart disease occurs as two major types of disorders: stenosis and regurgitation. Stenosis occurs when valve leaflets fuse together and cannot fully open or close. The valve opening narrows and becomes rigid (Figure 32–10A ■). Scarring of the valves from endocarditis or infarction, and calcium deposits can lead to stenosis. Stenotic valves impede the forward flow of blood, decreasing cardiac output because of impaired ventricular filling or ejection and stroke volume. Because stenotic

A. Thickened and stenotic valve leaflets

B. Retracted fibrosed valve openings

Figure 32–10 ■ Valvular heart disorders. *A*, Stenosis of a heart valve. *B*, An incompetent or regurgitant heart valve.

valves also do not close completely, some backflow of blood occurs when the valve should be fully closed.

Regurgitant valves (also called *insufficient* or *incompetent* valves) do not close completely (Figure 32–10B). This allows regurgitation, or backflow of blood, through the valve into the area it just left. Regurgitation can result from deformity or erosion of valve cusps caused by the vegetative lesions of bacterial endocarditis, by scarring or tearing from myocardial infarction, or by cardiac dilation. As the heart enlarges, the valve *annulus* (supporting ring of the valve) is stretched, and the valve edges no longer meet to allow complete closure.

Valvular disease causes hemodynamic changes both in front of and behind the affected valve. Blood volume and pressures are reduced in front of the valve, because flow is obstructed through a stenotic valve and backflow occurs through a regurgitant valve. By contrast, volumes and pressures characteristically increase behind the diseased valve. These hemodynamic changes may lead to pulmonary complications or heart failure. Higher pressures and compensatory changes to maintain cardiac output lead to remodeling and hypertrophy of the heart muscle.
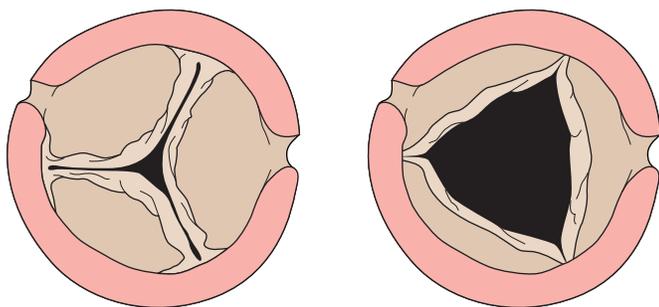
Stenosis increases the work of the chamber behind the affected valve as the heart attempts to move blood through the narrowed opening. Excess blood volume behind regurgitant valves causes dilation of the chamber. In mitral stenosis, for example, the left atrium hypertrophies to generate enough pressure to open and deliver its blood through the narrowed mitral valve. Not all of the blood is delivered before the valve closes, leaving blood to accumulate in the left atrium. This chamber dilates to accommodate the excess volume.

Eventually, cardiac output falls as compensatory mechanisms become less effective. The normal balance of oxygen supply and demand is upset, and the heart begins to fail. Increased muscle mass and size increase myocardial oxygen consumption. The size and workload of the heart exceed its blood supply, causing ischemia and chest pain. Eventually, necrosis occurs and functional muscle is lost. Contractile force, stroke volume, and cardiac output decrease. High pressures on the left side of the heart are reflected backward into the pulmonary system, causing pulmonary edema, pulmonary hypertension, and, eventually, right ventricular failure.

Valvular disorders interfere with the smooth flow of blood through the heart. The flow becomes turbulent, causing a **murmur**, a characteristic manifestation of valvular disease. Table 32–7 describes the murmurs associated with various types of valvular disorders.

Blood forced through the narrowed opening of a stenotic valve or regurgitated from a higher pressure chamber through an incompetent valve creates a jet stream effect (much like water spurting out of a partially occluded hose opening). The physical force of this jet stream damages the endocardium of the receiving chamber, increasing the risk for infective endocarditis.

The higher pressures on the left side of the heart subject its valves (the mitral and aortic valves) to more stress and damage than those on the right side of the heart (the tricuspid and pulmonic). Pulmonic valve disease is the least common of the valvular disorders.

TABLE 32–7 **Heart Murmur Timing and Characteristics**				
MURMUR	**CARDIAC CYCLE TIMING**	**AUSCULTATION SITE**	**CONFIGURATION OF SOUND**	**CONTINUITY**
Mitral stenosis	Diastole	Apical	S_2 ___ S_1	Rumble that increases in sound toward the end, continuous
Mitral regurgitation	Systole	Apex	S_1 ___ S_2	Holosystolic (occurs throughout systole), continuous
Aortic stenosis	Midsystolic	Right sternal border (RSB) 2nd intercostal space (ICS)	S_1 ___ S_2	Crescendo–decrescendo, continuous
Aortic regurgitation	Diastole (early)	3rd ICS, left sternal border (LSB)	S_2 ___ S_1	Decrescendo, continuous
Tricuspid stenosis	Diastole	Lower LSB	S_2 ___ S_1	Rumble that increases in sound toward the end, continuous
Tricuspid regurgitation	Systole	4th ICS, LSB	S_1 ___ S_2	Holosystolic, continuous

Mitral Stenosis

Mitral stenosis narrows the mitral valve, obstructing blood flow from the left atrium into the left ventricle during diastole. It is usually caused by rheumatic heart disease or bacterial endocarditis; it rarely results from congenital defects. It affects females more frequently (66%) than males (Kasper et al., 2005). Mitral stenosis is chronic and progressive.

In mitral valve stenosis, fibrous tissue replaces normal valve tissue, causing valve leaflets to stiffen and fuse. Resulting changes in blood flow through the valve lead to calcification of the valve leaflets. As calcium is deposited in and on the valve, the leaflets become more rigid and narrow the opening further. As the valve leaflets become less mobile, the chordae tendineae fuse, thicken, and shorten. Thromboemboli may form on the calcified leaflets.

The narrowed mitral opening impairs blood flow into the left ventricle, reducing end-diastolic volume and pressure, and decreasing stroke volume. The narrowed opening also forces the left atrium to generate higher pressure to deliver blood to the left ventricle. This leads to left atrial hypertrophy. The left atrium also dilates as obstructed blood flow increases its volume. As the resistance to blood flow increases, high atrial pressures are reflected back into the pulmonary vessels, increasing pulmonary pressures (Figure 32–11 ■). Pulmonary hypertension increases the workload of the right ventricle, causing it to dilate and hypertrophy. Eventually, heart failure occurs.

MANIFESTATIONS Mitral stenosis may be asymptomatic or cause severe impairment. Its manifestations depend on cardiac output and pulmonary vascular pressures. Dyspnea on exertion is typically the earliest manifestation. Others include cough, hemoptysis, frequent pulmonary infections such as bronchitis and pneumonia, paroxysmal nocturnal dyspnea, orthopnea, weakness, fatigue, and palpitations. As the stenosis worsens, manifestations of right heart failure, including jugular venous distention, hepatomegaly, ascites, and peripheral edema, develop. Crackles may be heard in the lung bases. In severe mitral stenosis, cyanosis of the face and extremities may be noted. Chest pain is rare but may occur.

On auscultation, a loud S_1, a split S_2, and a mitral opening snap may be heard. The opening snap reflects high left atrial pressure. The murmur of mitral stenosis occurs during diastole, and is typically low pitched, rumbling, crescendo–decrescendo. It is heard best with the bell of the stethoscope in the apical region. It may be accompanied by a palpable thrill (vibration).

COMPLICATIONS Atrial dysrhythmias, particularly atrial fibrillation, are common due to chronic atrial distention. Thrombi may form and subsequently embolize to the brain, coronary arteries, kidneys, spleen, and extremities—potentially devastating complications.

Women with mitral stenosis may be asymptomatic until pregnancy. As the heart tries to compensate for increased circulating volume (30% more in pregnancy) by increasing cardiac output,
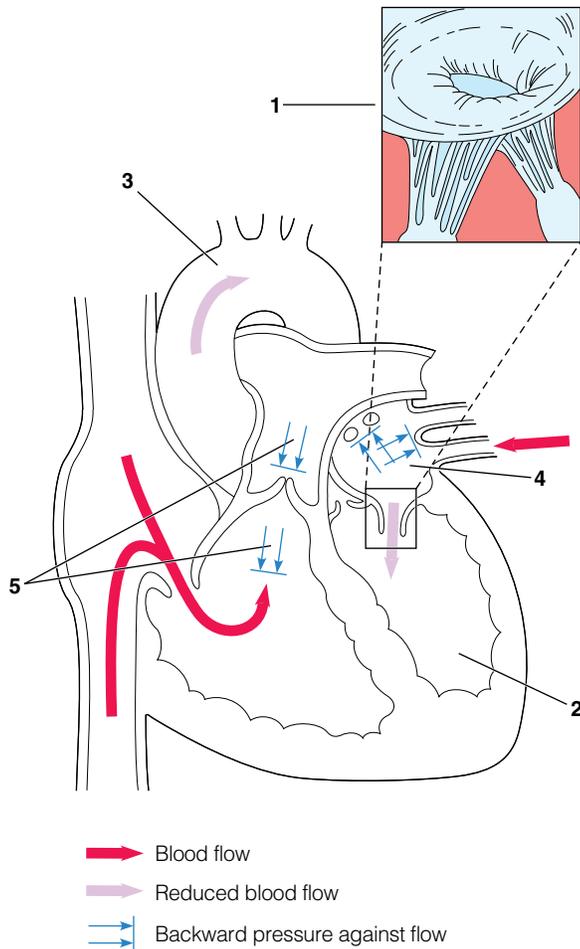
Blood flow

Reduced blood flow

Backward pressure against flow

Figure 32–11 ■ Mitral stenosis. Narrowing of the mitral valve orifice *(1)* reduces blood volume to left ventricle *(2)* reducing cardiac output *(3)*. Rising pressure in the left atrium *(4)* causes left atrial hypertrophy and pulmonary congestion. Increased pressure in pulmonary vessels *(5)* causes hypertrophy of the right ventricle and right atrium.

left atrial pressures rise, tachycardia reduces ventricular filling and stroke volume, and pulmonary pressures increase. Sudden pulmonary edema and heart failure may threaten the lives of the mother and fetus.

Mitral Regurgitation

Mitral regurgitation or *insufficiency* allows blood to flow back into the left atrium during systole because the valve does not close fully. Rheumatic heart disease is a common cause of mitral regurgitation. Men develop mitral regurgitation more frequently than women. Degenerative calcification of the mitral annulus may cause mitral regurgitation in older women. Processes that dilate the mitral annulus or affect the supporting structures, papillary muscles, or the chordae tendineae may cause mitral regurgitation (e.g., left ventricular hypertrophy and MI). Congenital defects also may cause mitral regurgitation.

