

# CHAPTER Nursing Care 31 of Clients with Coronary Heart Disease

## LEARNING OUTCOMES

- Discuss the coronary circulation and electrical properties of the heart.
- Compare and contrast the pathophysiology and manifestations of coronary heart disease and common cardiac dysrhythmias.
- Describe interdisciplinary and nursing care for clients with coronary heart disease and/or cardiac dysrhythmias.
- Relate the outcomes of diagnostic tests and procedures to the pathophysiology of cardiac disorders and implications for client responses to the disorder.
- Discuss nursing implications for medications and treatments used to prevent and treat coronary heart disease and dysrhythmias.
- Describe nursing care for the client undergoing diagnostic testing, an interventional procedure, or surgery for coronary heart disease or a dysrhythmia.

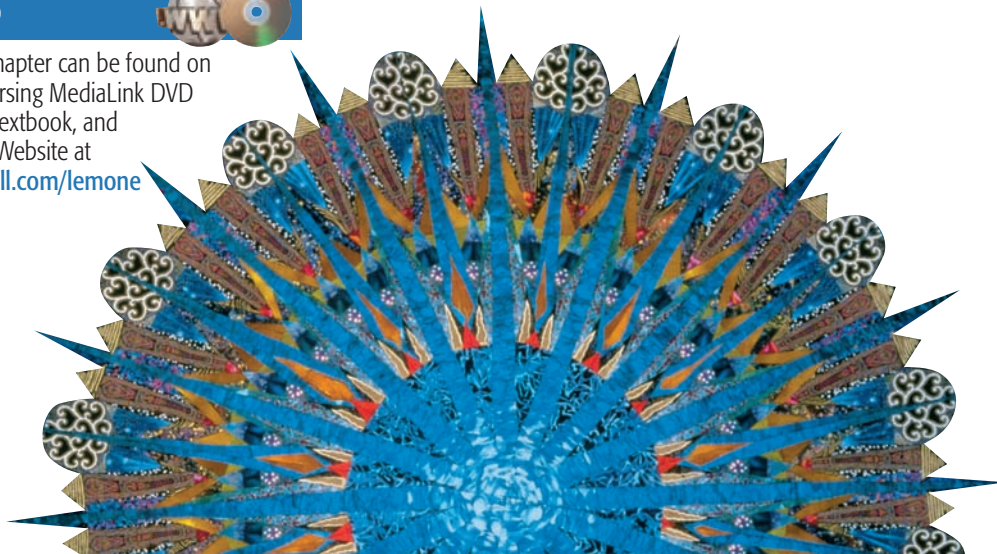
## CLINICAL COMPETENCIES

- Assess functional health status of clients with coronary heart disease and/or a dysrhythmia, including the impact of the disorder on the client's ability to perform activities of daily living and usual tasks.
- Use knowledge of the normal anatomy and physiology of the heart in caring for clients with coronary heart disease.
- Monitor clients with coronary heart disease or dysrhythmias for expected and unexpected manifestations, reporting and recording findings as indicated.
- Use assessed data to select nursing diagnoses, determine priorities of care, and develop and implement individualized nursing interventions for clients with coronary heart disease and dysrhythmias.
- Administer medications and treatments for clients with coronary heart disease and dysrhythmias safely and knowledgeably.
- Integrate interdisciplinary care into nursing care planning and implementation for clients with coronary heart disease and dysrhythmias.
- Provide appropriate teaching for prevention, health promotion, and self-care related to coronary heart disease and dysrhythmias.
- Evaluate the effectiveness of nursing interventions, revising or modifying the plan of care as needed to promote, maintain, or restore functional health for clients with coronary heart disease or dysrhythmias.

### MEDIA LINK



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

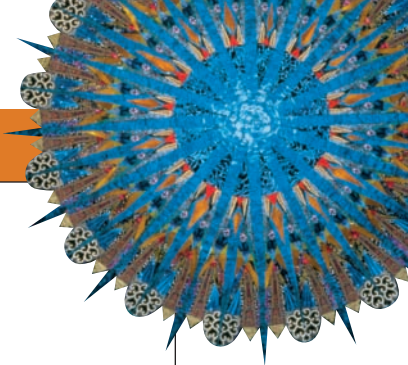


## KEY TERMS

**acute coronary syndrome (ACS)**, 974  
**acute myocardial infarction (AMI)**, 982  
**angina pectoris**, 969  
**atherosclerosis**, 959  
**atrial kick**, 996

**cardiac arrest**, 1002  
**cardiac rehabilitation**, 990  
**cardiovascular disease (CVD)**, 958  
**collateral channels**, 959  
**coronary heart disease**, 958  
**dysrhythmia**, 985  
**ectopic beats**, 996

**heart block**, 996  
**ischemia**, 969  
**normal sinus rhythm (NSR)**, 1000  
**pacemaker**, 1008  
**paroxysmal**, 1000  
**sudden cardiac death**, 1015



Impaired blood flow to the myocardium, changes in the conduction of electrical impulses through the heart, and structural changes in the heart itself affect the heart's ability to fulfill its major purpose: to pump enough blood to meet the body's demand for oxygen and nutrients. Impaired cardiac function, no matter what the underlying cause, affects the client's ability to participate in exercise and activities and to fulfill life roles. Disruptions in cardiac function affect other organ systems as well, potentially leading to organ system failure and death.

**Cardiovascular disease (CVD)** is a generic term for disorders of the heart and blood vessels. CVD is the leading cause of death and disability in the United States. Over 64 million people have some type of cardiovascular disease. The economic costs of CVD, both direct and indirect, to the nation are estimated at \$368 billion annually (National Heart, Lung, and Blood Institute [NHLBI], 2004).

On an encouraging note, however, the incidence of new CVD cases per year is decreasing. Public education aimed at reducing fat intake, increasing exercise, and lowering cholesterol levels

## FAST FACTS

- Heart disease accounts for nearly 696,000 deaths annually in the United States.
- Although heart disease is primarily thought of as a disease affecting older adults, it is the third leading killer of adults ages 25 to 44 (behind accidents and cancer), and the second leading cause of death in adults ages 45 to 64 (behind only cancer).

have made people more aware of risk factors associated with CVD. The mortality rate from heart disease peaked in 1963 and has shown a slow but steady decline since that time.

This chapter focuses on disorders of myocardial blood flow (coronary heart disease) and cardiac rhythm. Disorders of cardiac structure and function are discussed in Chapter 32 ∞. Review the normal anatomy and physiology and nursing assessment of the heart in Chapter 30 ∞ before proceeding with this chapter.

## DISORDERS OF MYOCARDIAL PERFUSION

### THE CLIENT WITH CORONARY HEART DISEASE

**Coronary heart disease (CHD)**, or *coronary artery disease (CAD)*, affects 13.2 million people in the United States and causes more than 500,000 deaths annually (NHLBI, 2004). CHD is caused by impaired blood flow to the myocardium. Accumulation of atherosclerotic plaque in the coronary arteries is the usual cause. Coronary heart disease may be asymptomatic, or may lead to angina pectoris, acute coronary syndrome, myocardial infarction (MI or heart attack), dysrhythmias, heart failure, and even sudden death.

### Incidence and Prevalence

Many risk factors for CHD can be controlled through lifestyle modification. In fact, with increased public awareness of risk factors related to CHD, mortality rates are declining by about 3.3% per year. Nevertheless, CHD remains a major public health problem. Heart disease is the leading cause of death for all U.S. ethnic groups except Asian females (NHLBI, 2004). See the accompanying box. Nurses are in a prime position to encourage and support positive lifestyle changes by teaching

## FOCUS ON CULTURAL DIVERSITY

### Heart Disease

- Native Americans (American Indians and Alaska Natives) have the highest prevalence of coronary heart disease, followed by whites, African Americans, Mexican Americans and native Hawaiians, and Asian Americans.
- People with less than a high school diploma have the highest rate of CHD; people with a bachelor's degree or higher have the lowest.
- Economic status is a factor; the rate of CHD in people with a family income of less than \$35,000 per year have a significantly higher rate than those with incomes greater than \$75,000 per year.
- Regionally, the rate of CHD is highest in the South and lowest in the West.

and promoting healthy living practices. Individual choices can and do affect health.

The highest incidence of CHD is in the Western world, mainly in white males age 45 and older. Both men and women are affected by coronary heart disease; in women, however, the onset

is about 10 years later because of the heart-protective effects of estrogen. After menopause, women's risk is equal to that of men.

## Physiology Review

The two main coronary arteries, the left and the right, supply blood, oxygen, and nutrients to the myocardium. They originate in the root of the aorta, just outside the aortic valve. The *left main coronary artery* divides to form the anterior descending and circumflex arteries. The *anterior descending* artery supplies the anterior interventricular septum and the left ventricle, including the apex of the heart. The *circumflex* branch supplies the lateral wall of the left ventricle. The *right coronary artery* supplies the right ventricle and forms the posterior descending artery. The *posterior descending* artery supplies the posterior portion of the heart (see Figure 30–5).

Blood flow through the coronary arteries is regulated by several factors. Aortic pressure is the primary factor. Other factors include the heart rate (most flow occurs during diastole, when the muscle is relaxed), metabolic activity of the heart, blood vessel tone (constriction), and collateral circulation. Although there are no connections between the large coronary arteries, small arteries are joined by **collateral channels**. If large vessels are gradually occluded, these channels enlarge, providing alternative routes for blood flow (Porth, 2005).

## Pathophysiology

Coronary atherosclerosis is the most common cause of reduced coronary blood flow.

### Atherosclerosis

**Atherosclerosis** is a progressive disease characterized by *atheroma* (plaque) formation, which affects the intimal and medial layers of large and midsize arteries. See *Pathophysiology Illustrated: Coronary Heart Disease* on pages 960–961.

Atherosclerosis is initiated by unknown precipitating factors that cause lipoproteins and fibrous tissue to accumulate in the arterial wall. Although the precise mechanisms are unknown, abnormal lipid metabolism and injury to or inflammation of endothelial cells lining the artery appear to be key to its development.

In the bloodstream, lipids are transported attached to proteins called apoproteins. High levels of certain *lipoproteins*, a type of apoprotein, increase the risk of atherosclerosis. *Low-density lipoproteins*, which are high in cholesterol, carry cholesterol to peripheral tissues where some of it is released to be taken up and incorporated into cells for use in producing energy. *Very-low-density lipoproteins*, large molecules primarily composed of triglycerides and cholesterol, carry triglycerides to muscle and fat cells. When the triglycerides are released into these tissues, the remainder of the molecule is a low-density lipoprotein. *High-density lipoproteins*, in contrast, attract cholesterol, returning it from peripheral tissues to the liver.

Hyperlipidemia itself may damage arterial endothelium. Other potential mechanisms of vessel injury include excessive pressures within the arterial system (hypertension), toxins found in cigarette smoke, infections, and inflammation (Cop-

stead & Banasik, 2005). Endothelial damage promotes platelet adhesion and aggregation, and attracts leukocytes to the area.

At the site of injury, *atherogenic* (atherosclerosis-promoting) lipoproteins collect in the intimal lining of the artery. These lipoproteins appear to actually bind with the extracellular portion of the vessel endothelium. Macrophages migrate to the injured site as part of the inflammatory process. Contact with platelets, cholesterol, and other blood components stimulates smooth muscle cells and connective tissue within the vessel wall to proliferate abnormally. Although blood flow is not affected at this stage, this early lesion appears as a yellowish fatty streak on the inner lining of the artery. Fibrous plaque develops as smooth muscle cells enlarge, collagen fibers proliferate, and blood lipids accumulate. The lesion protrudes into the arterial lumen and is fixed to the inner wall of the intima. It may invade the muscular media layer of the vessel as well. The developing plaque not only gradually occludes the vessel lumen but also impairs the vessel's ability to dilate in response to increased oxygen demands. Fibrous plaque lesions often develop at arterial bifurcations or curves or in areas of narrowing. As the plaque expands, it can produce severe stenosis or total occlusion of the artery.

The final stage of the process is the development of *atheromas*, complex lesions consisting of lipids, fibrous tissue, collagen, calcium, cellular debris, and capillaries. These calcified lesions can ulcerate or rupture, stimulating thrombosis. The vessel lumen may be rapidly occluded by the thrombus, or it may embolize to occlude a distal vessel.

Plaque formation may be *eccentric*, located in a specific, asymmetric region of the vessel wall, or *concentric*, involving the entire vessel circumference. Manifestations of the process usually do not appear until about 75% of the arterial lumen has been occluded.