In mitral regurgitation, blood flows into both the systemic circulation and back into the left atrium through the deformed valve during systole. This increases left atrial volume (Figure 32–12 ■). The left atrium dilates to accommodate its extra volume, pulling

the posterior valve leaflet further away from the valve opening and worsening the defect. The left ventricle dilates to accommodate its increased preload and low cardiac output, further aggravating the problem.

MANIFESTATIONS Mitral regurgitation may be asymptomatic or cause symptoms such as fatigue, weakness, exertional dyspnea, and orthopnea. In severe or acute regurgitation, manifestations of left-sided heart failure develop, including pulmonary congestion and edema. High pulmonary pressures may lead to manifestations of right-sided heart failure.

The murmur of mitral regurgitation is usually loud, high pitched, rumbling, and holosystolic (occurring throughout systole). It is often accompanied by a palpable thrill and is heard most clearly at the cardiac apex. It may be characterized as a cooing or gull-like sound or have a musical quality (Kasper et al., 2005).

MITRAL VALVE PROLAPSE *Mitral valve prolapse (MVP)* is a type of mitral insufficiency that occurs when one or both mitral valve cusps billow into the atrium during ventricular systole.

Blood flow

Reduced blood flow

Backward pressure against flow

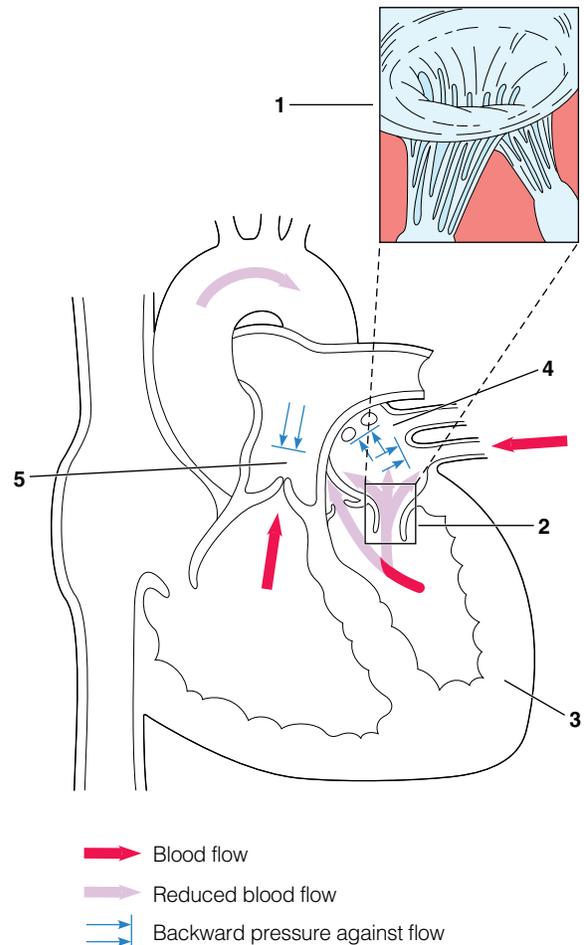**Figure 32–12** ■ Mitral regurgitation. The mitral valve closes incompletely *(1)*, allowing blood to regurgitate during systole from the left ventricle to the left atrium *(2)*. Cardiac output falls; to compensate, the left ventricle hypertrophies *(3)*. Rising left atrial pressure *(4)* causes left atrial hypertrophy and pulmonary congestion. Elevated pulmonary artery pressure *(5)* causes slight enlargement of the right ventricle.

MVP is more common in young women between ages 14 and 30; its incidence declines with age. Its cause often is unclear. It also can result from acute or chronic rheumatic damage, ischemic heart disease, or other cardiac disorders. It commonly affects people with inherited connective tissue disorders such as Marfan syndrome (see the Genetic Considerations box below). Mitral valve prolapse usually is benign, but about 0.01% to 0.02% of people with MVP have thickened mitral leaflets and a significant risk of morbidity and sudden death.

Excess collagen tissue in the valve leaflets and elongated chordae tendineae impair closure of the mitral valve, allowing the leaflets to billow into the left atrium during systole. Some ventricular blood volume regurgitates into the left atrium (Figure 32–13 ■).

MANIFESTATIONS AND COMPLICATIONS Mitral valve prolapse usually is asymptomatic. A midsystolic ejection click or murmur may be audible. A high-pitched late systolic murmur, sometimes described as a "whoop" or "honk," due to the regurgitation of blood through the valve, may develop in MVP. Atypical chest pain is the most common symptom of MVP. It may be left sided or substernal and is frequently related to fatigue, not exertion. Tachydysrhythmias may develop with MVP, causing palpitations, light-headedness, and syncope. Increased sympathetic nervous system tone may cause a sense of anxiety (Woods et al., 2004).

Mitral valve prolapse increases the risk for bacterial endocarditis. Progressive worsening of regurgitation can lead to heart failure. Thrombi may form on prolapsed valve leaflets; embolization may cause transient ischemic attacks (TIAs).

Aortic Stenosis

Aortic stenosis obstructs blood flow from the left ventricle into the aorta during systole. Aortic stenosis is more common in males (80%) than females (Kasper et al., 2005). Aortic stenosis may be idiopathic, or due to a congenital defect, rheumatic damage, or degenerative changes. When rheumatic heart disease is the cause, mitral valve deformity is also often present. RHD destroys aortic valve leaflets, with fibrosis and calcification causing rigidity and scarring. In the older adult, calcific aortic stenosis may result from degenerative changes associ-
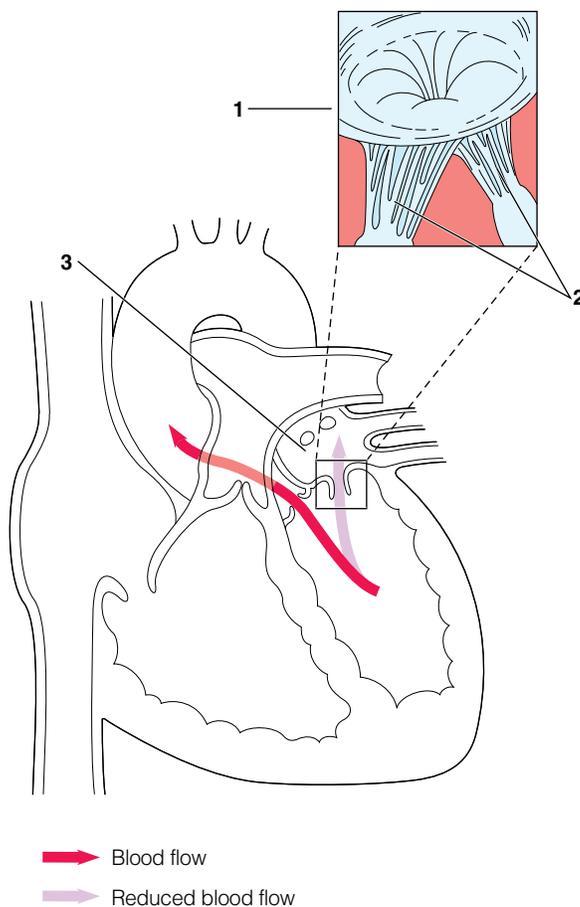

Blood flow
Reduced blood flow

Figure 32–13 ■ Mitral valve prolapse. Excess tissue in the valve leaflets *(1)* and elongated chordae tendineae *(2)* impair mitral valve closure during systole. Some ventricular blood regurgitates into the left atrium *(3)*.

ated with aging. Constant "wear and tear" on this valve can lead to fibrosis and calcification. Idiopathic calcific stenosis generally is mild and does not impair cardiac output.

As aortic stenosis progresses, the valve annulus decreases in size, increasing the work of the left ventricle to eject its volume through the narrowed opening into the aorta. To compensate, the ventricle hypertrophies to maintain an adequate stroke volume and cardiac output (Figure 32–14 ■). Left ventricular compliance also decreases. The additional workload increases myocardial oxygen consumption, which can precipitate myocardial ischemia. Coronary blood flow may also decrease in aortic stenosis. As left ventricular end-diastolic pressure increases because of reduced stroke volume, left atrial pressures increase. These pressures also affect the pulmonary vascular system; pulmonary vascular congestion and pulmonary edema may result.

COURSE AND MANIFESTATIONS Aortic stenosis may be asymptomatic for many years. As the disease progresses and compensation fails, usually between age 50 and 70 years, obstructed cardiac output causes manifestations of left ventricular failure. Dyspnea on exertion, angina pectoris, and exertional syncope are classic manifestations of aortic stenosis. Pulse pressure, an indicator of stroke volume, narrows to 30 mmHg

GENETIC CONSIDERATIONS
Clients with Marfan Syndrome

Marfan syndrome is a genetic (autosomal dominant) connective tissue disorder that affects the skeleton, eyes, and cardiovascular system. Skeletal characteristics include a long, thin body, with long extremities and long, tapering fingers, sometimes called *arachnodactyly* (spider fingers) (Copstead & Banasik, 2005). Joints are hyperextensible, and skeletal deformities such as kyphosis, scoliosis, pigeon chest, or pectus excavatum are common. The potentially life-threatening cardiovascular effects of Marfan syndrome include mitral valve prolapse, progressive dilation of the aortic valve ring, and weakness of arterial walls. People with Marfan syndrome frequently die young, between 30 and 40 years, often due to dissection and rupture of the aorta (Porth, 2005).
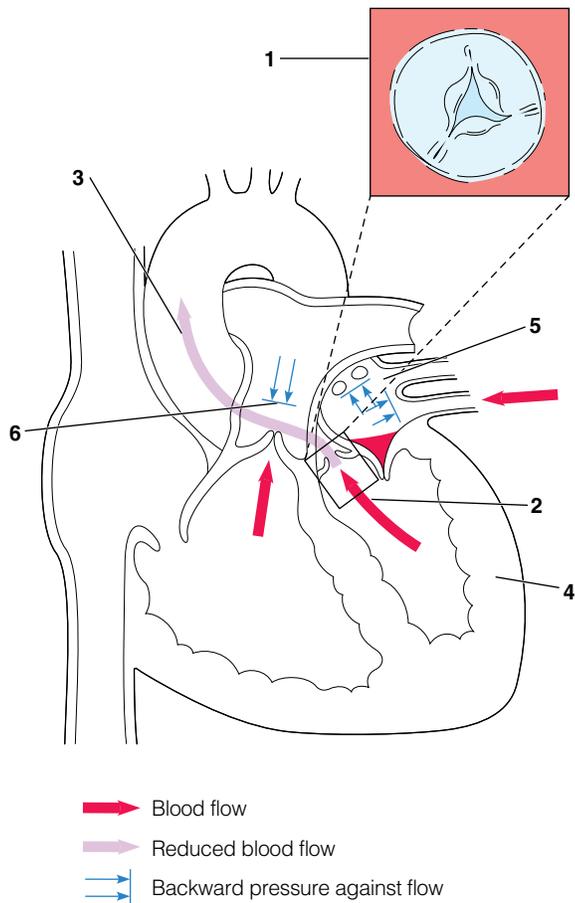
Figure 32–14 ■ Aortic stenosis. The narrowed aortic valve orifice *(1)* decreases the left ventricular ejection fraction during systole *(2)* and cardiac output *(3)*. The left ventricle hypertrophies *(4)*. Incomplete emptying of the left atrium *(5)* causes backward pressure through pulmonary veins and pulmonary hypertension. Elevated pulmonary artery pressure *(6)* causes right ventricular strain.

or less. Hemodynamic monitors show increased left atrial pressure and pulmonary artery wedge pressure, as well as decreased stroke volume and cardiac output.