Atherosclerosis tends to develop where arteries *bifurcate* or branch. Certain vessels have a higher likelihood of being affected, including the coronary arteries (the left anterior descending artery in particular), the renal arteries, the bifurcation of the carotid arteries, and branching sections of peripheral arteries. In addition to obstructing or occluding blood flow, atherosclerosis weakens arterial walls, and is a major cause of aneurysm in vessels such as the aorta and iliac arteries.

### Myocardial Ischemia

Myocardial cells become ischemic when the oxygen supply is inadequate to meet metabolic demands. The critical factors in meeting metabolic demands of cardiac cells are coronary perfusion and myocardial workload. Coronary perfusion can be affected by several different mechanisms:

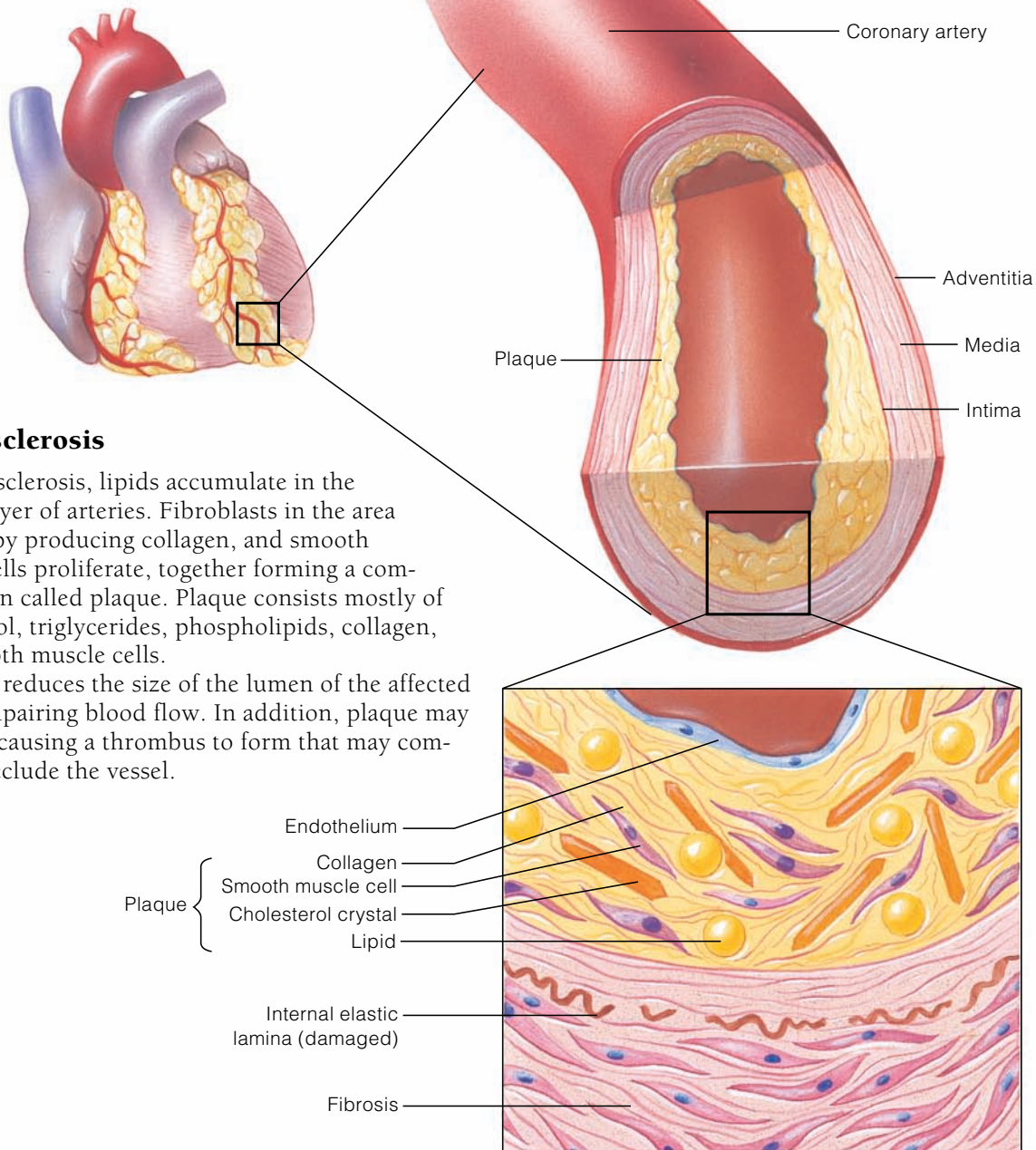
- One or more vessels may be partially occluded by large, stable areas of plaque.
- Platelets can aggregate in narrowed vessels, forming a thrombus.
- Normal or already narrowed vessels may spasm.
- A drop in blood pressure may lead to inadequate flow through coronary vessels.
- Normal autoregulatory mechanisms that increase flow to working muscles may fail (Copstead & Banasik, 2005).

# PATHOPHYSIOLOGY ILLUSTRATED

## Coronary Heart Disease

Coronary heart disease usually is due to *atherosclerosis*, occlusion of the coronary arteries by fibrous, fatty plaque. Coronary heart disease is manifested by *angina pectoris*, *acute coronary syndrome*, and/or *myocardial infarction*. Risk factors for

coronary heart disease include age (over 50 years), heredity, smoking, obesity, high serum cholesterol levels, hypertension, and diabetes mellitus. Other factors, such as diet and lack of exercise, also contribute to the risk of CHD.



### Atherosclerosis

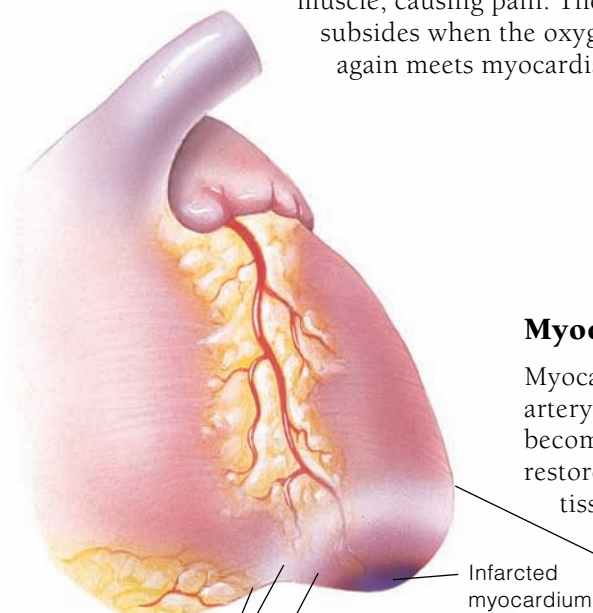
In atherosclerosis, lipids accumulate in the intimal layer of arteries. Fibroblasts in the area respond by producing collagen, and smooth muscle cells proliferate, together forming a complex lesion called plaque. Plaque consists mostly of cholesterol, triglycerides, phospholipids, collagen, and smooth muscle cells.

Plaque reduces the size of the lumen of the affected artery, impairing blood flow. In addition, plaque may ulcerate, causing a thrombus to form that may completely occlude the vessel.

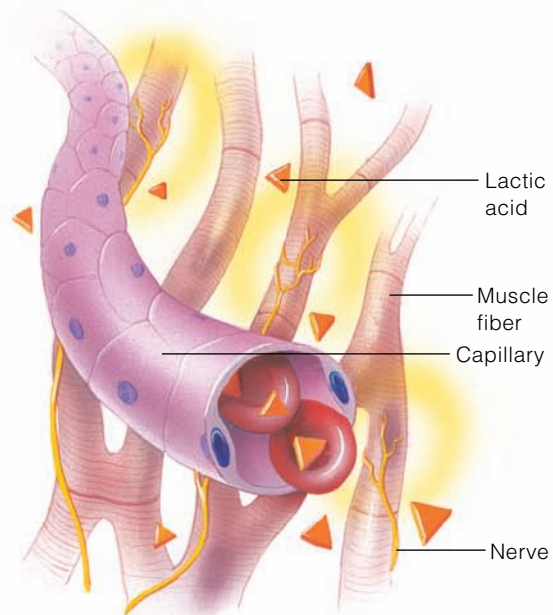
## Angina Pectoris

Angina is characterized by episodes of chest pain, usually precipitated by exercise and relieved by rest. When myocardial oxygen needs are greater than partially occluded vessels can supply, myocardial cells become ischemic and shift to anaerobic metabolism.

Anaerobic metabolism produces lactic acid that stimulates nerve endings in the muscle, causing pain. The pain subsides when the oxygen supply again meets myocardial demand.



Infarcted myocardium  
Ischemic myocardium  
Injured (stunned) myocardium

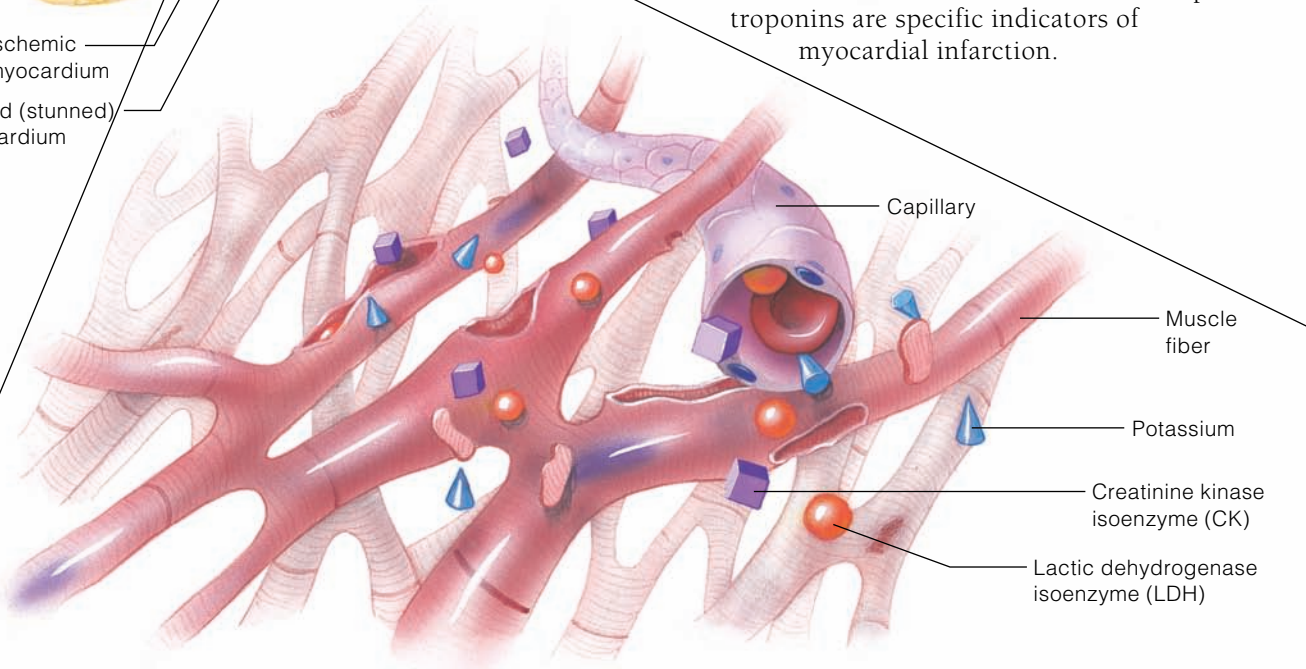


Lactic acid  
Muscle fiber  
Capillary  
Nerve

## Myocardial Infarction

Myocardial infarction occurs when complete obstruction of a coronary artery interrupts blood supply to an area of myocardium. Affected tissue becomes ischemic and eventually dies (infarcts) if the blood supply is not restored. The necrotic area is bordered by an area of injured or damaged tissue, which is in turn surrounded by an area of ischemic tissue.

As myocardial cells die, they lyse and release various cardiac isoenzymes into the circulation. Elevated serum levels of creatinine kinase (CK) and cardiac-specific troponins are specific indicators of myocardial infarction.