Aortic stenosis produces a harsh systolic murmur best heard in the second intercostal space to the right of the sternum. This crescendo–decrescendo murmur is produced by turbulence of blood entering the aorta through the stenotic valve. A palpable thrill is often felt. The murmur may radiate to the carotid arteries. Ventricular hypertrophy displaces the cardiac impulse to the left of the midclavicular line. As aortic stenosis progresses, S_3 and S_4 heart sounds may be heard, indicating heart failure and reduced left ventricular compliance.

As cardiac output falls, tissue perfusion decreases. Late in the disease, pulmonary hypertension and right ventricular failure develop. Untreated, symptomatic aortic stenosis has a poor prognosis; 10% to 20% of these clients experience sudden cardiac death (Kasper et al., 2005).

Aortic Regurgitation

Aortic regurgitation, also called *aortic insufficiency,* allows blood to flow back into the left ventricle from the aorta during diastole.

It is more common in males (75%) in its "pure" form; in females, it is commonly associated with coexisting mitral valve disease. Most aortic regurgitation (67%) results from rheumatic heart disease (Kasper et al., 2005). Other causes include congenital disorders, infective endocarditis, blunt chest trauma, aortic aneurysm, syphilis, Marfan syndrome, and chronic hypertension.

In aortic regurgitation, thickened and contracted valve cusps, scarring, fibrosis, and calcification impede complete valve closure. Chronic hypertension and aortic aneurysm may dilate and stretch the aortic valve opening, increasing the degree of regurgitation.

In aortic regurgitation, volume overload affects the left ventricle as blood from the aorta adds to blood received from the atrium during diastole. This increases diastolic left ventricular pressure. Increased preload causes more forceful contractions and a high stroke volume (Figure 32–15 ■). With time, muscle cells hypertrophy to compensate for increased cardiac work and afterload; eventually this hypertrophy compromises cardiac output and increases regurgitation.

High left-ventricular pressures increase left atrial workload and pressure. This pressure is transmitted to the pulmonary vessels, causing pulmonary congestion. The workload of the right
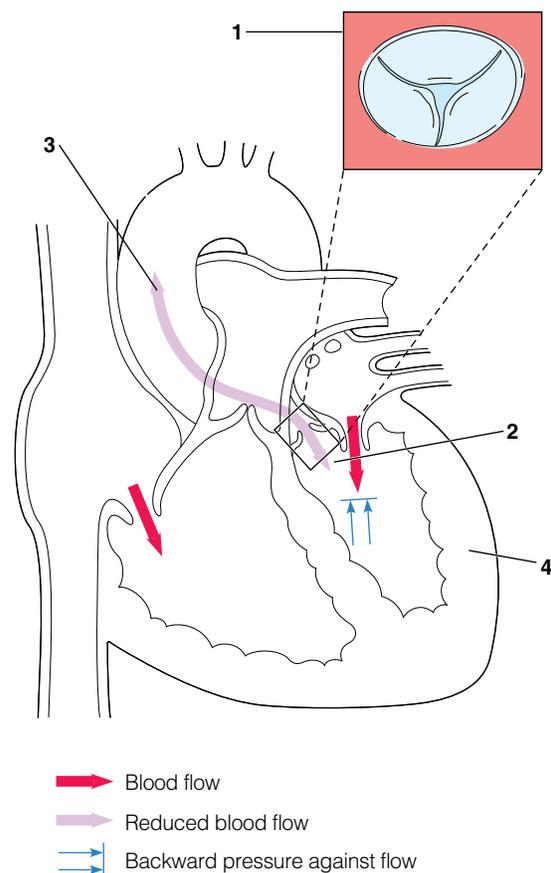

Figure 32–15 ■ Aortic regurgitation. The cusps of the aortic valve widen and fail to close during diastole *(1)*. Blood regurgitates from the aorta into the left ventricle *(2)*, increasing left ventricular volume and decreasing cardiac output *(3)*. The left ventricle dilates and hypertrophies *(4)* in response to the increase in blood volume and workload.

ventricle increases as a result, and right-sided heart failure may develop. Acute aortic regurgitation from traumatic injury or infective endocarditis causes a rapid decline in hemodynamic status from acute heart failure and pulmonary edema, because compensatory mechanisms do not have time to develop.

MANIFESTATIONS   Aortic regurgitation may be asymptomatic for many years, even when severe. The increased stroke volume may cause complaints of persistent palpitations, especially when recumbent. A throbbing pulse may be visible in arteries of the neck; the force of contraction may cause a characteristic head bob (Musset's sign) and shake the whole body. Other symptoms include dizziness and exercise intolerance.

Fatigue, exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea are common in aortic regurgitation. Anginal pain may result from excessive cardiac work and decreased coronary perfusion. Unlike CHD, angina often occurs at night and may not respond to conventional therapy.

The murmur of aortic regurgitation is heard during diastole as blood flows back into the left ventricle from the aorta. It is a "blowing," high-pitched sound heard most clearly at the third left intercostal space. A palpable thrill and ventricular heave may be noted. An S_3 and S_4 may be heard as the heart fails and ventricular compliance diminishes. The apical impulse is displaced to the left.

High systolic and low diastolic pressures cause a widened pulse pressure. The arterial pressure waveform has a rapid upstroke and quickly collapsing downstroke, known as a *water-hammer pulse.* It is caused by the force of rapid and early delivery of the stroke volume into the aorta.

Tricuspid Valve Disorders

Tricuspid stenosis obstructs blood flow from the right atrium to the right ventricle. It usually results from rheumatic heart disease; mitral stenosis often occurs concurrently with tricuspid stenosis.

Fibrosed, retracted tricuspid valve cusps and fused leaflets narrow the valve orifice and prevent complete closure. Right ventricular filling is impaired during diastole, and during systole, some blood regurgitates back into the right atrium. Pressure in the right atrium increases, and it enlarges in response to the increased pressure and workload. This increased right atrial pressure is reflected backward into the systemic circulation. Right ventricular stroke volume decreases, reducing the volume delivered to the pulmonary system and left heart. Stroke volume, cardiac output, and tissue perfusion fall.

Manifestations of tricuspid stenosis relate to systemic congestion and right-sided heart failure. They include increased CVP, jugular venous distention, ascites, hepatomegaly, and peripheral edema. Low cardiac output causes fatigue and weakness. The low-pitched, rumbling diastolic murmur of tricuspid stenosis is most clearly heard in the fourth intercostal space at the left sternal border or over the xiphoid process.

Tricuspid regurgitation usually occurs secondarily to right ventricular dilation. Stretching distorts the valve and its supporting structures, preventing complete valve closure. Left ventricular failure is the usual cause of right ventricular overload; pulmonary hypertension is another cause. The valve may also be damaged by rheumatic heart disease, infective endocarditis, inferior MI, trauma, or other conditions.

Tricuspid regurgitation allows blood to flow back into the right atrium during systole, increasing right atrial pressures. Increased right atrial pressure causes manifestations of right-sided heart failure, including systemic venous congestion and low cardiac output. Atrial fibrillation due to atrial distention is common. The retrograde flow of blood over the deformed tricuspid valve causes a high-pitched, blowing systolic murmur heard over the tricuspid or xiphoid area.

Pulmonic Valve Disorders

Pulmonic stenosis obstructs blood flow from the right ventricle into the pulmonary system. It usually is a congenital disorder, although rheumatic heart disease or cancer also may cause pulmonic stenosis. The right ventricle hypertrophies to generate the pressure needed to pump blood into the pulmonary system. The right atrium also hypertrophies to overcome the high pressures generated in the right ventricle. Right-sided heart failure occurs when the ventricle can no longer generate adequate pressure to force blood past the narrowed valve opening.

Pulmonic stenosis typically is asymptomatic unless severe. Dyspnea on exertion and fatigue are early signs. As the condition progresses, right-sided heart failure develops, with peripheral edema, ascites, hepatomegaly, and increased venous pressures. Turbulent blood flow caused by the narrowed valve generates a harsh, systolic crescendo–decrescendo murmur heard in the pulmonic area, the second left intercostal space.

Pulmonic regurgitation is more common than pulmonary stenosis. It is a complication of pulmonary hypertension, which stretches and dilates the pulmonary orifice, causing incomplete valve closure. Infective endocarditis, pulmonary artery aneurysm, and syphilis also may cause pulmonic regurgitation.

Incomplete valve closure allows blood to flow back into the right ventricle during diastole, decreasing blood flow to the pulmonary circuit. The extra blood increases right ventricular end-diastolic volume. When the ventricle can no longer compensate for the increased volume, right-sided heart failure develops. The murmur of pulmonic regurgitation is a high-pitched, decrescendo, blowing sound heard along the left sternal border during diastole.

INTERDISCIPLINARY CARE

A heart murmur identified during routine physical examination often is the initial indication of valvular disease. If no symptoms are present, close observation for disease progression and prophylactic therapy to prevent infection of the diseased heart may be the only treatment.

Manifestations of heart failure are treated with diet and medications (see the preceding section on heart failure). When medical management is no longer effective, surgery is considered.

Diagnosis

The following diagnostic tests help to identify and diagnose valvular disease. See Chapter 30 ∞ for more information about these tests and related nursing care.

- *Echocardiography* is used routinely to diagnose valvular disease. Thickened valve leaflets, vegetations or growths on valve leaflets, myocardial function, and chamber size can be determined, and pressure gradients across valves and pulmonary artery pressures can be estimated. Either transthoracic or transesophageal echocardiography may be used.
- *Chest x-ray* can identify cardiac hypertrophy, chamber and great vessel enlargement, and dilation of the pulmonary vasculature. Calcification of the valve leaflets and annular openings may also be visible.
- *Electrocardiography* can demonstrate atrial and ventricular hypertrophy, conduction defects, and dysrhythmias associated with valvular disease.
- *Cardiac catheterization* may be used to assess contractility and to determine the pressure gradients across the heart valves, in the heart chambers, and in the pulmonary system.

Medications

Heart failure resulting from valvular disease is treated with diuretics, ACE inhibitors, vasodilators, and possibly digitalis glycosides. Digitalis increases the force of myocardial contraction to maintain cardiac output. Diuretics, ACE inhibitors, and vasodilators reduce preload and afterload. (See the Medication Administration box on pages 1033–1034.)