Capillary  
Muscle fiber  
Potassium  
Creatinine kinase isoenzyme (CK)  
Lactic dehydrogenase isoenzyme (LDH)

TABLE 31–1 Factors Contributing to Myocardial Ischemia

CORONARY PERFUSION	MYOCARDIAL WORKLOAD	BLOOD OXYGEN CONTENT
<ul style="list-style-type: none"> <li>■ Atherosclerosis</li> <li>■ Thrombosis</li> <li>■ Vasospasm</li> <li>■ Poor perfusion pressure</li> </ul>	<ul style="list-style-type: none"> <li>■ Rapid heart rate</li> <li>■ Increased preload, afterload, or contractility</li> <li>■ Increased metabolic demands (e.g., hyperthyroidism)</li> </ul>	<ul style="list-style-type: none"> <li>■ Reduced atmospheric oxygen pressure</li> <li>■ Impaired gas exchange</li> <li>■ Low red blood cells and hemoglobin content</li> </ul>

Workload is affected by the heart rate, myocardial contractility, preload (the amount of blood in the ventricles just prior to systole), and afterload (the peripheral pressure that must be overcome to move blood out of the heart into the circulation). The oxygen content of the blood and hematocrit are contributing factors to myocardial ischemia. Table 31–1 lists factors that may lead to myocardial ischemia.

Myocardial cells have limited supplies of adenosine triphosphate (ATP) for energy storage. When myocardial workload increases or the supply of blood and oxygen falls, cellular ATP stores are quickly depleted, affecting their contractility. Cellular metabolism switches from an efficient aerobic process to anaerobic metabolism. Lactic acid accumulates, and cells are damaged. If blood flow is restored within 20 minutes, aerobic metabolism and contractility are restored, and cellular repair begins (McCance & Huether, 2006). Continued ischemia results in cell necrosis and death (infarction).

Coronary heart disease is generally divided into two categories, chronic ischemic heart disease and acute coronary syndromes. *Chronic ischemic heart disease* includes stable and vasospastic angina, and silent myocardial ischemia. In women, angina is the most common presenting symptom of CHD. *Acute coronary syndromes* range from unstable angina to myocardial infarction (Porth, 2005). Acute coronary syndromes and

myocardial infarction are the most common presenting symptoms of CHD in men. These disorders are discussed in the following sections of this chapter.

## Risk Factors

The causes of atherosclerosis are not known, but certain risk factors have been linked with the development of atherosclerotic plaques. The Framingham Heart Study provided vital research into the relationship between risk factors and the development of heart disease (Box 31–1). Research into CHD is ongoing, looking at causative factors, manifestations, and protective measures for many populations. Risk factors for CHD are frequently classified as *nonmodifiable*, factors that cannot be changed, and *modifiable*, those factors that can be changed (Table 31–2).

### Nonmodifiable Risk Factors

*Age* is a nonmodifiable risk factor. More than 50% of heart attack victims are 65 or older; 80% of deaths due to myocardial infarction occur in this age group. *Gender* and *genetic factors* also are nonmodifiable risk factors for CHD. Men are affected by CHD at an earlier age than women. A family history of CHD in a male first-degree relative younger than age 55 or a female first-degree relative younger than 65 years is identified as a risk

### BOX 31–1 The Framingham Heart Study

The Framingham Heart Study (FHS) is an ongoing, significant clinical research study that has provided data about cardiovascular disease for over 50 years. The study was initiated in 1948 with an original study group of 5209 participants in the town of Framingham, Massachusetts. Every 2 years, this original group is evaluated for cardiovascular “events” via their medical history, physical findings, and diagnostic testing. Children of the original group have also been studied as part of the Framingham Offspring Study. It was in reports of the Framingham study that the term “risk factor” first appeared.

#### Implications for Nursing

The data collected from both the Framingham Heart Study and the Framingham Offspring Study provide a rich database from which to develop evidence-based approaches for clients with heart disease. A major application of these research findings to practice is in primary preventive education, for example, through community cardiovascular health programs. As noted in the text, although research shows that increased public awareness of cardiovascular risk factors has lowered morbidity and mortality from heart disease, heart disease remains the number-one killer in the United States. Education about the effects of

lifestyle on the cardiovascular system must begin in the early school years and be reinforced throughout the formative years. When healthy choices become habit, cardiac disease will be reduced.

A second application of these findings is in interdisciplinary treatment. Nurses should keep up to date on the latest strategies for medical treatment so that they can provide accurate rationales to clients and formulate effective nursing treatment plans that complement medical management strategies. The result is better communication, a sense of collegiality and teamwork, and positive client outcomes.

#### Critical Thinking in Client Care

1. What kinds of strategies can be used in elementary school settings to teach cardiovascular health in a fun, informative manner?
2. Which healthcare providers should be included in a multidisciplinary effort to encourage clients to modify their lifestyles?
3. What changes do you need to make in your lifestyle to role model heart healthy living?

TABLE 31–2 Risk Factors for Coronary Heart Disease

NONMODIFIABLE	MODIFIABLE	
	Pathophysiologic	Lifestyle
Age Men ≥45 years Women ≥55 years	Hyperlipidemia Elevated LDL cholesterol Elevated triglycerides Low HDL cholesterol	Cigarette smoking Obesity Physical inactivity Atherogenic diet
Gender	Hypertension	<i>Women only:</i> use of oral contraceptives, hormone replacement therapy
Heredity	Diabetes mellitus <i>Women only:</i> premature menopause Emerging risk factors: Elevated homocysteine levels Thrombogenic factors Inflammatory factors Impaired fasting glucose	

factor for CHD (National Cholesterol Education Program [NCEP], 2002).

### Modifiable Risk Factors

Modifiable risk factors include lifestyle factors and pathologic conditions that predispose the client to developing CHD. Disease conditions that contribute to CHD include hypertension, diabetes mellitus, and hyperlipidemia. Although these conditions are not a matter of choice, they are modifiable risk factors that can often be controlled through medication, weight control, diet, and exercise.

Behavioral or lifestyle factors can be controlled or completely eliminated. Lifestyle changes require significant commitment by the client; ongoing support from the healthcare team is vital for success.

**HYPERTENSION** *Hypertension* is consistent blood pressure readings greater than 140 mmHg systolic or 90 mmHg diastolic. Hypertension is common, affecting more than one-third of people over age 50 in the United States. Its prevalence is higher in African Americans than in Hispanics, and higher in Hispanics than in white Americans. Hypertension damages the endothelial cells of arteries, possibly by excess pressure and altered characteristics of blood flow. This damage can stimulate the development of atherosclerotic plaque.

**DIABETES** *Diabetes mellitus* contributes to CHD in several ways. Diabetes is associated with higher blood lipid levels, a higher incidence of hypertension, and obesity—all risk factors in their own right. In addition, diabetes affects the endothelium of blood vessels, contributing to the process of atherosclerosis. Hyperglycemia and hyperinsulinemia, altered platelet function, elevated fibrinogen levels, and inflammation also are thought to play a role in the development of atherosclerosis in people with diabetes.

**ABNORMAL BLOOD LIPIDS** *Hyperlipidemia* is an abnormally high level of blood lipids and lipoproteins. Lipoproteins carry cholesterol in the blood. Low-density lipoproteins (LDLs) are the primary carriers of cholesterol. High levels of LDL (memory cue: LDLs = less desirable lipoproteins) promote atherosclerosis because LDL deposits cholesterol on artery walls. Table 31–3 lists desirable and high-risk levels for total and LDL cholesterol. In contrast, high-density lipoproteins (HDLs = highly desirable lipoproteins) help clear cholesterol from the arteries, transporting it to the liver for excretion. HDL levels above 35 mg/dL have a protective effect, reducing the risk of CHD; in contrast, HDL levels lower than 35 mg/dL are associated with an increased risk for CHD. Triglycerides, compounds of fatty acids bound to glycerol and used for fat storage by the body, are carried on very low-density lipoprotein

TABLE 31–3 Classification of Serum Cholesterol and Triglyceride Values\*

	TOTAL CHOLESTEROL (MG/DL)	LDL CHOLESTEROL (MG/DL)	TRIGLYCERIDE (MG/DL)
Optimal		Less than 100	
Desirable	Under 200	100–129	<150
Borderline high	200 to 239	130 to 159	150 to 199
High	240 or higher	160 to 189	200 to 499
Very high		≥190	≥500

\*As defined by the National Blood, Lung, and Heart Institute's National Cholesterol Education Program.

(VLDL) molecules. Elevated triglycerides also contribute to the risk for CHD.

**CIGARETTE SMOKING** *Cigarette smoking* is an independent risk factor for CHD, responsible for more deaths from CHD than from lung cancer or pulmonary disease (Woods et al., 2004). The effects of smoking on the cardiovascular system are dose dependent (NCEP, 2002). The male cigarette smoker has two to three times the risk of developing heart disease of the nonsmoker; the female who smokes has up to four times the risk. For both men and women who stop smoking, the risk of mortality from CHD is reduced by half. Second-hand (or environmental) tobacco smoke also increases the risk of death from CHD, by as much as 30% (Woods et al., 2004). Tobacco smoke promotes CHD in several ways. Carbon monoxide damages vascular endothelium, promoting cholesterol deposition. Nicotine stimulates catecholamine release, increasing blood pressure, heart rate, and myocardial oxygen use. Nicotine also constricts arteries, limiting tissue perfusion (blood flow and oxygen delivery). Further, nicotine reduces HDL levels and increases platelet aggregation, increasing the risk of thrombus formation.

### FAST FACTS

- Cigarette smoking is the leading independent risk factor for coronary heart disease and a primary target of risk factor management.

**OBESITY** *Obesity* (excess adipose tissue), generally defined as a body mass index (BMI) of 30 kg/m<sup>2</sup> or greater, and fat distribution affect the risk for CHD. Obese people have higher rates of hypertension, diabetes, and hyperlipidemia. In the Framingham study, obese men over age 50 had twice the incidence of CHD and acute myocardial infarction (MI) of those who were within 10% of their ideal weight. Central obesity, or intra-abdominal fat, is associated with an increased risk for CHD. The best indicator of central obesity is the waist circumference. A waist-to-hip ratio of greater than 0.8 (women) or 0.9 (men) increases the risk for CHD.

**PHYSICAL INACTIVITY** *Physical inactivity* is associated with a higher risk of CHD. Research data indicate that people who maintain a regular program of physical activity are less prone to developing CHD than sedentary people. Cardiovascular benefits of exercise include increased availability of oxygen to the heart muscle, decreased oxygen demand and cardiac workload, and increased myocardial function and electrical stability. Other positive effects of regular physical activity include decreased blood pressure, blood lipids, insulin levels, platelet aggregation, and weight.

**DIET** *Diet* is a risk factor for CHD, independent of fat and cholesterol intake. Diets high in fruits, vegetables, whole grains, and unsaturated fatty acids appear to have a protective effect. The underlying factors are not clear, but probably relate to nutrients such as antioxidants, folic acid, other B vitamins, omega-3 fatty acids, and other unidentified micronutrients (NCEP, 2002).

**EMERGING RISK FACTORS** Recent research demonstrates a link between elevated serum *homocysteine levels* and CHD.

Until menopause, women have lower homocysteine levels than men, which may partially explain their lower risk for CHD. Homocysteine levels are negatively correlated with serum folate and dietary folate intake; that is, increasing folate intake lowers homocysteine levels.

Based on evidence that aspirin and antiplatelet therapies reduce the risk for MI, *clot-promoting factors* are identified as CHD risk factors. *Inflammation* also has recently been identified as a risk factor. Inflammatory processes may increase the development of atherosclerotic plaque, and are implicated in plaque rupture (NCEP, 2002). Inflammation also promotes clot formation at the site of ruptured plaque. Although identified as risk factors, it is not generally recommended that clients routinely be tested for these factors.

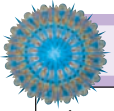
**METABOLIC SYNDROME** *The metabolic syndrome*, a group of metabolic risk factors occurring in an individual, is a strong risk factor for CHD (Box 31–2). The metabolic syndrome has emerged as a risk factor for premature CHD that is equal to cigarette smoking. Three underlying causes of metabolic syndrome have been identified: overweight/obesity, physical inactivity, and genetic factors. It is closely associated with *insulin resistance*, impaired tissue responses to insulin. Genetic factors play a role in insulin resistance, as do the acquired factors of abdominal obesity and physical inactivity (NCEP, 2002).

**RISK FACTORS UNIQUE TO WOMEN** Risk factors unique to women include *premature menopause*, *oral contraceptive use*, and *hormone replacement therapy (HRT)*. At menopause, serum HDL levels drop and LDL levels rise, increasing the risk of CHD. Early menopause (natural or surgically induced) increases the risk of CHD and MI. Women who have bilateral oophorectomy before age 35 without hormone replacement are eight times more likely to have an MI than women experiencing natural menopause. Estrogen replacement therapy reduces the risk of CHD and MI in these women. Oral contraceptives, by contrast, increase the risk for myocardial infarction, particularly in women who also smoke. This increased risk is due to the tendency of oral contraceptives to promote clotting, and their effects on blood pressure, serum lipids, and glucose tolerance (Woods et al., 2004). The Women's Health Initiative randomized trial of HRT showed an increased risk for CHD in previously healthy women taking a commonly prescribed combination of estrogen and progestin (Writing Group, 2002). This well-controlled research study (see the box on the next page) was terminated early when it showed a small but significant increased risk for CHD, stroke, pulmonary embolism, and invasive breast cancer in women taking HRT.

### BOX 31–2 Characteristics of the Metabolic Syndrome

- Abdominal obesity
- Abnormal blood lipids (low HDL, high triglycerides)
- Hypertension
- Elevated fasting blood glucose
- Clotting tendency
- Inflammatory factors





## NURSING RESEARCH Evidence-Based Practice: Postmenopausal Women

The Women's Health Initiative (WHI) is studying the risks and benefits of strategies to reduce the incidence of heart disease, breast and colorectal cancer, and fractures in postmenopausal women (Writing Group, 2002). A group of 161,809 postmenopausal women between ages 50 and 79 were originally enrolled in WHI trials. Of these women, a subgroup of 16,608 women with intact uteri became part of a randomized trial to assess the risks and benefits of HRT, using the most frequently prescribed combined hormone (estrogen and progestin [Prempro]) replacement in the United States.

After a mean of 5.2 years of follow-up, this study was stopped due to convincing evidence that the risk for invasive breast cancer exceeded the benefits of HRT. The study also demonstrated increased risks for coronary heart disease, stroke, deep vein thrombosis, and pulmonary embolism, although overall mortality was not affected. HRT reduced the risk for colorectal cancer and hip fracture in this study group. The risk for CHD appears to be independent of other CHD risk factors such as age, ethnicity, hypertension, diabetes, smoking, obesity, and other identified risk factors.

### IMPLICATIONS FOR NURSING

Nurses often are in the position of advising women about menopause, its manifestations, and hormone replacement ther-

apy. While HRT does reduce unpleasant menopausal effects such as night sweats and hot flashes, and it reduces the risk of osteoporosis and subsequent fractures, it carries associated risks. Advise each client about the risks and benefits of HRT, clearly presenting the evidence. Suggest alternative strategies to reduce menopausal symptoms, such as complementary medicines (see Chapter 51 ∞). Encourage measures such as weight-bearing exercise, calcium supplements, and a diet high in fiber and antioxidants to reduce the risks for osteoporosis, fracture, and colorectal cancer. Ultimately, each client will make her own decision about postmenopausal HRT.

### CRITICAL THINKING IN CLIENT CARE

1. What factors might you suggest that a client consider when deciding whether to use HRT for menopausal manifestations and risks?
2. In this study, the increased risk for CHD was not related to the duration of time taking HRT, whereas the increased risk for stroke and invasive breast cancer emerged more than 1 year after randomization (stroke in the second through fifth year of the study, breast cancer within several years following randomization). Will this data affect your advice to menopausal women inquiring about HRT? If so, how?

### FAST FACTS

- Risk factors for coronary heart disease are those factors that promote atherosclerosis and plaque development.
- Angina pectoris, acute coronary syndromes, and myocardial infarction are the manifestations of myocardial ischemia and coronary heart disease due to atherosclerosis.
- Atherosclerosis also is the primary underlying cause of stroke and peripheral vascular disease; therefore, the risk factors for atherosclerosis also are the risk factors for coronary heart disease, including angina, acute coronary syndromes, and myocardial infarction.

## INTERDISCIPLINARY CARE



Care of clients with coronary heart disease focuses on aggressive risk factor management to slow the atherosclerotic process and maintain myocardial perfusion. Until manifestations of chronic or acute ischemia are experienced, the diagnosis often is presumptive, based on history and the presence of risk factors.

### Diagnosis


Laboratory testing is used to assess for risk factors such as an abnormal blood lipid profile (elevated triglyceride and LDL levels and decreased HDL levels).

- *Total serum cholesterol* is elevated in hyperlipidemia. A *lipid profile* includes triglyceride, HDL, and LDL levels as well, and enables calculation of the ratio of HDL to total cholesterol. The ratio should be at least 1:5, with 1:3 being the ideal ratio. Elevated lipid levels are associated with an increased risk of atherosclerosis (see Table 31–3). In clients with a

strong family history of premature CHD or familial hypercholesterolemia, *lipoprotein (a)* also may be measured. Elevated levels of Lp(a) may independently increase the risk of CHD. Other subsets of blood lipids may also be measured in selected clients. See Chapter 30 ∞ for nursing care related to lipid profile studies.

Diagnostic tests to identify subclinical (asymptomatic) CHD may be indicated when multiple risk factors are present.

- *C-reactive protein* is a serum protein associated with inflammatory processes. Recent evidence suggests that elevated blood levels of this protein may be predictive of CHD.
- The *ankle-brachial blood pressure index (ABI)* is an inexpensive, noninvasive test for peripheral vascular disease that may be predictive of CHD. The systolic blood pressure in the brachial, posterior tibial, and dorsalis pedis arteries is measured by Doppler. An ABI of <0.9 in either leg indicates the presence of peripheral arterial disease and a significant risk for CHD.
- *Exercise ECG testing* may be performed. ECGs are used to assess the response to increased cardiac workload induced by exercise. The test is considered “positive” for CHD if myocardial ischemia is detected on the ECG (depression of the ST segment by greater than 3 mm; see Figure 31–1), the client develops chest pain, or the test is stopped due to excess fatigue, dysrhythmias, or other symptoms before the predicted maximal heart rate is achieved.
- *Electron beam computed tomography (EBCT)* creates a three-dimensional image of the heart and coronary arteries that can reveal plaque and other abnormalities. This noninvasive test requires no special preparation, and can identify clients at risk for developing myocardial ischemia.

- **Myocardial perfusion imaging** (see the section on angina that follows) may be used to evaluate myocardial blood flow and perfusion, both at rest and during stress testing (exercise or mental stress). These diagnostic tests are further explained in Chapter 30  and the section on angina. Perfusion imaging studies are costly, and therefore not recommended for routine CHD risk assessment.

## Risk Factor Management

Conservative management of CHD focuses on risk factor modification, including smoking, diet, exercise, and management of contributing conditions.

**SMOKING** Smoking cessation reduces the risk for CHD within months after quitting and improves cardiovascular status. People who quit reduce their risk by 50%, regardless of how long they smoked before quitting. For women, the risk becomes equivalent to a nonsmoker within 3 to 5 years of smoking cessation (Woods et al., 2004). In addition, stopping smoking improves HDL levels, lowers LDL levels, and reduces blood viscosity. All smokers are advised to quit. Health promotion activities focus on preventing children, teenagers, and adults from starting to smoke.

**DIET** Dietary recommendations by the National Cholesterol Education Program (2002) include reduced saturated fat and cholesterol intake, and strategies to lower LDL levels (Table 31–4). Most fats are a mixture of saturated and unsaturated fatty acids. The highest proportions of saturated fat are found in whole-milk products, red meats, and coconut oil. Nonfat dairy products, fish, and poultry as primary protein sources are recommended. Solidified vegetable fats (e.g., margarine, shortening) contain *trans* fatty acids, which behave more like saturated fats. Soft margarines and vegetable oil spreads contain low levels of *trans* fatty acids, and should be used instead of butter, stick margarine, and shortening. Monounsaturated fats, found in olive, canola, and peanut oils, actually lower LDL and cholesterol levels. Certain


cold-water fish, such as tuna, salmon, and mackerel, contain high levels of omega-3 fatty acids, which help raise HDL levels, and decrease serum triglycerides, total serum cholesterol, and blood pressure.


In addition, increased intake of soluble fiber (found in oats, psyllium, pectin-rich fruit, and beans) and insoluble fiber (found in whole grains, vegetables, and fruit) is recommended. Folic acid and vitamins B<sub>6</sub> and B<sub>12</sub> affect homocysteine metabolism, reducing serum levels. Leafy green vegetables (e.g., spinach and broccoli) and legumes (e.g., black-eyed peas, dried beans, and lentils) are rich sources of folate. Meat, fish, and poultry are rich in vitamins B<sub>6</sub> and B<sub>12</sub>. Vitamin B<sub>6</sub> also is found in soy products; B<sub>12</sub> is in fortified cereals. Increased intake of antioxidant nutrients (vitamin E, in particular) and foods rich in antioxidants (fruits and vegetables) appears to increase HDL levels and have a protective effect on CHD.

In middle-aged and older adults, moderate alcohol intake may reduce the risk for CHD (NCEP, 2002). Consumption of no more than two drinks per day for men or one drink per day for women is recommended. A drink is 5 ounces of wine, 12 ounces of beer, or 1.5 ounces of whiskey. People who do not drink alcohol, however, should not be encouraged to start consuming it as a heart-protective measure.

People who are overweight or obese are encouraged to lose weight through a combination of reduced calorie intake (maintaining a nutritionally sound diet) and increased exercise. High-protein, high-fat weight loss programs are not recommended for weight reduction.

**EXERCISE** Regular physical exercise reduces the risk for CHD in several ways. It lowers VLDL, LDL, and triglyceride levels, and raises HDL levels. Regular exercise reduces the blood pressure and insulin resistance. Unless contraindicated, all clients are encouraged to participate in at least 30 minutes of moderate intensity physical activity 5 to 6 days each week. To achieve weight loss and prevent weight gain, 60 to 90 minutes of moderate intensity exercise daily is recommended (U.S. Department of Health and Human Services, 2005).

**HYPERTENSION** Although hypertension often cannot be prevented or cured, it can be controlled. Hypertension control (maintaining a blood pressure lower than 140/90 mmHg) is vital to reduce its atherosclerosis-promoting effects and to reduce the workload of the heart. Management strategies include reducing sodium intake, increasing calcium intake, regular exercise, stress management, and medications. Hypertension management is discussed in Chapter 35 .

**DIABETES** Diabetes increases the risk of CHD by accelerating the atherosclerotic process. Weight loss (if appropriate), reduced fat intake, and exercise are particularly important for the diabetic client. Because hyperglycemia apparently also contributes to atherosclerosis, consistent blood glucose management is vital. See Chapter 20  for a detailed discussion about diabetes and blood glucose management.

## Medications

Drug therapy to lower total serum cholesterol and LDL levels and to raise HDL levels now is an integral part of CHD man-

**TABLE 31–4 Dietary Recommendations to Reduce Total Cholesterol, LDL Levels, and CHD Risk**

NUTRIENT	RECOMMENDATION
Calories	Adjusted to attain/maintain desirable body weight
Total fat	25%–35% of total calories
■ Saturated fats	■ <7% of total calories
■ Polyunsaturated fat	■ Up to 10% of total calories
■ Monounsaturated fat	■ Up to 20% of total calories
■ Cholesterol	■ <200 mg/day
Carbohydrate (primarily complex carbohydrates, such as whole grains, fruits, and vegetables)	50%–60% of total calories
Dietary fiber	20–30 g/day
Protein	About 15% of total calories

Source: Compiled from *Adult Treatment Panel III Final Report by the National Cholesterol Education Program, 2002*.

agement. It is used in conjunction with diet and other lifestyle changes, and is based on the client's overall risk for CHD.

Drugs used to treat hyperlipidemia act specifically by lowering LDL levels. The goal of treatment is to achieve an LDL level of <130 mg/dL (NCEP, 2002). Medications to treat hyperlipidemia are not inexpensive; the cost–benefit ra-

tio needs to be considered, because long-term treatment may be required. The four major classes of cholesterol-lowering drugs are statins, bile acid sequestrants, nicotinic acid, and fibrates. The nursing implications and client teaching for these drug classes are outlined in the Medication Administration box below.

## MEDICATION ADMINISTRATION Cholesterol-Lowering Drugs



### STATINS

**Lovastatin (Mevacor)**  
**Pravastatin (Pravachol)**  
**Simvastatin (Zocor)**  
**Fluvastatin (Lescol)**  
**Atorvastatin (Lipitor)**

Statins inhibit the enzyme HMG-CoA reductase in the liver, lowering LDL synthesis and serum levels. The statins are first-line treatment for elevated LDL, used in conjunction with diet and lifestyle changes. Although their side effects are minimal, they may cause increased serum liver enzyme levels and myopathy.

#### Nursing Responsibilities

- Monitor serum cholesterol and liver enzyme levels before and during therapy. Report elevated liver enzyme levels.
- Assess for muscle pain and tenderness. Monitor CPK level if present.
- If taking digoxin concurrently, monitor for and report digoxin toxicity.