In clients with valvular disorders, atrial distention often causes atrial fibrillation. Digitalis or small doses of beta-blockers are given to slow the ventricular response. (See Chapter 31 ∞ for more information about atrial fibrillation and its treatment.) Anticoagulant therapy is added to prevent clot and embolus formation, a common complication of atrial fibrillation as blood pools in the noncontracting atria. Anticoagulant therapy also is required following insertion of a mechanical heart valve. See Chapter 35 ∞ for more information about anticoagulant therapy.

Valvular damage increases the risk for infective endocarditis as altered blood flow allows bacterial colonization. Antibiotics are prescribed prophylactically prior to any dental work, invasive procedures, or surgery to minimize the risk of bacteremia (bacteria in the blood) and subsequent endocarditis.

Percutaneous Balloon Valvuloplasty

Percutaneous balloon valvuloplasty is an invasive procedure performed in the cardiac catheterization laboratory. A balloon catheter similar to that used in coronary angioplasty procedures is inserted into the femoral vein or artery. Guided by fluoroscopy, the catheter is advanced into the heart and positioned with the balloon straddling the stenotic valve. The balloon is then inflated for approximately 90 seconds to divide the fused leaflets and enlarge the valve orifice (Figure 32–16 ■). Balloon valvuloplasty is the treatment of choice for symptomatic mitral valve stenosis. It is used to treat children and young adults with aortic stenosis, and may be indicated for older adults who are poor surgical risks, and as a "bridge to surgery" when heart function is severely compromised (Kasper et al., 2005). Nursing care of the client with a balloon valvuloplasty is similar to that of the client following percutaneous coronary revascularization (see page 978).
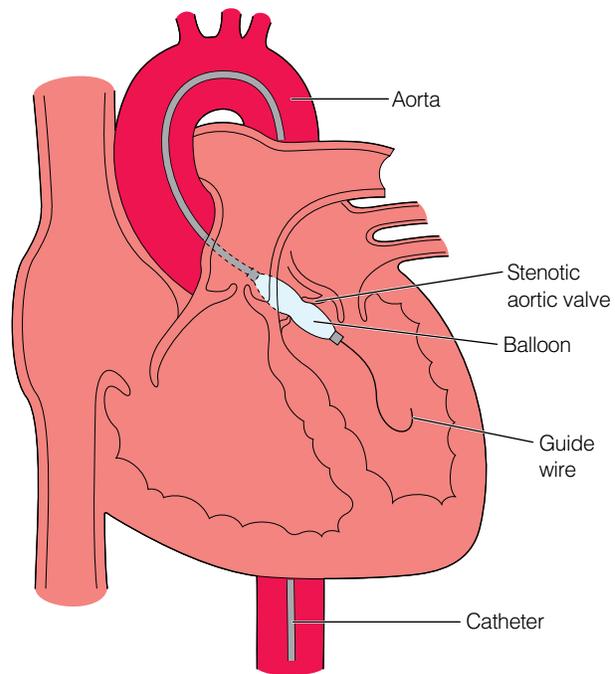

Figure 32–16 ■ Balloon valvuloplasty. The balloon catheter is guided into position straddling the stenosed valve. The balloon is then inflated to increase the size of the valve opening.

Surgery

Surgery to repair or replace the diseased valve may be done to restore valve function, alleviate symptoms, and prevent complications and death. Ideally, diseased valves are repaired or replaced before cardiopulmonary function is severely compromised. The diseased valve is repaired when possible, because the risk for surgical mortality and complications is lower than with valve replacement.

RECONSTRUCTIVE SURGERY *Valvuloplasty* is a general term for reconstruction or repair of a heart valve. Methods include "patching" the perforated portion of the leaflet, resecting excess tissue, debriding vegetations or calcification, and other techniques. Valvuloplasty may be used for stenotic or regurgitant mitral and tricuspid valves, mitral valve prolapse, and aortic stenosis. Common valvuloplasty procedures include the following:

- *Open commissurotomy,* surgical division of fused valve leaflets, is done to open stenotic valves. Fused commissures (junctions between valve leaflets or cusps) are incised, and calcium deposits are debrided as needed.
- *Annuloplasty* repairs a narrowed or an enlarged or dilated valve annulus, the supporting ring of the valve. A prosthetic ring may be used to resize the opening, or stitches and purse-string sutures may be used to reduce and gather excess tissue. Annuloplasty may be used for either stenotic or regurgitant valves.

VALVE REPLACEMENT Valve replacement is indicated when manifestations of valve dysfunction develop, preferably before left heart function is seriously impaired. In general, three factors determine the outcome of valve replacement surgery: (1) heart function at the time of surgery, (2) intraoperative and

TABLE 32–8 **Advantages and Disadvantages of Prosthetic Heart Valves**			
CATEGORY	**TYPES**	**ADVANTAGES**	**DISADVANTAGES**
Mechanical valves	Ball-and-cage Tilting disk	Long-term durability Good hemodynamics	Lifetime anticoagulation Audible click Risk of thromboembolism Infections are harder to treat
Biologic tissue valves	Porcine heterograft Bovine heterograft Human aortic homograft	Low incidence of thromboembolism No long-term anticoagulation Good hemodynamics Quiet Infections are easier to treat	Prone to deterioration Frequent replacement is required

postoperative care, and (3) characteristics and durability of the replacement valve.

Many different prosthetic heart valves are available, including mechanical and biologic tissue valves. Selection depends on the valve hemodynamics, resistance to clot formation, ease of insertion, anatomic suitability, and client acceptance (Rothrock, 2003). The client's age, underlying condition, and contraindications to anticoagulation (such as a desire to become pregnant) also are considered in selecting the appropriate prosthesis. Table 32–8 lists the advantages and disadvantages of biologic and mechanical valves.

Biologic tissue valves may be *heterografts,* excised from a pig (Figure 32–17A ■) or made of calf pericardium, or *homografts* from a human (obtained from a cadaver or during heart transplant). Biologic valves allow more normal blood flow and have a low risk of thrombus formation. As a result, long-term anticoagulation rarely is necessary. They are less durable, however, than mechanical valves. Up to 50% of biologic valves must be replaced by 15 years.

Mechanical prosthetic valves have the major advantage of long-term durability. These valves are frequently used when life expectancy exceeds 10 years. Their major disadvantage is the need for lifetime anticoagulation to prevent the development of clots on the valve.

Most mechanical valves are either a tilting-disk (Figure 32–17B) or a ball-and-cage design. The tilting-disk valve designs are frequently used because they have a lower profile than the caged-ball types, allowing blood to flow through the valve with less obstruction. The St. Jude bileaflet design has good hemodynamics and low risk for clot formation. Both biologic and mechanical valves increase the risk of endocarditis, although its incidence is fairly low.

NURSING CARE

Health Promotion

Preventing rheumatic heart disease is a key element in preventing heart valve disorders. Rheumatic heart disease is a consequence of rheumatic fever (see the previous section of this chapter), an immune process that may be a sequela to beta-hemolytic streptococcal infection of the pharynx (strep throat). Early treatment of strep throat prevents rheumatic fever. Teach individual clients, families, and communities

A   B

Figure 32–17 ■ Prosthetic heart valves. *A,* Mosaic tissue valve. *B,* Medtronic Hall Prosthetic valve.
Courtesy of Medtronic, Inc.

about the importance of timely and effective treatment of strep throat. Emphasize the importance of completing the full prescription of antibiotics to prevent development of resistant bacteria. Prophylactic antibiotic therapy before invasive procedures to prevent infectious endocarditis is an important health promotion measure for clients with preexisting heart disease.

Assessment

Assessment data related to valvular heart disease include the following:

- *Health history:* Complaints of decreasing exercise tolerance, dyspnea on exertion, palpitations; history of frequent respiratory infections; previous history of rheumatic heart disease, endocarditis, or a heart murmur.
- *Physical examination:* Vital signs; skin color and temperature, evidence of clubbing or peripheral edema; neck vein distention; breath sounds; heart sounds and presence of S_3, S_4, or murmur; timing, grade, and characteristics of any murmur; palpate for cardiac heave and thrills; abdominal contour, liver and spleen size.
- *Diagnostic tests:* Echocardiogram and cardiac catheterization reports; cardiac index and cardiac output.

Nursing Diagnoses and Interventions

Nursing priorities include maintaining cardiac output, managing manifestations of the disorder, teaching about the disease process and its management, and preventing complications. Nursing care of the client undergoing valve surgery is similar to that of the client having other types of open-heart surgery, with increased attention to anticoagulation and preventing endocarditis.

Decreased Cardiac Output

Nearly all valve disorders affect ventricular filling and/or emptying, reducing cardiac output. Stenosis of the AV valves impairs ventricular filling and increases atrial pressures. Regurgitation of these valves reduces cardiac output as a portion of the blood in the ventricle regurgitates into the atria during systole. Stenosis of the semilunar valves obstructs ventricular outflow to the great vessels; regurgitation allows blood to flow back into the ventricles, creating higher filling pressures. When compensatory measures fail, heart failure develops.

- Monitor vital signs and hemodynamic parameters, reporting changes from the baseline. *A fall in systolic blood pressure and tachycardia may indicate decreased cardiac output. Increasing pulmonary artery and pulmonary wedge pressures may also indicate decreased cardiac output, causing increased congestion and pressure in the pulmonary vascular system.*

PRACTICE ALERT

Promptly report changes in level of consciousness; distended neck veins; dyspnea or respiratory crackles; urine output less than 30 mL/h; cool, clammy, or cyanotic skin; diminished peripheral pulses; or slow capillary refill. These findings indicate decreased cardiac output and impaired tissue and organ perfusion.

- Monitor intake and output; weigh daily. Report weight gain of 3 to 5 lb within 24 hours. *Fluid retention is a compensatory mechanism that occurs when cardiac output decreases; 2.2 lb (1 kg) of weight equals 1 L of fluid.*
- Restrict fluids as ordered. *Fluid intake may be restricted to reduce cardiac workload and pressures within the heart and pulmonary circuit.*
- Monitor oxygen saturation continuously and arterial blood gases as ordered. Report oxygen saturation less than 95% (or as specified) and abnormal ABG results. *Oxygen saturation levels and ABGs allow assessment of oxygenation.*
- Elevate the head of the bed. Administer supplemental oxygen as ordered. *These measures improve alveolar ventilation and oxygenation.*
- Provide for physical, emotional, and mental rest. *Physical and psychologic rest decrease the cardiac workload.*
- Administer prescribed medications as ordered to reduce cardiac workload. *Diuretics, ACE inhibitors, and direct vasodilators may be prescribed to reduce fluid volume and afterload, reducing cardiac work.*

Activity Intolerance

Altered blood flow through the heart impairs delivery of oxygen and nutrients to the tissues. As the heart muscle fails and is unable to compensate for altered blood flow, tissue perfusion is further compromised. Dyspnea on exertion is often an early symptom of valvular disease.

- Monitor vital signs before and during activities. *A change in heart rate of more than 20 bpm, a change of 20 mmHg or more in systolic BP, and complaints of dyspnea, shortness of breath, excessive fatigue, chest pain, diaphoresis, dizziness, or syncope may indicate activity intolerance.*
- Encourage self-care and gradually increasing activities as allowed and tolerated. Provide for rest periods, uninterrupted sleep, and adequate nutritional intake. *Gradual progression of activities avoids excessive cardiac stress. Encouraging self-care increases the client's self-esteem and sense of power. Adequate rest and nutrition facilitate healing, decrease fatigue, and increase energy reserves.*
- Provide assistance as needed. Suggest use of a shower chair, sitting while brushing hair or teeth, and other energy-saving measures. *Reducing energy expenditure helps maintain a balance of oxygen supply and demand.*
- Consult with cardiac rehabilitation specialist or physical therapist for in-bed exercises and an activity plan. *In-bed exercises may help improve strength.*
- Discuss ways to conserve energy at home. *Information provides practical ways to deal with activity limitations and empowers the client to manage these limitations.*

Risk for Infection

Damaged and deformed valve leaflets and turbulent blood flow through the heart significantly increase the risk of infective endocarditis. Invasive diagnostic and monitoring lines (e.g., cardiac catheterization, hemodynamic monitoring) and disrupted skin with surgery also increase the risk of infection.

- Use aseptic technique for all invasive procedures. *Invasive procedures breach the body's protective mechanisms, poten-*

tially allowing bacteria to enter. Aseptic technique reduces this risk.

> **PRACTICE ALERT**
> Record temperature every 4 hours; notify physician if temperature exceeds 100.5°F (38.5°C). Fever may be an early indication of infection.

- Assess wounds and catheter sites for redness, swelling, warmth, pain, or evidence of drainage. *These signs of inflammation may signal infection.*
- Administer antibiotics as ordered. Ensure completion of the full course. *Antibiotics are used to prevent and treat infection. Completion of the full course of therapy prevents drug-resistant organisms from multiplying.*
- Monitor WBC and differential. Notify physician of leukocytosis or leukopenia. *A high WBC count and increased percentage of immature WBCs (bands) may indicate bacterial infection; a low WBC count may indicate an impaired immune response and increased susceptibility to infection.*

Ineffective Protection

Anticoagulant therapy commonly is prescribed for clients with chronic atrial fibrillation, a history of emboli, and following valve replacement surgery. Although chronic anticoagulant therapy decreases the risk of clots and emboli, it increases the risk for bleeding and hemorrhage.

> **PRACTICE ALERT**
> Monitor the International Normalized Ratio (INR) or prothrombin time (PT or protime). Report an INR > 3.5 or a PT > 2.5 times the normal to the physician. An excessively high INR or PT indicate excessive anticoagulation and an increased risk for bleeding.

- Test stools and vomitus for occult blood. *Bleeding due to excessive anticoagulation may not be apparent.*
- Instruct to avoid using aspirin or other NSAIDs. Encourage reading ingredient labels on over-the-counter drugs; many contain aspirin. *Aspirin and other NSAIDs interfere with clotting and may potentiate the effects of the anticoagulant therapy.*
- Advise using a soft-bristled toothbrush, electric razor, and gentle touch when cleaning fragile skin. *These measures decrease the risk of skin or gum trauma and bleeding.*

> **PRACTICE ALERT**
> Monitor hemoglobin, hematocrit, and platelet count as ordered. Notify the physician of decreasing hemoglobin and hematocrit levels or if the platelet count falls below 50,000/mm^3. Low hemoglobin and hematocrit indicate blood loss. Platelet counts below 50,000/mm^3 significantly increase the risk of bleeding.

Community-Based Care

For most clients, valvular disease is a chronic condition. The client has the primary responsibility for managing effects of the disorder. To prepare the client and family for home care, discuss the following topics:

- Management of symptoms, including any necessary activity restrictions or lifestyle changes
- The importance of adequate rest to prevent fatigue
- Diet restrictions to reduce fluid retention and symptoms of heart failure
- Information about prescribed medications, including purpose, desired and possible adverse effects, scheduling, and possible interactions with other drugs
- The importance of keeping follow-up appointments to monitor the disease and its treatment
- Notifying all healthcare providers about valve disease or surgery to facilitate prescription of prophylactic antibiotics before invasive procedures or dental work
- Manifestations to immediately report to the healthcare provider: increasing severity of symptoms, especially of worsening heart failure or pulmonary edema; signs of transient ischemic attacks or other embolic events; evidence of bleeding, such as joint pain, easy bruising, black and tarry stools, bleeding gums, or blood in the urine or sputum.

Provide referrals to community resources such as home maintenance services, home health services, and structured cardiac rehabilitation programs. Refer the client and family (especially the primary food preparer) to a dietitian or nutritionist for teaching and assistance with menu planning. See the accompanying Nursing Care Plan for additional nursing care and teaching for a client with mitral valve prolapse.

THE CLIENT WITH CARDIOMYOPATHY

The **cardiomyopathies** are disorders that affect the heart muscle itself. They are a diverse group of disorders that affect both systolic and diastolic functions. Cardiomyopathies may be either primary or secondary in origin. Primary cardiomyopathies are idiopathic; their cause is unknown. Secondary cardiomyopathies occur as a result of other processes, such as ischemia, infectious disease, exposure to toxins, connective tissue disorders, metabolic disorders, or nutritional deficiencies. In many cases, the cause of cardiomyopathy is unknown. Close to 27,000 deaths annually are directly attributed to cardiomyopathy. Mortality associated with cardiomyopathy is higher in older adults, men, and African Americans (AHA, 2005).

Pathophysiology

The cardiomyopathies are categorized by their pathophysiology and presentation into three groups: dilated, hypertrophic, and restrictive. Table 32–9 compares the causes, pathophysiology, manifestations, and management of the cardiomyopathies.

Dilated Cardiomyopathy

Dilated cardiomyopathy is the most common type of cardiomyopathy, accounting for 87% of cases (AHA, 2005). Dilated cardiomyopathy also is a common cause of heart failure, accounting for about one in three cases. It is primarily a disease of middle age males; African American males have a higher risk than whites (Kasper et al., 2005).
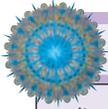
NURSING CARE PLAN   A Client with Mitral Valve Prolapse

Julie Snow, a 22-year-old college student, sees a nurse practitioner at the college health clinic for a physical examination after experiencing palpitations, fatigue, and a headache during midterm examinations. Ms. Snow tells Lakisha Johnson, FNP, "I'm scared that something is wrong with me."

Over the last few months, Ms. Snow has had occasional palpitations that she describes as "feeling like my heart is doing flip-flops." Rarely, these palpitations have been accompanied by a sharp, stabbing pain in her chest that lasts only a few seconds. She initially attributed her symptoms to stress, but she is increasingly concerned because the "attacks" are becoming more frequent. Ms. Snow states that she has "always been healthy," does not smoke, uses alcohol socially, and exercises, albeit intermittently. Ms. Snow admits that she has been drinking a lot of coffee and cola and eating a lot of "junk food" lately.

ASSESSMENT

Ms. Johnson's assessment of Ms. Snow documents the following: height 66 in. (168 cm), weight 140 lb (63.6 kg), T 99.3, BP 118/64, P 82, and R 18. Slightly anxious but in no acute distress. Systolic click and soft crescendo murmur grade II/VI noted on auscultation. Apical impulse at fifth ICS left MCL. Lungs clear to auscultation. Review of remaining systems reveals no apparent abnormalities. An ECG shows sinus rhythm with occasional PACs. Based on the admission history, manifestations, and physical assessment, Ms. Johnson suspects mitral valve prolapse (MVP).

DIAGNOSES

- *Anxiety* related to fear of heart disease and implications for lifestyle
- *Powerlessness* related to unpredictability of symptoms
- *Risk of Infection (Endocarditis)* related to altered valve function

EXPECTED OUTCOMES

- Verbalize an understanding of MVP and its management.
- Discuss ways to decrease or relieve MVP symptoms.
- Acknowledge the risk for endocarditis and identify precautions to prevent it.

PLANNING AND IMPLEMENTATION

- Consult with and refer to cardiologist for continued monitoring and follow-up.
- Teach about MVP, including heart valve anatomy, physiology, and function, common manifestations of MVP, and treatment rationale.
- Discuss symptoms of progressive mitral regurgitation, and the need to report these to the cardiologist.
- Discuss recommended follow-up care and its rationale.

- Allow to verbalize feelings and share concerns about MVP. Encourage to attend an MVP support group meeting.
- Discuss the prognosis for MVP, emphasizing that most clients live normal lives using diet and lifestyle management.
- Instruct to keep a weekly record of symptoms and their frequency for 1 month.
- Discuss lifestyle changes to manage symptoms: aerobic exercise with warm-up and cooldown periods; maintaining adequate fluid intake, especially during hot weather or exercise; relaxation techniques (e.g., meditation, deep-breathing exercises, music therapy, yoga, guided imagery, heat therapy, or progressive muscle relaxation) to perform daily; avoiding caffeine and crash diets; forming healthy eating habits.
- Teach about infective endocarditis risk and prevention with prophylactic antibiotics. Encourage notifying dentist and other healthcare providers of MVP before dental or any invasive procedure.

EVALUATION

After several educational sessions at the college health clinic, Ms. Snow verbalizes an understanding of MVP by explaining heart valve function, listing common manifestations of MVP, and describing indications of deteriorating heart function. She states she will report these manifestations to her cardiologist if they occur. She is given a booklet on MVP for additional reading. She also verbalizes understanding of the risk of endocarditis, and states that she will notify her doctors of her MVP and the need for antibiotics before invasive procedures. Ms. Snow is attending a monthly MVP support group (led by a cardiology clinical nurse specialist) on campus and states, "I am so glad to know I'm not alone! It really helps to know that others are living well with MVP." Her weekly symptom log shows her symptoms are associated with late-night studying and drinking large amounts of coffee and cola. Ms. Snow has moderated her caffeine intake and increased her fluids, relieving her symptoms. In addition, Ms. Snow is taking a relaxation music therapy class. Ms. Snow states that she realizes that she has "the ability to control my life through the choices I make."

CRITICAL THINKING IN THE NURSING PROCESS

1. Develop an action plan for Ms. Snow that outlines specific activities she can use to manage symptoms of MVP.
2. Why are clients with symptomatic MVP encouraged to include regular exercise in their health habits?
3. How does the support of family, friends, and other people with MVP assist MVP clients in managing their condition?
4. What manifestations would indicate a progressive worsening of Ms. Snow's mitral regurgitation?
See Evaluating Your Response in Appendix C.