#### Health Education for the Client and Family

- Promptly report muscle pain, tenderness, or weakness; skin rash or hives, or changes in skin color; abdominal pain, nausea, or vomiting.
- Do not use these drugs if you are pregnant or plan to become pregnant.
- Inform your doctor if you are taking any other medications concurrently.

### BILE ACID SEQUESTRANTS

**Cholestyramine (Questran)**  
**Colestipol (Colestid)**  
**Colesevelam (Welchol)**

Bile acid sequestrants lower LDL levels by binding bile acids in the intestine, reducing its reabsorption and cholesterol production in the liver. They are used in combination therapy regimens and for women who are considering pregnancy. Their primary disadvantages are inconvenience of administration due to bulk and gastrointestinal side effects such as constipation.

#### Nursing Responsibilities

- Mix cholestyramine and colestipol powders with 4 to 6 oz of water or juice; administer once or twice a day as ordered with meals.
- Store in a tightly closed container.

#### Health Education for the Client and Family

- Promptly report constipation, severe gastric distress with nausea and vomiting, unexplained weight loss, black or bloody stools, or sudden back pain to your doctor.
- Drinking ample amounts of fluid while taking these drugs reduces problems of constipation and bloating.
- Do not omit doses as this may affect the absorption of other drugs you are taking.

### NICOTINIC ACID

**Niacin (Nicobid, Nicolar, Niaspan, others)**

Nicotinic acid in both prescription and nonprescription forms lowers total and LDL cholesterol and triglyceride levels. The crystalline form and Niaspan, a prescription extended release tablet, also raise HDL levels. Because the doses required to achieve significant cholesterol-lowering effects are associated with multiple side effects, nicotinic acid generally is used in combination therapy, particularly with the statin drugs.

#### Nursing Responsibilities

- Give oral preparations with meals and accompanied by a cold beverage to minimize GI effects.
- Administer with caution to clients with active liver disease, peptic ulcer disease, gout, or type 2 diabetes.
- Monitor blood glucose, uric acid levels, and liver function tests during treatment.

#### Health Education for the Client and Family

- Flushing of face, neck, and ears may occur within 2 hours following dose; these effects generally subside as treatment continues. Alcohol use during nicotinic acid therapy may worsen this effect.
- Report weakness or dizziness with changes in posture (lying to sitting; sitting to standing) to your doctor. Change positions slowly to reduce the risk of injury.

### FIBRIC ACID DERIVATIVES

**Gemfibrozil (Lopid)**  
**Fenofibrate (Tricor)**  
**Clofibrate (Atromid-S)**

The fibrates are used to lower serum triglyceride levels; they have only a slight to modest effect on LDL. They affect lipid regulation by blocking triglyceride synthesis. They are used to treat very high triglyceride levels, and may be used in combination with statins.

#### Nursing Responsibilities

- Monitor serum LDL and VLDL levels, electrolytes, glucose, liver enzymes, renal function tests, and CBC during therapy. Report abnormal values.
- Up to 2 months of treatment may be required to achieve a therapeutic effect; rebound, with decreasing benefit, may occur in the second or third month of treatment.

#### Health Education for the Client and Family

- Take with meals if the drug causes gastric distress.
- Promptly report flulike symptoms (fatigue, muscle aching, soreness, or weakness) to your doctor.
- Do not use this drug if you are pregnant or plan to become pregnant. Use reliable birth control measures while taking this drug.
- Contact your doctor before stopping this drug and before taking any over-the-counter preparations.

The statins, including lovastatin (Mevacor), pravastatin (Pravachol), simvastatin (Zocor), and others, are first-line drugs for treating hyperlipidemia. They effectively lower LDL levels and may also increase HDL levels. The statins can cause myopathy; all clients are instructed to report muscle pain and weakness or brown urine. Liver function tests are monitored during therapy, because these drugs may increase liver enzyme levels.

The other cholesterol-lowering drugs, such as the bile acid sequestrants, nicotinic acid, and fibrates, are primarily used when combination therapy is required to effectively lower serum cholesterol levels. They also may be used for selected clients, such as younger adults, women who wish to become pregnant, or to specifically lower triglyceride levels.

Clients at high risk for MI are often started on prophylactic low-dose aspirin therapy. The dose ranges from 80 to 325 mg/day (Tierney et al., 2005). In women, the benefit of low-dose aspirin in reducing the risk for CHD is not clear prior to age 65 (NHLBI, 2005). Aspirin is contraindicated for clients who have a history of aspirin sensitivity, bleeding disorders, or active peptic ulcer disease. Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers also may be prescribed for high-risk clients, including diabetics with other CHD risk factors.

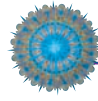
### Complementary Therapies

Diet and exercise programs that emphasize physical conditioning and a low-fat diet rich in antioxidants have been shown to be effective in managing CHD (Box 31–3). Supplements of vitamins C, E, B<sub>6</sub>, B<sub>12</sub>, and folic acid may be beneficial. Other potentially helpful complementary therapies include herbals such as ginkgo biloba, garlic, curcumin, and green tea; and consumption of red wine, foods containing bioflavonoids, and nuts. Emphasize the need for clients to talk to their physician prior to taking any herbal preparations, as interactions with prescribed drugs are common. Behavioral therapies of benefit for clients with CHD include relaxation and stress management, guided imagery, treatment of depression, anger/hostility management, and meditation, tai chi, and yoga.

#### BOX 31–3 Complementary Therapies: Diet for CHD

Two diet programs have been shown to have a beneficial effect on CHD. The *Pritikin diet* is basically vegetarian, high in complex carbohydrates and fiber, low in cholesterol, and extremely low in fat (<10% of daily calories). Egg whites and limited amounts of nonfat dairy or soy products are allowed. The Pritikin program requires 45 minutes of walking daily and recommends multivitamin supplements, including vitamins C and E and folate.

The *Ornish diet* also is vegetarian, although egg whites and a cup of nonfat milk or yogurt per day are allowed. No oil or fat is permitted, even for cooking. Two ounces of alcohol a day are permitted. The Ornish program also calls for stress reduction, emotional social support systems, daily stretching, and walking for 1 hour three times a week.



## NURSING CARE

Nurses are instrumental in educating adults about their risk for coronary heart disease, promoting participation in screening programs to identify that risk, and teaching all clients measures to reduce their risk for CHD.

### Health Promotion

Present information about healthy lifestyle habits to community and religious groups, school children (grades K through 12), and through the print media. In promoting healthy lifestyle habits, nurses can positively affect the incidence, morbidity, and mortality from CHD.

Strongly encourage all clients to avoid smoking in the first place, and to stop all forms of tobacco use. Discuss the adverse effects of smoking and the benefits of quitting. Provide information about dietary recommendations to maintain a healthy weight and optimal cholesterol levels. Discuss the benefits and importance of regular exercise. Finally, encourage clients with cardiovascular risk factors to undergo regular screening for hypertension, diabetes, and abnormal blood lipids.

### Assessment


Nursing assessment for CHD focuses on identifying risk factors.

- **Health history:** Current manifestations such as chest pain or heaviness, shortness of breath, weakness; current diet, exercise patterns, and medications; smoking history and pattern of alcohol intake; history of heart disease, hypertension, or diabetes; family history of CHD or other cardiac problems.
- **Physical examination:** Current weight and its appropriateness for height; body mass index; waist-to-hip ratio; blood pressure; strength and equality of peripheral pulses.

## Nursing Diagnoses and Interventions

### Imbalanced Nutrition:

#### More than Body Requirements

This nursing diagnosis may be appropriate for clients who are obese, have a waist-to-hip ratio greater than 0.8 (female) or 0.9 (male), or whose diet history or serum cholesterol levels indicate a need to reduce fat and cholesterol intake. See Chapters 21  and 22 for more information about assessing obesity.

- Encourage assessment of food intake and eating patterns to help identify areas that can be improved. *Clients often are unaware of their fat and cholesterol intake, particularly when many meals are eaten away from home. Careful assessment increases awareness and allows the client to make conscious changes.*
- Discuss American Heart Association and therapeutic lifestyle change (TLC) dietary recommendations, emphasizing the role of diet in heart disease. Provide guidance regarding specific food choices with healthy alternatives. *Specific diet information and suggestions help the client make better food choices.*
- Refer to a clinical dietitian for diet planning and further teaching. Suggest cookbooks that offer low-fat recipes to encourage healthier eating, and provide American Heart Association and American Cancer Society recipe pamphlets and information

on low-fat eating. *These resources provide tools for the client to use as eating patterns change.*

- Encourage gradual but progressive dietary changes. *Drastic changes in eating patterns may cause frustration and discourage the client from maintaining a healthy diet over the long term.*
- Discourage use of high-fat, low-carbohydrate, or other fad diets for weight loss. *These diets may adversely affect serum cholesterol and triglyceride levels, and often are too drastic to maintain over the long term.*
- Encourage reasonable goals for weight loss (e.g., 1.0 to 1.5 lb per week and a 10% weight loss over 6 months). Provide information about weight loss programs and support groups such as Weight Watchers and Take Off Pounds Sensibly (TOPS). *Gradual but steady weight loss is more likely to be sustained. Recognized programs that emphasize healthy eating provide support and incentive for making lifetime dietary changes.*

### Ineffective Health Maintenance

Clients with risk factors for CHD may be unable to identify or independently manage their risk factors.

- Discuss risk factors for CHD, stressing that changing or managing those factors that can be modified reduces the client's overall risk for the disease. *Clients with significant nonmodifiable risk factors may be discouraged, reducing their ability to eliminate or control modifiable risk factors.*
- Discuss the immediate benefits of smoking cessation. Provide resource materials from the American Heart Association, the American Lung Association, and the American Cancer Society. Refer to a structured smoking cessation program to increase the likelihood of success in quitting. *Long-time smokers may assume that the damage from smoking has already been done, and quitting would not be "worth the price."*
- Help the client identify specific sources of psychosocial and physical support for smoking cessation, dietary, and lifestyle changes. *Support persons, groups, and aids such as nicotine patches help the client achieve success and provide encouragement during difficult times (such as withdrawal symptoms).*
- Discuss the benefits of regular exercise for cardiovascular health and weight loss. Help identify favorite forms of exercise or physical activity. Encourage planning for 30 minutes of continuous aerobic activity (i.e., walking, running, bicycling, swimming) four to five times a week. Encourage identification of an "exercise buddy" to help maintain motivation. *Engaging in preferred activities with a partner maintains motivation and increases the likelihood of maintaining an exercise program. Encourage continuation of the plan, even when days are missed. Exercise is cumulative, so increasing the duration of exercise on subsequent days can "make up" for a lost day.*
- Provide information and teaching about prescribed medications such as cholesterol-lowering drugs. Discuss the relationship between hypertension, diabetes, and CHD. *Teaching is important to promote understanding of and compliance with the prescribed drug regimen.*

## Community-Based Care

Encourage participation in some form of cardiac rehabilitation program. Formal programs provide comprehensive assessment of, interventions for, and teaching of clients with cardiac disease. Monitored exercise and information about risk factors help clients identify ways to lower their risk for CHD.

Because clients themselves are primarily responsible for maintaining the lifestyle changes necessary to reduce the risk of CHD, provide teaching and support as outlined in the previous section. Assist the client to make healthy choices and reinforce positive changes. Emphasize the importance of regular follow-up appointments to monitor progress.

## THE CLIENT WITH ANGINA PECTORIS

**Angina pectoris**, or *angina*, is chest pain resulting from reduced coronary blood flow, which causes a temporary imbalance between myocardial blood supply and demand. The imbalance may be due to coronary heart disease, atherosclerosis, or vessel constriction that impairs myocardial blood supply. Hypermetabolic conditions such as exercise, thyrotoxicosis, stimulant abuse (e.g., cocaine), hyperthyroidism, and emotional stress can increase myocardial oxygen demand, precipitating angina. Anemia, heart failure, ventricular hypertrophy, or pulmonary diseases may affect blood and oxygen supplies as well, causing angina.

### Pathophysiology

The imbalance between myocardial blood supply and demand causes temporary and reversible myocardial ischemia. **Ischemia**, deficient blood flow to tissue, may be caused by partial obstruction of a coronary artery, coronary artery spasm, or a thrombus. Obstruction of a coronary artery deprives cells in the region of the heart normally supplied by that vessel of oxygen and nutrients needed for metabolic processes. Cellular processes are compromised as ATP stores are depleted. Reduced oxygen causes cells to switch from aerobic metabolism to anaerobic metabolism. Anaerobic metabolism causes lactic acid to build up in the cells. It also affects cell membrane permeability, releasing substances such as histamine, kinins, and specific enzymes that stimulate terminal nerve fibers in the cardiac muscle and send pain impulses to the central nervous system. The pain radiates to the upper body because the heart shares the same dermatome as this region. Return of adequate circulation provides the nutrients needed by cells, and clears the waste products. More than 30 minutes of ischemia irreversibly damages myocardial cells (necrosis).