The cause of dilated cardiomyopathy is unknown, although it appears to frequently result from toxins, metabolic conditions, or infection. Reversible dilated cardiomyopathy may develop due to alcohol and cocaine abuse, chemotherapeutic drugs, pregnancy, and systemic hypertension. Up to 20% of cases of dilated cardiomyopathy may be genetic in origin, most commonly transmitted in an autosomal dominant pattern, although autosomal recessive, X-linked, and mitochondrial

patterns of inheritance also are seen (Kasper et al., 2005; Porth, 2005).

In dilated cardiomyopathy, heart chambers dilate and ventricular contraction is impaired. Both end-diastolic and end-systolic volumes increase, and the left ventricular ejection fraction is substantially reduced, decreasing cardiac output. Left ventricular dilation is prominent; left ventricular hypertrophy is usually minimal. The right ventricle also may be enlarged. Ex-

TABLE 32–9 **Classifications of Cardiomyopathy**			
	DILATED	**HYPERTROPHIC**	**RESTRICTIVE**
Causes	Usually idiopathic; may be secondary to chronic alcoholism or myocarditis	Hereditary; may be secondary to chronic hypertension	Usually secondary to amyloidosis, radiation, or myocardial fibrosis
Pathophysiology	Scarring and atrophy of myocardial cells Thickening of ventricular wall Dilation of heart chambers Impaired ventricular pumping Increased end-diastolic and end-systolic volumes Mural thrombi common	Hypertrophy of ventricular muscle mass Small left ventricular volume Septal hypertrophy may obstruct left ventricular outflow Left atrial dilation	Excess rigidity of ventricular walls restricts filling Myocardial contractility remains relatively normal
Manifestations	Heart failure Cardiomegaly Dysrhythmias S_3 and S_4 gallop; murmur of mitral regurgitation	Dyspnea, anginal pain, syncope Left ventricular hypertrophy Dysrhythmias Loud S_4 Sudden death	Dyspnea, fatigue Right-sided heart failure Mild to moderate cardiomegaly S_3 and S_4 Mitral regurgitation murmur
Management	Management of heart failure Implantable cardioverter-defibrillator (ICD) as needed Cardiac transplantation	Beta-blockers Calcium channel blockers Antidysrhythmic agents ICD, dual-chamber pacing Surgical excision of part of the ventricular septum	Management of heart failure Exercise restriction

tensive interstitial fibrosis (scarring) is evident; necrotic myocardial cells also may be seen (Kasper et al., 2005).

MANIFESTATIONS AND COURSE Manifestations of dilated cardiomyopathy develop gradually. Heart failure often presents years after the onset of dilation and pump failure. Both right- and left-sided failure occur, with dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, weakness, fatigue, peripheral edema, and ascites. Both S_3 and S_4 heart sounds are commonly heard, as well as an AV regurgitation murmur. Dysrhythmias are common, including supraventricular tachycardias, atrial fibrillation, and complex ventricular tachycardias. Untreated dysrhythmias can lead to sudden death (Porth, 2005). Mural thrombi (blood clots in the heart wall) may form in the left ventricular apex and embolize to other parts of the body.

The prognosis of dilated cardiomyopathy is grim; most clients get progressively worse and 50% die within 5 years after the diagnosis; 75% die within 10 years (AHA, 2005).

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is characterized by decreased compliance of the left ventricle and hypertrophy of the ventricular muscle mass. This impairs ventricular filling, leading to small end-diastolic volumes and low cardiac output. About half of all clients with hypertrophic cardiomyopathy have a family history of the disease. It is genetically transmitted in an autosomal dominant pattern (Kasper et al., 2005).

The pattern of left ventricular hypertrophy is unique in that the muscle may not hypertrophy "equally." In a majority of clients, the interventricular septal mass, especially the upper portion, increases to a greater extent than the free wall of the

ventricle. The enlarged upper septum narrows the passageway of blood into the aorta, impairing ventricular outflow. For this reason, this disorder is also known as *idiopathic hypertrophic subaortic stenosis (IHSS)* or *hypertrophic obstructive cardiomyopathy (HOCM).*

MANIFESTATIONS AND COURSE Hypertrophic cardiomyopathy may be asymptomatic for many years. Symptoms typically occur when increased oxygen demand causes increased ventricular contractility. They may develop suddenly during or after physical activity; in children and young adults, sudden cardiac death may be the first sign of the disorder. Hypertrophic cardiomyopathy is the probable or definite cause of death in 36% of young athletes who die suddenly (AHA, 2005). It is hypothesized that sudden cardiac death is due to ventricular dysrhythmias or hemodynamic factors. Predictors of sudden cardiac death in this population include age of less than 30 years, a family history of sudden death, syncopal episodes, severe ventricular hypertrophy, and ventricular tachycardia seen on ambulatory ECG monitoring (Kasper et al., 2005). (For a brief synopsis of a nursing research study regarding family presence during CPR and invasive procedures, see the box below.)

The usual manifestations of hypertrophic cardiomyopathy are dyspnea, angina, and syncope. Angina may result from ischemia due to overgrowth of the ventricular muscle, coronary artery abnormalities, or decreased coronary artery perfusion. Syncope may occur when the outflow tract obstruction severely decreases cardiac output and blood flow to the brain. Ventricular dysrhythmias are common; atrial fibrillation also may develop. Other manifestations of hypertrophic cardiomyopathy include fatigue, dizziness, and palpitations. A harsh, crescendo–decrescendo systolic murmur of variable intensity heard best at the lower left sternal border and apex is characteristic in hypertrophic cardiomyopathy. An S_4 may also be noted on auscultation.

Restrictive Cardiomyopathy

The least common form of cardiomyopathy, *restrictive cardiomyopathy,* is characterized by rigid ventricular walls that impair diastolic filling. Causes of restrictive cardiomyopathy include myocardial fibrosis and infiltrative processes, such as amyloidosis. Fibrosis of the myocardium and endocardium causes excessive stiffness and rigidity of the ventricles. Decreased ventricular compliance impairs filling, with decreased ventricular size, elevated end-diastolic pressures, and decreased cardiac output. Contractility is unaffected, and the ejection fraction is normal.

MANIFESTATIONS AND COURSE The manifestations of restrictive cardiomyopathy are those of heart failure and decreased tissue perfusion. Dyspnea on exertion and exercise intolerance are common. Jugular venous pressure is elevated, and S_3 and S_4 are common. The prognosis for restrictive cardiomyopathy is poor. Most clients die within 3 years, and the systemic nature of the underlying disease process precludes effective treatment.

INTERDISCIPLINARY CARE

With the exception of treating an underlying cause, little can be done to treat either dilated or restrictive cardiomyopathies. For these disorders, treatment focuses on managing heart failure and treating dysrhythmias. Refer to the section of this chapter

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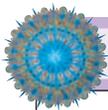
NURSING RESEARCH Evidence-Based Practice: Sudden Cardiac Death

When cardiac arrest occurs or invasive procedures are performed, family members typically are asked to leave the client's care unit. The traditional rationale for this practice is fear of disrupted clinical interventions, trauma of the witnesses, and risk for increased hospital liability. However, in 1995, the Emergency Nurses Association (ENA) adopted a position supporting family presence during invasive procedures, including resuscitation efforts (CPR), as a means of "preserving the wholeness, dignity, and integrity of the family unit from birth to death" (Myers et al., 2000, p. 33). This study evaluated the responses of families, nurses, and physicians to family presence during invasive procedures and CPR.

Results of the study showed that families saw their presence as a positive experience and their right. They viewed themselves as active care partners, and being present met their needs for information and providing comfort and connection with the client. Nurses overwhelmingly supported family presence; attending physicians also demonstrated a positive response. Physician residents were the least supportive of family presence.

IMPLICATIONS FOR NURSING
Family members often are asked to leave the client's side during invasive procedures and CPR with the intention of protecting them from the trauma of witnessing painful or distressing events.

This study clearly showed being present as a positive experience, even when the ultimate outcome was the client's death.

Offering the opportunity to be present and providing information as well as psychologic and emotional support to an appropriate family member during invasive procedures and CPR supports the family unit and the client during times of crisis. Screening is important: People who are combative, emotionally unstable, or have altered mental status (e.g., dementia, alcohol intoxication) probably are not appropriate. It also is important to allow families to decline the invitation without guilt.

CRITICAL THINKING IN CLIENT CARE
1. Identify procedures and situations in which family members are often asked to leave the client's side. When would it be appropriate to allow at least one significant other to remain with the client?
2. How would you present the option and prepare a family member for being present during a traumatic event such as CPR following the sudden death of a young adult with undiagnosed hypertrophic cardiomyopathy?
3. You support family presence during traumatic events and procedures, but your charge nurse does not. What steps might you use to effect a change in policy on your unit?

on heart failure and Chapter 31 ∞ for specific treatment strategies. Treatment of hypertrophic cardiomyopathy focuses on reducing contractility and preventing sudden cardiac death. Strenuous physical exertion is restricted, because it may precipitate dysrhythmias or sudden cardiac death. Dietary and sodium restrictions may help diminish the manifestations.

Diagnosis

Diagnosis begins with a history and physical assessment to rule out known causes of heart failure. Other tests may include the following:

- *Echocardiography* is done to assess chamber size and thickness, ventricular wall motion, valvular function, and systolic and diastolic function of the heart.
- *Electrocardiography* and *ambulatory ECG monitoring* demonstrate cardiac enlargement and detect dysrhythmias.
- *Chest x-ray* shows cardiomegaly, enlargement of the heart, and any pulmonary congestion or edema.
- *Hemodynamic studies* are used to assess cardiac output and pressures in the cardiac chambers and pulmonary vascular system.
- *Radionuclear scans* help identify changes in ventricular volume and mass, as well as perfusion deficits.
- *Cardiac catheterization* and *coronary angiography* may be done to evaluate coronary perfusion, the cardiac chambers, valves, and great vessels for function and structure, pressure relationships, and cardiac output.
- *Myocardial biopsy* uses the transvenous route to obtain myocardial tissue for biopsy. The cells are examined for infiltration, fibrosis, or inflammation.

Medications

The drug regimen used to treat heart failure also is used for dilated or restrictive cardiomyopathy. This includes ACE inhibitors, vasodilators, and digitalis (see the previous section of this chapter). Beta-blockers also may be used with caution in clients with dilated cardiomyopathy. Anticoagulants are given to reduce the risk of thrombus formation and embolization. Antidysrhythmic drugs are avoided if possible due to their tendency to precipitate further dysrhythmias (Kasper et al., 2005).

Beta-blockers are the drugs of choice to reduce anginal symptoms and syncopal episodes associated with hypertrophic cardiomyopathy. The negative inotropic effects of beta-blockers and calcium channel blockers decrease the myocardial contractility, decreasing obstruction of the outflow tract. Beta-blockers also decrease heart rate and increase ventricular compliance, increasing diastolic filling time and cardiac output. Vasodilators, digitalis, nitrates, and diuretics are contraindicated. Amiodarone may be used to treat ventricular dysrhythmias (Kasper et al., 2005).

Surgery

Without definitive treatment, clients with cardiomyopathy develop end-stage heart failure. Cardiac transplant is the definitive treatment for dilated cardiomyopathy. Ventricular assist devices may be used to support cardiac output until a donor heart is available. Transplantation is not a viable option for restrictive cardiomyopathy, because transplantation does not eliminate the underlying process causing infiltration or fibrosis, and eventually the transplanted organ is affected as well. See the section on heart failure for more information about cardiac transplantation.

In severely symptomatic clients with obstructive hypertrophic cardiomyopathy, excess muscle may be surgically resected from the aortic valve outflow tract. The septum is incised, and tissue is removed. This procedure provides lasting improvement in about 75% of clients (Kasper et al., 2005).

An implantable cardioverter-defibrillator (ICD) often is inserted to treat potentially lethal dysrhythmias, reducing the need for antidysrhythmic medications. A dual-chamber pacemaker also may be used to treat hypertrophic cardiomyopathy.

NURSING CARE

Nursing assessment and care for clients with dilated and restrictive cardiomyopathy are similar to those for clients with heart failure. Teaching about the disease process and its management is vital. Some degree of activity restriction often is necessary; assist to conserve energy while encouraging self-care. Support coping skills and adaptation to required lifestyle changes. Provide information and support for decision making about cardiac transplantation if that is an option. Discuss the toxic and vasodilator effects of alcohol, and encourage abstinence. See the nursing care section for heart failure for nursing diagnoses and suggested interventions.

The client with hypertrophic cardiomyopathy requires care similar to that provided for myocardial ischemia; nitrates and other vasodilators, however, are avoided. If surgery is performed, nursing care is similar to that for any client undergoing open-heart surgery or cardiac transplant. Discuss the genetic transmission of hypertrophic cardiomyopathy, and suggest screening of close relatives (parents and siblings).

Provide pre- and postoperative care and teaching as appropriate for clients undergoing invasive procedures or surgery for cardiomyopathy.

Nursing diagnoses that may be appropriate for clients with cardiomyopathy include:

- *Decreased Cardiac Output* related to impaired left ventricular filling, contractility, or outflow obstruction
- *Fatigue* related to decreased cardiac output
- *Ineffective Breathing Pattern* related to heart failure
- *Fear* related to risk for sudden cardiac death
- *Ineffective Role Performance* related to decreasing cardiac function and activity restrictions
- *Anticipatory Grieving* related to poor prognosis.

Community-Based Care

Cardiomyopathies are chronic, progressive disorders generally managed in home and community care settings unless surgery or transplant is planned or end-stage heart failure develops. When teaching the client and family for home care, include the following topics:

- Activity restrictions and dietary changes to reduce manifestations and prevent complications

- Prescribed drug regimen, its rationale, intended and possible adverse effects
- The disease process, its expected ultimate outcome, and treatment options
- Cardiac transplantation, including the procedure, the need for lifetime immunosuppression to prevent transplant rejection, and the risks of postoperative infection and long-term immunosuppression

- Symptoms to report to the physician or for which immediate care is needed.
- Cardiopulmonary resuscitation procedures and available training sites.

Refer the client and family for home and social services and counseling as indicated. Provide community resources such as support groups or the AHA.

EXPLORE MEDIALINK

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

Animations
Digoxin
Hemodynamics

COMPANION WEBSITE www.prenhall.com/lemone
Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Acute Pulmonary Edema
Case Study: Rheumatic Fever
MediaLink Applications
Beta Blockers
Heart Failure
Links to Resources

CHAPTER HIGHLIGHTS

- All of the disorders discussed in this chapter can lead to heart failure, a condition in which the heart is unable to pump effectively to meet the body's needs for blood and oxygen to the tissues.
- Coronary heart disease (myocardial ischemia) and cardiomyopathies are the leading causes of heart failure.
- When the heart starts to fail, compensatory mechanisms are activated to help maintain tissue perfusion. While these mechanisms, including increased contractile force, vasoconstriction, sodium and water retention, and remodeling of the heart, effectively maintain cardiac output in the short term, in the long term they hasten deterioration of heart function.
- The goals of heart failure management are to reduce the workload of the heart and improve its function. Medical management includes administration of drugs such as ACE inhibitors, beta blockers, diuretics, and vasodilators to reduce the workload of the heart, and inotropic medications such as digitalis to improve the strength of cardiac muscle contraction.
- Nursing care of the client with heart failure is primarily supportive and educative, providing the client and family with necessary knowledge and resources to manage this chronic condition.
- Cardiogenic pulmonary edema, a manifestation of severe cardiac decompensation, is a medical emergency, requiring immediate and effective treatment to preserve life. The nurse's role in managing pulmonary edema focuses on supporting respiratory

and cardiac function, administering prescribed medications, and providing reassurance to the client and family.
- Inflammatory and infectious processes, such as rheumatic fever, endocarditis, myocarditis, and pericarditis, can affect any layer of the heart. While some, such as myocarditis and pericarditis, typically are mild and self-limiting, others can have long-term effects on cardiac structure and function.
- Processes such as rheumatic heart disease, endocarditis, and congenital conditions can affect the structure and function of the heart valves, resulting in either stenosis (narrowing) of the valve and restricted flow through it, or regurgitation, backflow of blood through a valve that does not fully close. The mitral and aortic valves are commonly affected due to the higher pressures and increased workload of the left side of the heart.
- Valve disorders may be mild, producing a heart murmur but no functional impairment for the client, or severe, causing symptoms of heart failure even at rest. Repair or replacement of the valve may ultimately be required.
- Cardiomyopathies affect the heart muscle and its ability to stretch during filling and contract effectively. Dilated cardiomyopathy, the most common type, is progressive, ultimately necessitating heart transplant. Hypertrophic cardiomyopathy affects both ventricular filling and outflow through the aortic valve. Surgical resection of excess tissue may relieve its manifestations.

TEST YOURSELF NCLEX-RN® REVIEW

1 In reviewing the physician's admitting notes for a client with heart failure, the nurse notes that the client has an ejection fraction of 25%. The nurse recognizes this as meaning:
   1. ventricular function is severely impaired.
   2. the amount of blood being ejected from the ventricles is within normal limits.
   3. 25% of the blood entering the ventricle remains in the ventricle after systole.
   4. cardiac output is greater than normal, overtaxing the heart.

2 In assessing a client admitted 24 hours previously with heart failure, the nurse notes that the client has lost 2.5 lb (1 kg) of weight, his heart rate is 88 (HR was 105 on admission), and he now has crackles in the bases of his lung fields only. The nurse correctly interprets these data as indicating:
   1. the client's condition is unchanged from admission.
   2. a need for more aggressive treatment.
   3. the treatment regimen is achieving the desired effect.
   4. no further treatment is required at this time as the failure has resolved.

3 The nurse assessing a client admitted with left ventricular failure would recognize which of the following findings as consistent with the diagnosis? (Select all that apply.)
   1. 5 cm jugular vein distention at 30°
   2. complaints of shortness of breath with minimal exertion
   3. substernal chest pain during exercise
   4. bilateral inspiratory crackles to midscapula
   5. fatigue

4 The nurse caring for a client undergoing pulmonary artery pressure monitoring provides appropriate care when he
   1. secures the intravenous line to the bed linens.
   2. maintains flush solution flow by gravity.
   3. reports waveform dampening during wedge pressure measurements.
   4. calibrates and levels the system every shift.

5 Morphine 2 to 5 mg IV as needed for pain and dyspnea is ordered for a client in acute pulmonary edema. The nurse appropriately:
   1. questions this order because no time intervals have been specified.
   2. administers the drug as ordered, monitoring respiratory status.
   3. withholds the drug until the client's respiratory status improves.
   4. administers the drug only when the client complains of chest pain.

6 The nurse notes a grating heart sound when auscultating the apical pulse of a client with pericarditis. The most appropriate response is to

   1. note the finding in the client's medical record.
   2. obtain an electrocardiogram.
   3. immediately notify the physician.
   4. initiate resuscitation measures.

7 An appropriate goal of nursing care for the client with acute infective endocarditis would be:
   1. "Will resume usual activities within 1 week of treatment."
   2. "Will relate the benign and self-limiting nature of the disease."
   3. "Will consider cardiac transplantation as a viable treatment option."
   4. "Will state the importance of continuing intravenous antibiotic therapy as ordered."

8 An expected assessment finding in a client with mitral stenosis being admitted for a valve replacement would be:
   1. muffled heart sounds.
   2. S_3 and S_4 heart sounds.
   3. diastolic murmur heard at the apex.
   4. cardiac heave.

9 A client facing heart valve replacement asks the nurse which type of valve is the best, biologic or mechanical. An appropriate response would be:
   1. the need to take drugs to prevent rejection of biologic tissue is a major consideration.
   2. clotting is a risk with mechanical valves, necessitating anticoagulant drug therapy after insertion.
   3. biologic valves tend to be more durable than mechanical valves.
   4. endocarditis is a risk following valve replacement; it is more easily treated with mechanical valves.

10 The parents of a young athlete who collapsed and died due to hypertrophic cardiomyopathy ask the nurse how it is possible that their son had no symptoms of this disorder before experiencing sudden cardiac death. The nurse responds:
   1. "Exercise causes the heart to contract more forcefully, and can lead to changes in the heart's rhythm or the outflow of blood from the heart in people with hypertrophic cardiomyopathy."
   2. "It is likely that your son had symptoms of the disorder before he died, but he may not have thought them important enough to tell someone about."
   3. "In this type of cardiomyopathy, the ventricle does not fill normally. During exercise, the heart may not be able to meet the body's needs for blood and oxygen."
   4. "Cardiomyopathy results in destruction and scarring of cardiac muscle cells. As a result, the ventricle may rupture during strenuous exercise, leading to sudden death."

See Test Yourself answers in Appendix C.

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UNIT 9 BUILDING CLINICAL COMPETENCE
Responses to Altered Cardiac Function

FUNCTIONAL HEALTH PATTERN: Activity-Exercise

■ Think about clients with cardiac disorders and altered activity and/or exercise patterns for whom you have cared in your clinical experiences.

- What were the clients' major pathophysiologic conditions (medical diagnoses) (e.g., myocardial infarction, heart failure, valvular heart disease)?
- What manifestations did each of these clients have? Were these manifestations similar or different?
- How did each of these clients respond to activities such as talking, eating, completing activities of daily living (ADLs), moving from bed to chair or bathroom, ambulating? Did their heart rates increase or decrease? How about their respiratory rates and effort? Did any of your clients become light-headed or develop chest pain during activity? Did their oxygen saturation levels remain stable or drop during activity?