Three types of angina have been identified:

- **Stable angina** is the most common and predictable form of angina. It occurs with a predictable amount of activity or stress, and is a common manifestation of CHD. Stable angina usually occurs when the work of the heart is increased by physical exertion, exposure to cold, or by stress. Stable angina is relieved by rest and nitrates.
- **Prinzmetal's (variant) angina** is atypical angina that occurs unpredictably (unrelated to activity), and often at night. It is

caused by coronary artery spasm with or without an atherosclerotic lesion. The exact mechanism of coronary artery spasm is unknown. It may result from hyperactive sympathetic nervous system responses, altered calcium flow in smooth muscle, or reduced prostaglandins that promote vasodilation.

- *Unstable angina* occurs with increasing frequency, severity, and duration. Pain is unpredictable and occurs with decreasing levels of activity or stress and may occur at rest. Clients with unstable angina are at risk for myocardial infarction. Unstable angina is discussed further in the section on acute coronary syndromes that follows.

*Silent myocardial ischemia*, or asymptomatic ischemia, is thought to be common in people with CHD. Silent ischemia may occur with either activity or with mental stress. Mental stress increases the heart rate and blood pressure, increasing myocardial oxygen demand (McCance & Huether, 2006). Like symptomatic angina, silent myocardial ischemia is associated with an increased chance of myocardial infarction and death (Kasper et al., 2005).

### FAST FACTS

- Stable angina occurs with a predictable amount of activity or stress.
- Unstable angina occurs with increasing frequency and severity; it may occur at times unrelated to activity or stress.
- Prinzmetal's angina is the only type of angina not necessarily related to coronary heart disease and atherosclerosis, developing due to coronary artery spasm.

## Course and Manifestations

The cardinal manifestation of angina is chest pain. The pain typically is precipitated by an identifiable event, such as physical activity, strong emotion, stress, eating a heavy meal, or exposure to cold. The classic sequence of angina is activity–pain, rest–relief. The client may describe the pain as a tight, squeezing, heavy pressure, or constricting sensation. It characteristically begins beneath the sternum and may radiate to the jaw, neck, shoulder, or arm. Less characteristically, the pain may be felt in the jaw, epigastric region, or back. Anginal pain usually occurs in a *crescendo–decrescendo* pattern (increasing to a peak, then gradually decreasing) and typically lasts 2 to 5 minutes. It generally is relieved by rest. Additional manifestations of angina include dyspnea, pallor, tachycardia, and great anxiety and fear.

Women frequently present with atypical symptoms of angina, including indigestion or nausea, vomiting, and upper back pain. The manifestations of angina are summarized in the accompanying box.

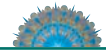
The severity of angina can be graded by the degree to which it limits the client's activities. Class I angina does not occur with ordinary physical activities. It is prompted by strenuous, rapid, or prolonged physical exertion. Class II angina may develop with rapid or prolonged walking or stair climbing, whereas Class III angina significantly limits ordinary physical activities. The client with Class IV angina may have angina at rest, as well as with any physical activity (Kasper et al., 2005).



### MANIFESTATIONS of Angina

- Chest pain: Substernal or precordial (across the chest wall); may radiate to neck, arms, shoulders, or jaw
- Quality: Tight, squeezing, constricting, or heavy sensation; may also be described as burning, aching, choking, dull, or constant
- Associated manifestations: Dyspnea, pallor, tachycardia, anxiety, and fear
- Atypical manifestations: Indigestion, nausea, vomiting, upper back pain
- Precipitating factors: Exercise or activity, strong emotion, stress, cold, heavy meal
- Relieving factors: Rest, position change; nitroglycerin

## INTERDISCIPLINARY CARE



The management of stable angina focuses on maintaining coronary blood flow and cardiac function. Stable angina often can be managed by medical therapy. Measures to restore coronary blood flow are discussed in the section on acute coronary syndrome. As for CHD, risk factor management is a vital component of care for the client with angina (see the preceding section of this chapter).

### Diagnosis

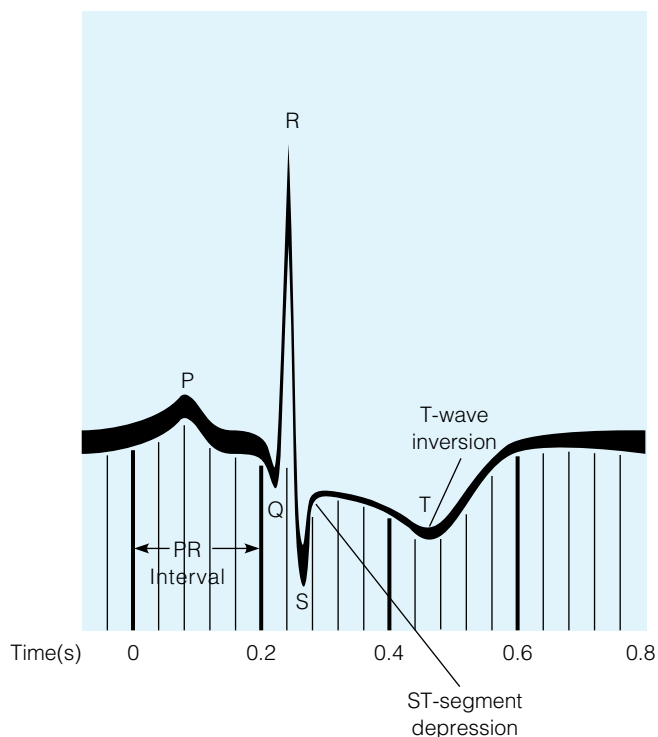
The diagnosis of angina is based on past medical history and family history, a comprehensive description of the chest pain, and physical assessment findings. Laboratory tests may confirm the presence of risk factors, such as an abnormal blood lipid profile and elevated blood glucose. Diagnostic tests provide information about overall cardiac function.

Common diagnostic tests to assess for coronary heart disease and angina include electrocardiography, stress testing, nuclear medicine studies, echocardiography (ultrasound), and coronary angiography.

**ELECTROCARDIOGRAPHY** A resting ECG may be normal, may show nonspecific changes in the ST segment and T wave, or may show evidence of previous myocardial infarction. Characteristic ECG changes are seen during anginal episodes. During periods of ischemia, the ST segment is depressed or downsloping, and the T wave may flatten or invert (Figure 31–1 ■). These changes reverse when ischemia is relieved. For more details about the ECG, its waveforms, and its uses, see Box 30–1 in Chapter 30 ∞.

**STRESS ELECTROCARDIOGRAPHY** Stress electrocardiography (exercise stress test) uses ECGs to monitor the cardiac response to an increased workload during progressive exercise. See the Diagnostic Tests box in the previous chapter for more information about exercise stress tests.

**RADIONUCLIDE TESTING** Radionuclide testing is a safe, non-invasive technique to evaluate myocardial perfusion and left ventricular function. The amount of radioisotope injected is very small; no special radiation precautions are required during or after the scan. Thallium-201 or a technetium-based radiocompound is injected intravenously, and the heart is scanned with a radiation detector. Ischemic or infarcted cells of the



**Figure 31–1** ■ ECG changes during an episode of angina. Note characteristic T-wave inversion and ST-segment depression of myocardial ischemia.

myocardium do not take up the substance normally, appearing as a “cold spot” on the scan. If the ischemia is transient, these spots gradually fill in, indicating the reversibility of the process. With severe ischemia or a myocardial infarction, these areas may remain devoid of radioactivity.

Left ventricular function can also be evaluated. Whereas the ejection fraction, or portion of blood ejected from the left ventricle during systole, normally increases during exercise, it may actually decrease in coronary heart disease and stress-induced ischemia.

Radionuclide testing may be combined with pharmacologic stress testing for clients who are physically unable to exercise or to detect subclinical myocardial ischemia. A vasodilator is injected to induce the same ischemic changes that occur with exercise in the diseased heart. Coronary arteries unaffected by atherosclerosis dilate in response to the drugs, increasing blood flow to already well-perfused tissue. This reduces flow to ischemic muscle, called *myocardial steal syndrome*.

**ECHOCARDIOGRAPHY** *Echocardiography* is a noninvasive test that uses ultrasound to evaluate cardiac structure and function. It may be done at rest, during supine exercise, or immediately following upright exercise to evaluate movement of the myocardial wall and assess for possible ischemia or infarction.

*Transesophageal echocardiography (TEE)* uses ultrasound to identify abnormal blood flow patterns as well as cardiac structures. In TEE, the probe is on the tip of an endoscope inserted into the esophagus, positioning it close to the posterior heart (especially the left atrium and the aorta). It avoids interference by breasts, ribs, or lungs. See the Diagnostic Tests box

in Chapter 30 ∞ for more information about and the nursing care for clients undergoing these tests.

**CORONARY ANGIOGRAPHY** *Coronary angiography* is the gold standard for evaluating the coronary arteries. Guided by fluoroscopy, a catheter introduced into the femoral or brachial artery is threaded into the coronary artery. Dye is injected into each coronary opening, allowing visualization of the main coronary branches and any abnormalities, such as stenosis or obstruction. Narrowing of the vessel lumen by more than 50% is considered significant; most lesions that cause symptoms involve more than 70% narrowing. Vessel obstructions are noted on a coronary artery “map” that provides a guide for tracking disease progression and for elective treatment with angioplasty or cardiac surgery. During angiogram, the drug ergonovine maleate may be injected to induce coronary artery spasm and diagnose Prinzmetal’s angina. See the Diagnostic Tests box in Chapter 30 for nursing care of clients undergoing coronary angiography.

### Medications

Drugs may be used for both acute and long-term relief of angina. The goal of drug treatment is to reduce oxygen demand and increase oxygen supply to the myocardium. Three main classes of drugs are used to treat angina: nitrates, beta blockers, and calcium channel blockers.

**NITRATES** Nitrates, including nitroglycerin and longer-acting nitrate preparations, are used to treat acute anginal attacks and prevent angina.

Sublingual nitroglycerin is the drug of choice to treat acute angina. It acts within 1 to 2 minutes, decreasing myocardial work and oxygen demand through venous and arterial dilation, which in turn reduce preload and afterload. It may also improve myocardial oxygen supply by dilating collateral blood vessels and reducing stenosis. Rapid-acting nitroglycerin is also available as a buccal spray in a metered system. For some clients, this may be easier to handle than small nitroglycerin tablets.

### PRACTICE ALERT


- Sublingual nitroglycerin tablets and nitroglycerin spray are the only medications appropriate to treat an acute anginal attack.

Longer-acting nitroglycerin preparations (oral tablets, ointment, or transdermal patches) are used to prevent attacks of angina, not to treat an acute attack. The primary problem with long-term nitrate use is the development of *tolerance*, a decreasing effect from the same dose of medication. Tolerance can be limited by a dosing schedule that allows a nitrate-free period of at least 8 to 10 hours daily. This is usually scheduled at night, when angina is less likely to occur.

Headache is a common side effect of nitrates, and may limit their usefulness. Nausea, dizziness, and hypotension are also common effects of therapy.

**BETA BLOCKERS** Beta blockers, including propranolol, metoprolol, nadolol, and atenolol, are considered first-line drugs to treat stable angina. They block the cardiac-stimulating effects of norepinephrine and epinephrine, preventing anginal attacks by

reducing heart rate, myocardial contractility, and blood pressure, thus reducing myocardial oxygen demand. Beta blockers may be used alone or with other medications to prevent angina.

Beta blockers are contraindicated for clients with asthma or severe COPD (see Chapter 39 ) because they may cause severe bronchospasm. They are not used in clients with significant bradycardia, or AV conduction blocks, and are used cautiously in heart failure. Beta blockers are not used to treat Prinzmetal's angina because they may make it worse.

**CALCIUM CHANNEL BLOCKERS** Calcium channel blockers reduce myocardial oxygen demand and increase myocardial blood and oxygen supply. These drugs, which include verapamil, diltiazem, and nifedipine, lower blood pressure, reduce myocardial contractility, and, in some cases, lower the heart rate, decreasing myocardial oxygen demand. They are also potent coronary vasodilators, effectively increasing oxygen supply. Like beta blockers, calcium channel blockers act too slowly to effectively treat an acute attack of angina; they are used for long-term prophylaxis. Because they may actually increase ischemia and mortality in clients with heart failure or left ventricular dysfunction, these drugs are not usually prescribed in the initial treatment of angina. They are used cautiously in clients with dysrhythmias, heart failure, or hypotension.

The nursing implications of antianginal medications are summarized in the Medication Administration box on the next page.

**ASPIRIN** The client with angina, particularly unstable angina, is at risk for myocardial infarction because of significant narrowing of the coronary arteries. Low-dose aspirin (80 to 325 mg/day) is often prescribed to reduce the risk of platelet aggregation and thrombus formation.



## NURSING CARE

The focus of nursing care for clients with angina is similar to the interdisciplinary care focus: to reduce myocardial oxygen demand and improve the oxygen supply. Angina usually is treated in community settings; the primary nursing focus is education.

### Health Promotion

In addition to health promotion measures identified for CHD, emphasize the importance of active CHD risk factor management to slow progression of the disease. Encourage clients to stop smoking. Discuss the use of cholesterol-lowering drug therapy with clients who have hypercholesterolemia. Encourage regular aerobic exercise and a diet based on American Heart Association or National Cholesterol Education Program guidelines.

### Assessment

Focused assessment data for the client with angina includes the following:

- **Health history:** Chest pain, including type, intensity, duration, frequency, aggravating factors, and relief measures; associated symptoms; history of other cardiovascular disorders, peripheral vascular disease, or stroke; current medications

and treatment; usual diet, exercise, and alcohol intake patterns; smoking history; use of other recreational drugs.

- **Physical assessment:** Vital signs and heart sounds; strength and equality of peripheral pulses; skin color and temperature (central and peripheral); physical appearance during pain episode (e.g., shortness of breath, apparent anxiety, color, diaphoresis).

## Nursing Diagnoses and Interventions

High-priority nursing problems for clients with angina include ineffective cardiac tissue perfusion and management of the prescribed therapeutic regimen.

### Ineffective Tissue Perfusion: Cardiac

The pain of angina results from impaired blood flow and oxygen supply to the myocardium. Nursing interventions can both prevent ischemia and shorten the duration of pain.

- Keep prescribed nitroglycerin tablets at the client's side so one can be taken at the onset of pain. *Anginal pain indicates myocardial ischemia. Nitroglycerin reduces cardiac work and may improve myocardial blood flow, relieving ischemia and pain.*
- Start oxygen at 4 to 6 L/min per nasal cannula or as prescribed. *Supplemental oxygen reduces myocardial hypoxia.*
- Space activities to allow rest between them. *Activity increases cardiac work and may precipitate angina. Spacing of activities allows the heart to recover.*
- Teach about prescribed medications to maintain myocardial perfusion and reduce cardiac work. Emphasize that long-acting nitrates, beta blockers, and calcium channel blockers are used to *prevent* anginal attacks, not to *treat* an acute attack. *It is important for the client to understand the purpose and use of prescribed drugs to maintain optimal myocardial perfusion.*
- Instruct to take sublingual nitroglycerin before engaging in activities that precipitate angina (e.g., climbing stairs, sexual intercourse). *This prophylactic dose of nitroglycerin helps maintain cardiac perfusion when increased work is anticipated, preventing ischemia and chest pain.*
- Encourage to implement and maintain a progressive exercise program under the supervision of the primary care provider or a cardiac rehabilitation professional. *Exercise slows the atherosclerotic process and helps develop collateral circulation to the heart muscle.*
- Refer to a smoking cessation program as indicated. *Nicotine causes vasoconstriction and increases the heart rate, decreasing myocardial perfusion and increasing cardiac workload.*

### Risk for Ineffective Therapeutic Regimen Management

Denial may be strong in the client with angina pectoris. Because many people think of the heart as the locus of life itself, problems such as angina remind people of their mortality, an uncomfortable fact. Denial may lead to “forgetting” to take prescribed medications or to attempting activities that will precipitate angina. Some clients, by contrast, may become “cardiac cripples,” afraid



## MEDICATION ADMINISTRATION

## Antianginal Medications

**ORGANIC NITRATES**

**Nitroglycerin (Nitropaste, Nitro-Dur, Nitro-Bid, Nitrol, Transderm-Nitro, Nitrogard, Nitrodisc, Tridil)**

**Isosorbide dinitrate (Isordil)**

**Isosorbide mononitrate (ISMO)**

**Amyl nitrite**

Nitrates dilate both arterial and venous vessels, depending on the dose. Coronary artery vasodilation increases blood flow and myocardial oxygen supply. Venous dilation allows peripheral blood pooling, reducing venous return, preload, and cardiac work. Arterial dilation reduces vascular resistance and afterload, also reducing cardiac work. Sublingual nitroglycerin (NTG) tablets are used to treat and prevent acute anginal attacks (when taken prophylactically before activity). Nitrates are administered sublingually, by buccal spray, or intravenously for immediate effect; or orally or topically for sustained effect.

**Nursing Responsibilities**

- Dilute intravenous nitroglycerin before infusing; use only glass bottles for the mixture. Nitroglycerin adheres to PVC bags and tubing, affecting the amount of drug that is delivered. Use non-PVC infusion tubing.
- Wear gloves when applying nitroglycerin paste or ointment to prevent absorbing the drug through the skin. Measure dose carefully and spread evenly in a 2- by 3-inch area.
- Remove nitroglycerin patches or ointment at night to help prevent tolerance.

**Health Education for the Client and Family**

- Use only the sublingual, buccal, and spray forms of nitrates to treat acute angina.
- If the first nitrate dose does not relieve angina within 5 minutes, take a second dose. After 5 more minutes, you may take a third dose if needed. If the pain is unrelieved or lasts for 20 minutes or longer, seek medical assistance immediately.
- Carry a supply of nitroglycerin tablets with you. Dissolve sublingual nitroglycerin tablets under the tongue or between the upper lip and gum. Do not eat, drink, or smoke until the tablet is completely dissolved.
- Keep sublingual tablets in their original amber glass bottle to protect them from heat, light, and moisture. Replace your supply every 6 months.
- You may experience a burning or tingling sensation under the tongue and develop a transient headache when you take the drug. These are expected; the headache will diminish over time.
- Use caution when standing from a sitting position; nitroglycerin may make you light-headed.
- Rotate ointment or transdermal patch application sites. Apply to a hairless area; spread ointment evenly without rubbing or massaging. Remove the patch or residual ointment at bedtime daily. Apply a fresh dose in the morning.
- If you are using a long-acting nitrate, keep a supply of immediate-acting nitrates to treat acute angina.

**BETA BLOCKERS**

**Atenolol (Tenormin)**

**Metoprolol (Lopressor)**

**Propranolol (Inderal)**

**Nadolol (Corgard)**

Beta blockers decrease cardiac workload by blocking beta receptors on the heart muscle, decreasing heart rate, contractility, myocardial oxygen consumption, and blood pressure. Beta blockers also reduce *reflex tachycardia* (an increased heart rate in response to stimuli such as increased SNS activity or vasodilation), which may develop with other antianginal drugs. Beta blockers are frequently prescribed as antianginal and antihypertensive agents.

**Nursing Responsibilities**

- Document heart rate and blood pressure before administering the medication. Withhold drug if the heart rate is below 50 bpm or the blood pressure is below prescribed limits. Notify the physician.
- Assess for and report possible contraindications to therapy, including heart failure, bradycardia, AV block, asthma, or COPD.
- Concurrent use of beta blockers and calcium channel blockers increases the risk for heart failure; notify the physician if these drugs are prescribed together.
- Do not abruptly discontinue these drugs after long-term therapy, as this can increase heart rate, contractility, and blood pressure, and cause fatal dysrhythmia, myocardial infarction, or stroke.

**Health Education for the Client and Family**

- Beta blockers help prevent angina but will not relieve an acute attack. Keep a supply of fast-acting nitrates on hand for acute anginal attacks.
- Do not suddenly stop taking this medication. Discuss discontinuing this medication with your doctor.
- Take your pulse daily. Do not take the drug, and contact your doctor, if your heart rate is below 50 bpm. Check your blood pressure frequently.
- Report a slow or irregular pulse, swelling or weight gain, or difficulty breathing to your doctor.

**CALCIUM CHANNEL BLOCKERS**

**Nifedipine (Adalat, Procardia)**

**Diltiazem (Cardizem)**

**Verapamil (Isoptin, Calan)**

**Bepidil (Vascor)**

**Felodipine (Plendil)**

**Isradipine (DynaCirc)**

**Nicardipine (Cardene)**

**Nimodipine (Nimotop)**

Calcium channel blockers are used to control angina, hypertension, and dysrhythmias. By blocking the entry of calcium into cells, these drugs reduce contractility, slow the heart rate and conduction, and cause vasodilation. Calcium channel blockers increase myocardial oxygen supply by dilating the coronary arteries; they decrease the workload of the heart by lowering vascular resistance and oxygen demand. Calcium channel blockers are often prescribed for clients with coronary artery spasm (Prinzmetal's angina).

**Nursing Responsibilities**

- Do not mix verapamil in any solution containing sodium bicarbonate. Administer IV push verapamil over 2 to 3 minutes.
- Document blood pressure and heart rate before administering the drug. Withhold the drug if the heart rate is below 50 bpm. Notify the physician.

(continued)



## MEDICATION ADMINISTRATION

### Antianginal Medications (continued)

- The nifedipine capsule may be punctured and administered by extracting the liquid with a syringe and squirting the dose under the client's tongue (discard the needle first!).
- Use caution when giving a calcium channel blocker with other cardiac depressants, such as beta blockers. Concomitant administration with nitrates may cause excessive vasodilation.
- Manifestations of toxicity include nausea, generalized weakness, signs of decreased cardiac output, hypotension, bradycardia, and AV block. Report these findings immediately. Maintain intravenous access, and slowly administer intravenous calcium

chloride. Do not infuse large volumes of fluid to treat hypotension as heart failure may result.

#### Health Education for the Client and Family

- Take your pulse before taking the drug. Do not take the drug and notify physician if your heart rate drops below 50 bpm.
- Keep a fresh supply of immediate-acting nitrate available to treat acute anginal attacks. Calcium channel blockers will not work fast enough to relieve an acute attack.

to engage in activities because of anticipated chest pain. Their inactivity may actually hasten the atherosclerotic process and inhibit collateral circulation development, worsening angina.

- Assess knowledge and understanding of angina. *Assessment allows tailoring of teaching and interventions to the needs of the client.*
- Teach about angina and atherosclerosis as needed, building on current knowledge base. *This can help the client understand that angina is a manageable disease and that pain can usually be controlled and the disease progress slowed.*
- Provide written and verbal instructions about prescribed medications and their use. *Written instructions reinforce teaching and are available to the client for future reference.*
- Stress the importance of taking chest pains seriously while maintaining a positive attitude. *Although it is vital to recognize the significance of chest pain and deal with it appropriately, it is also important to maintain a positive outlook.*
- Refer to a cardiac rehabilitation program or other organized activities and support groups for clients with coronary heart disease. *Programs such as these help the client develop risk factor management strategies, maintain a program of supervised activity, and gain coping skills.*

## Community-Based Care

Many clients with stable angina manage their pain effectively, continuing to live active and productive lives. To promote effective management of this disorder, include the following topics in teaching for home care:

- Coronary heart disease and the processes that cause chest pain, including the relationship between the pain and reduced blood flow to the heart muscle
- Use and effects (desired and adverse) of prescribed medications; importance of not discontinuing medications abruptly
- Nitroglycerin use for acute angina: always carry several tablets (not the entire supply); prophylactic use before activities that often cause chest pain; take tablet at first indication of pain rather than waiting to see if the pain develops; seek immediate medical assistance if three nitroglycerin tablets over 15 to 20 minutes do not relieve the pain
- The importance of calling 911 or going to the emergency department immediately for unrelieved chest pain

- Appropriate storage of nitroglycerin: This unstable compound needs to be stored in a cool, dry, dark place; no more than a 6-month supply should be kept on hand.

For the client who has undergone cardiac surgery, also include the following:

- Respiratory care, activity, and pain management
- The importance of actively participating in rehabilitation
- Manifestations of infection or other potential complications and their management.

## THE CLIENT WITH ACUTE CORONARY SYNDROME

**Acute coronary syndrome (ACS)** is a condition of unstable cardiac ischemia. ACS includes unstable angina and acute myocardial ischemia with or without significant injury of myocardial tissue. Although the term ACS may, in some cases, be applied to acute myocardial infarction (myocardial tissue death), myocardial infarction is discussed separately in the next section of this chapter. An estimated 1.4 million Americans are admitted to the hospital annually with ACS (Kasper et al., 2005).