■ The Activity-Exercise Pattern includes ADLs and patterns of exercise and activity—all of which require the expenditure of energy. The heart functions as a pump to deliver fuel for energy production to the cells. Cardiac disorders affect the heart's pumping ability in two primary ways:

- The heart muscle is directly damaged by ischemic processes (e.g., coronary heart disease), inflammatory responses (e.g., rheumatic carditis, endocarditis), or a primary cardiac muscle disorder (cardiomyopathy).
- Excessive cardiac work (e.g., resulting from hypertension, valve disorders, or congenital defects) prompts compensatory mechanisms to meet cellular needs for fuel; over time, these compensatory mechanisms damage the heart muscle.

■ When the cardiac pump fails, less fuel is delivered to the cells and energy production falls. This in turn affects the client's ability to exercise and maintain ADLs, leading to manifestations such as:

- Fatigue (decreased oxygen and glucose delivery to cells ▶ decreased ATP production)
- Shortness of breath, dyspnea (decreased cardiac output ▶ decreased pulmonary blood flow ▶ carbon dioxide retention stimulates the respiratory center; decreased cellular oxygen ▶ shift to anaerobic cellular metabolism ▶ increased production of nonvolatile acids and stimulation of the respiratory center)
- Tachycardia (decreased stroke volume ▶ decreased cardiac output ▶ catecholamine release ▶ increased heart rate and contractility ▶ increased cardiac work)

■ Priority nursing diagnoses within the Activity-Exercise Pattern that may be appropriate for clients with cardiac disease include:

- *Decreased Cardiac Output* as evidenced by increased heart rate, fatigue, shortness of breath, decreased urine output, impaired mental processing, decreased level of consciousness
- *Activity Intolerance* as evidenced by prolonged heart rate increases following activity, shortness of breath with exercise, activity-related chest pain, fatigue
- *Fatigue* as evidenced by inability to consume a full meal without resting, frequent napping or dozing, expressions of tiredness, weakness
- *Ineffective Tissue Perfusion* as evidenced by cool, dusky skin, decreased urine output, chest pain.

■ Two nursing diagnoses from other functional health patterns often are of high priority for the client with heart disease because the physiologic responses to these problems increase cardiac work:

- *Acute Pain* (Cognitive-Perceptual)
- *Anxiety* (Coping-Stress-Tolerance)

Directions: Read the clinical scenario below and answer the questions that follow. To complete this exercise successfully, you will use not only knowledge of the content in this unit, but also principles related to setting priorities and maintaining client safety.

CLINICAL SCENARIO

You have been assigned to work with the following four clients for the shift on a cardiac telemetry unit. Significant data obtained during report is as follows:

- Betty Williams, age 62, was admitted with acute anterior MI and had successful fibrinolytic therapy 3 days ago. Significant history includes type 2 diabetes, angina, hypertension, and a history of smoking (1.5 to 2 packs per day for 45 years). Her CCU course was uneventful. Current vital signs are T 98.8°F, P 76, R 16, BP 148/88. Cardiac monitor shows normal sinus rhythm with no ectopy. She has been pain free since her nitroglycerin drip was titrated off in the CCU 2 days ago. Lungs sounds are clear.
- Arnold Markus, age 71, was admitted with acute heart failure 2 days ago, treated and stabilized in the CCU, and transferred to the cardiac telemetry unit. Current vital signs are T 98.4°F, P 88, R 18, BP 112/74. Cardiac monitor shows normal sinus rhythm with new iso-

lated premature ventricular contractions (PVCs). Lung sounds are clear in upper lobes with crackles in the left base, and he was able to sleep intermittently during the night using 2 pillows.
- Sandra Thomas, age 53, was admitted for mitral valve replacement 4 days ago (mechanical valve) to treat mitral regurgitation secondary to rheumatic heart disease. Her surgical course was uneventful, but she continues to experience sternal incision pain requiring medication every 4 to 6 hours. Current vital signs are T 99.6°F, P 78, R 20, BP 136/84. Cardiac monitor shows normal sinus rhythm. Lung sounds are diminished at the bases with poor cough effort due to pain. She was last medicated an hour before intershift report with good effect.
- Randall Stevens, age 34, was admitted with infective endocarditis 4 hours ago. Current vital signs are T 102.2°F, P 104, R 18, BP 128/72. Monitor shows sinus tachycardia. He reports chills and generalized weakness.

Questions

1 In what order would you visit these clients after report?
1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the clients presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Betty Williams		
Arnold Markus		
Sandra Thomas		
Randall Stevens		

3 You plan to do client teaching with Ms. Williams later in the day. What are the top three priority items to include in the teaching session for today?
1. signs of MI, smoking cessation, and age as a risk factor
2. smoking cessation, age, and benefits of a cardiac rehabilitation program
3. controlling hypertension and diabetes, and smoking cessation
4. all modifiable and nonmodifiable risk factors for recurrent MI

4 You are completing a beginning-of-shift assessment with Mr. Markus. Which of the following items would take priority?
1. rhythm strip analysis
2. lung sounds
3. oxygen saturation
4. heart sounds

5 After conducting a baseline assessment, which of the following should be a priority item to review with Ms. Thomas?
1. calling for pain medication whenever pain is a 4 or higher on a scale of 1 to 10
2. getting out of bed and ambulating to improve breath sounds
3. care of leg incisions
4. splinting of incision to cough and deep breathe

6 A telephone laboratory report is received for Mr. Stevens. You are most interested in the results for which of the following studies? (Select all that apply.)
1. serum potassium
2. white blood cell count
3. erythrocyte sedimentation rate
4. blood urea nitrogen
5. troponins
6. LDH

7 The charge nurse tells you that the monitor at the nursing station shows that Mr. Markus is having an increase in PVCs. You would be most concerned after reviewing a rhythm strip if the PVCs had which of the following characteristics?
1. had a frequency of 4 per minute
2. could be felt by the patient
3. were unifocal in morphology
4. fell on the T wave of the preceding beat

8 Ms. Williams rings the call bell and tells you that she is having pressure in her chest. On assessment, she rates it as a 5. Which of the following nursing actions should take priority?
1. Obtain a full symptom assessment.
2. Administer a prn nitroglycerin tablet SL.
3. Call for an electrocardiogram.
4. Notify the physician.

9 Which additional piece of cardiac assessment data is the priority for Mr. Stevens at this time?
1. heart sounds to detect murmurs
2. peripheral pulses to determine adequacy of cardiac output
3. point of maximal impulse to detect cardiac enlargement
4. jugular vein filling to determine right heart failure

10 Considering the mitral valve replacement surgery that Ms. Thomas underwent, the nurse would be most concerned about possible risks to medication therapy if she had a history of which of the following disorders listed in the medical record?
1. glaucoma
2. gout
3. duodenal ulcer
4. osteoarthritis

11 A priority item when planning discharge teaching for Mr. Stevens and his family would be which of the following?
1. home administration of antibiotic therapy
2. avoid going to malls or any crowded places
3. expect intermittent shortness of breath indefinitely
4. need for future antibiotic prophylaxis before invasive procedures

12 The physician prescribes captopril (Capoten) for Mr. Markus to reduce his risk for future episodes of acute heart failure. The nurse includes which of the following instructions in her teaching about this drug?
1. Take the drug after meals to prevent gastric upset.
2. Edema of the face and ankles are expected initially; they will clear with time.
3. Change positions slowly, as this drug may cause dizziness or light-headedness.
4. Monitor your pulse daily. Do not take the drug and notify your physician if it is less than 60 bpm.

CASE STUDY

Fritz Matthews, age 72, has had uncontrolled hypertension for the past 2 years. His healthcare provider has tried different antihypertensives to control his disease. Mr. Matthews has been admitted to the hospital four times in the last 6 months. This is his fourth admission with acute heart failure. The physician explained to Mr. Matthews that hypertension that is not controlled can cause an excessive workload to the heart, thus decreasing the heart's ability to pump efficiently.

Mr. Matthews has recently lost his wife and is living alone. He states that he forgets to take his medication at the prescribed times. He also eats a lot of frozen dinners and does not have the energy to cook his meals. Mr. Matthews has gained 10 pounds since his last admission. He has had difficulty sleeping at night and uses two pillows due to shortness of breath. He also reports that he frequently gets up at night, at least twice to urinate. Mr. Matthews complains of fatigue during the day and is unable to even walk to his mailbox. He also tells the nurse that he is tired even on rising in the morning.

Based on Mr. Matthews' physical assessment and complaints of fatigue and orthopnea, *Decreased Cardiac Output* is identified as the highest priority nursing diagnosis.

[Green box — top]
- ↓ CO → SNS stimulation → ↑ HR, contractility, vasoconstriction → ↑ cardiac work
- ↓ CO → ↓ renal perfusion → renin released → vasoconstriction; ADH and aldosterone release → Na+ & water retention → ↑ cardiac work
- ↑ fluid volume and pressure → ventricular remodeling and hypertrophy → ↓ pump effectiveness

affects

Mr. M
72 y.o. male
Heart failure

leads to

assess

[Yellow box]
- 5' 8" tall, 242 lbs.
- T 98.4°, P. 88, R 18
- BP 172/74
- ECG normal sinus rhythm with new isolated PVCs
- Crackles in lung bases bilaterally
- Dyspnea noted with activity
- Dozing frequently, c/o fatigue
- Moderate edema to mid calf bilaterally and of sacral region
- BNP 500 pg/mL

generates

[Green box — right]
- *Excess Fluid Volume* as evidenced by increased heart rate, respiratory crackles, and edema
- *Activity Intolerance* as evidenced by DOE
- *Fatigue* as evidenced by expressions of tiredness

generate nursing diagnosis

prioritized to

Decreased Cardiac Output

expected outcomes

[Orange box]
- Lose 1-2 pounds per week.
- Attend formal cardiac rehab program 3 X/week as prescribed.
- Take antihypertensive medications as prescribed; have BP checked weekly.
- Report increased activity tolerance and decreased fatigue with ADLs.

evaluate

[Purple box — right]
Two days after admission:
- BP 146/79, P 84, R 20; lungs clear; heart rhythm: Isolated PVC's
- BNP 250
- Weight 231 lbs.
- Attended a heart healthy diet program while on cardiac telemetry
- Worked with the dietitian to establish a food chart and plan home meals
- Relates importance of taking medications as ordered; will buy pill-minder box and fill weekly
- Enrolled in cardiac rehab program as prescribed; states he is looking forward to meeting other people "in as bad shape as I am"

[Purple box — bottom]
Independent Interventions
- Discuss strategies for improved medication management.
- Teach to weigh self at same time daily.
- Instruct how to read food labels and select low-sodium, low-fat foods.
- Discuss manifestations to report to primary care provider.
- Discuss benefits of cardiac rehab program and assist to plan ways to incorporate increasing activities into daily regimen.