### FAST FACTS

- Acute coronary syndrome (severe cardiac ischemia), a common cause of hospital admission, includes unstable angina and acute myocardial infarction.
- Unstable angina is characterized by injury to myocardial cells; with prompt restoration of blood flow, muscle tissue recovers.
- Myocardial infarction is characterized by necrosis and death of myocardial cells; scar tissue forms and functional muscle is lost.
- ACS is the most common identified cause of sudden cardiac death (American Heart Association [AHA], 2005a).

## Pathophysiology

ACS is a dynamic state in which coronary blood flow is acutely reduced, but not fully occluded. Myocardial cells are injured by the acute ischemia that results. Most people affected by ACS have significant stenosis of one or more coronary arteries.

ACS is precipitated by one or more of the following processes: (1) rupture or erosion of atherosclerotic plaque with formation of a blood clot that does not fully occlude the vessel; (2) coronary ar-

tery spasm (e.g., Prinzmetal's angina); (3) progressive vessel obstruction by atherosclerotic plaque or restenosis following a percutaneous revascularization (PCR) procedure; (4) inflammation of a coronary artery; or (5) increased myocardial oxygen demand and/or decreased supply (e.g., acute blood loss or anemia) (Braunwald et al., 2002). Of these, ruptured or eroded plaque is the predominant pathophysiology underlying ACS (AHA, 2005a). Plaque rupture often is triggered by hemodynamic factors such as increased heart rate, blood flow, and blood pressure in response to a surge of sympathetic nervous system (SNS) activity. Increased SNS activity also is thought to contribute to the higher incidence of plaque rupture within the first hour of arising from bed in the morning (Porth, 2005).

When atherosclerotic plaque ruptures or erodes, the exposed lipid core of the plaque stimulates platelet aggregation and the extrinsic clotting pathway. Thrombin is generated and fibrin is deposited, forming a clot that severely impairs or obstructs blood flow to tissue distal to the area of plaque rupture. As a result, these cells become ischemic.

Injured myocardial cells contract less effectively, potentially reducing cardiac output if a large area of myocardium is affected. Lactic acid released from ischemic cells stimulates pain receptors, causing chest pain. Ischemia and injury affect electrical impulse conduction, producing inversion of the T wave and possibly elevation of the ST segment on the ECG.

## Manifestations

The cardinal manifestation of ACS is chest pain, usually substernal or epigastric. The pain often radiates to the neck, left shoulder, and/or left arm. The pain may occur at rest and typically lasts longer than 10 to 20 minutes. In ACS, the chest pain

is more severe and prolonged than that previously experienced by the client. It may be a new onset of pain, or may represent a pattern of increasing frequency and severity of anginal pain. Dyspnea, diaphoresis, pallor, and cool skin may be present. Tachycardia and hypotension may occur. The client may be nauseated or feel light-headed. Table 31–5 compares the features of stable angina, ACS, and acute myocardial infarction.

## INTERDISCIPLINARY CARE



The client with ACS generally presents at the emergency department or physician's office with complaints of severe chest pain. The pain may be unrelieved by nitroglycerin or may be more severe and of longer duration than previous anginal episodes. The ECG is used in conjunction with blood levels of cardiac markers to differentiate between unstable angina and acute myocardial infarction. Clients with unstable angina generally are admitted to the acute care unit on bed rest with cardiac monitoring for 12 to 24 hours. Coronary revascularization procedures may be performed within 48 hours if significant CHD is identified.

### Diagnosis

The ECG and serum cardiac markers are the primary tests used to establish the diagnosis of ACS. Serum cardiac markers, proteins released from injured and necrotic heart muscle, can be measured. (See the following section on acute myocardial infarction and Table 31–6 for more information about serum cardiac markers.)

- Cardiac muscle troponins, *cardiac-specific troponin T* ( $cT_nT$ ) and *cardiac-specific troponin I* ( $cT_nI$ ), are sensitive indicators of myocardial damage. Troponins may be elevated in

**TABLE 31–5 Comparing Stable Angina, Acute Coronary Syndrome, and Acute Myocardial Infarction**

	STABLE ANGINA	ACUTE CORONARY SYNDROME	ACUTE MYOCARDIAL INFARCTION
<b>Pathophysiology</b>	Myocardial ischemia occurs with increased workload (e.g., during exercise) due to stable atherosclerotic plaque narrowing the coronary arteries.	Coronary artery spasm or partial occlusion results from unstable plaque and thrombus formation with increasing myocardial ischemia.	Obstruction of a coronary artery by a thrombus blocks blood supply to a portion of myocardium, resulting in necrosis.
<b>Chest Pain</b>	Stable and predictable, occurring with exertion or emotion Crescendo–decrescendo pattern May radiate to neck, shoulder, arms Usually lasts 2 to 5 minutes, relieved by rest	Occurs at rest; increasing frequency and severity Lasts 10 minutes or longer Radiates to neck, left shoulder, and arm	Begins abruptly, unrelated to rest or exercise Severe, “crushing” Unrelieved by rest or nitroglycerin Radiates to arms, neck, jaw
<b>Other Manifestations</b>	Indigestion, nausea Possible shortness of breath Anxiety	Epigastric pain Dyspnea Tachycardia, hypotension Cool, pale skin	Epigastric pain, nausea Dyspnea Pallor, diaphoresis Tachycardia or bradycardia, hyper- or hypotension
<b>Diagnosis</b>	ECG: T-wave inversion during anginal episodes Cardiac markers: within normal range	ECG: ST-segment depression, T-wave inversion Cardiac markers: within normal range or transient elevation	ECG: ST-segment elevation, possible Q wave Cardiac markers: elevated

ACS or may be within normal limits if chest pain is due to unstable angina.

- Creatine kinase (CK) and CK-MB (specific to myocardial muscle) levels are likely to be within normal limits or demonstrate transient elevation, returning to normal levels within 12 to 24 hours.

The ECG, particularly when done during the acute episode of chest pain, is a valuable diagnostic tool for ACS. ST-segment changes (elevation or depression) during chest pain that resolve when the pain abates usually indicate acute myocardial ischemia and severe underlying CHD.

## Medications

Medications include drugs to reduce myocardial ischemia and those to reduce the risk for blood clotting. Fibrinolytic drugs (that is, drugs that break down the fibrin in blood clots) may be given prior to or on admission to the emergency department. These drugs restore blood flow to ischemic cardiac muscle and can prevent permanent damage. See the section on myocardial infarction for more information about fibrinolytic drugs and their nursing implications.

Nitrates and beta blockers are used to restore blood flow to the ischemic myocardium and reduce the workload of the heart. Nitroglycerin is given by sublingual tablet or buccal spray. If

chest pain is unrelieved after three doses 5 minutes apart, an intravenous nitroglycerin infusion is initiated. The infusion may be continued until the chest pain is relieved or for 12 to 24 hours. Topical or oral nitrates are then initiated. Beta-adrenergic blockers are initially given intravenously, followed by oral beta-blockers. See the Medication Administration box on page 973 for the nursing implications of these drugs.

Aspirin, other antiplatelet drugs, and heparin are given to inhibit blood clotting and reduce the risk of thrombus formation. Aspirin and clopidogrel (Plavix) are given to clients with ACS who do not have an excessive bleeding risk. Aspirin and clopidogrel suppress platelet aggregation, interrupting the process of forming a stable blood clot. Both increase the risk of serious hemorrhage; for most clients, however, the benefit outweighs the risk. Intravenous antiplatelet drugs such as abciximab (ReoPro), eptifibatide (Integrilin), or tirofiban (Aggrastat) may be used when an invasive coronary revascularization procedure is anticipated in the immediate or near future. Nursing implications for the antiplatelet drugs are outlined in the Medication Administration box below.

## Revascularization Procedures

Several procedures may be used to restore blood flow and oxygen to ischemic tissue. Nonsurgical techniques include translu-

### MEDICATION ADMINISTRATION Antiplatelet Drugs



#### ORAL ANTIPLATELET DRUGS

##### Aspirin

##### Clopidogrel (Plavix)

Antiplatelet drugs suppress platelet aggregation in arteries, preventing the development of an arterial thrombus. Aspirin and clopidogrel block different platelet activation pathways to inhibit platelet aggregation and clot formation. The dose of aspirin given to achieve antiplatelet effects is low, typically 80 to 325 mg/day.

#### Nursing Responsibilities

- Inquire about a history of intracranial hemorrhage, upper gastrointestinal bleeding, peptic ulcer disease, or known bleeding tendency.
- Observe for and report increased bruising, petechiae, purpura, apparent or occult bleeding (e.g., melena, hematemesis).
- Do not administer concurrently with warfarin (Coumadin).

#### Health Education for the Client and Family

- Take as directed. Take aspirin with food or milk; clopidogrel may be taken at any time of day.
- Do not use NSAIDs or other over-the-counter drugs that may contain aspirin or an NSAID unless prescribed by your physician.
- Check with your physician before taking any herbal remedies such as evening primrose oil, feverfew, garlic, ginkgo biloba, or grapeseed extract while taking these medications.
- Report unusual bruising or excessive bleeding.
- Inform all care providers (including dental professionals) of use of these drugs.

#### INTRAVENOUS ANTIPLATELET DRUGS

##### Abciximab (ReoPro)

##### Eptifibatide (Integrilin)

##### Tirofiban (Aggrastat)

The intravenously administered antiplatelet drugs, abciximab, eptifibatide, and tirofiban, block the final common pathway of platelet activation, and thus are more effective. However, the risk of bleeding is greater than with the orally administered antiplatelet drugs.

#### Nursing Responsibilities

- Determine history of bleeding disorders, intracranial hemorrhage, recent trauma or surgery.
- Inquire about recent use of oral antiplatelet or anticoagulant drugs.
- Monitor CBC including hemoglobin, hematocrit, and platelet count; clotting studies, including PT, INR, PTT; vital signs; and ECG during therapy.
- Maintain a separate intravenous line for blood draws and administration of other drugs during infusion.
- Closely observe for and immediately report anaphylaxis or bleeding uncontrolled by pressure. Keep resuscitation equipment readily available.
- Maintain bed rest during infusion.

#### Health Education for the Client and Family

- This drug is given to reduce the risk of clotting and myocardial infarction. It helps maintain blood flow through the affected vessel following angioplasty and stent placement.
- Immediately report any chest tightness, difficulty breathing, shortness of breath, or itching that develops during the infusion.
- Your risk of bleeding should return to normal within about 2 days following the infusion.
- Immediately report any unusual bruising or bleeding to your doctor.

minimal coronary angioplasty, laser angioplasty, coronary atherectomy, and intracoronary stents. Coronary artery bypass grafting (CABG) is a surgical procedure that may be used.

**PERCUTANEOUS CORONARY REVASCULARIZATION** *Percutaneous coronary revascularization (PCR)* procedures are used to restore blood flow to the ischemic myocardium in clients with CHD. Approximately 600,000 PCR procedures are done annually in the United States. PCR is used to treat clients with:

- Moderately severe, chronic stable angina unrelieved by medical therapy
  - Unstable angina
  - Acute myocardial infarction
  - Significant stenosis of the left anterior descending coronary artery
  - Stenosis of a CABG (Kasper et al., 2005; Tierney et al., 2005).
- PCR procedures are similar to the procedure used for coronary angiography. A catheter introduced into the arterial circulation is guided into the opening of the narrowed coronary artery. A flexible guidewire is inserted through the catheter lumen into the affected vessel. The guidewire is then used to thread an angioplasty balloon, arterial stent, or other therapeutic device into the narrowed segment of the artery. The procedure is performed in the cardiac catheterization laboratory using local anesthesia. The hospital stay is short (1 to 2 days), minimizing costs.

In a *percutaneous transluminal coronary angioplasty (PTCA)*, a balloon-tipped catheter is threaded over the guidewire, with the balloon positioned across the area of narrowing (Figure 31–2 ■). The balloon is inflated in a step-by-step fashion for about 30 seconds to 2 minutes to compress the plaque against the arterial wall, with a goal of reducing the vessel obstruction to less than 50% of the arterial lumen. PTCA typically is accompanied by placement of a stent. *Intracoronary stents* are metallic scaffolds used to maintain an

open arterial lumen. Stents reduce the rate of restenosis following angioplasty by about one-third, and are now used in the majority of all PCR procedures (Kasper et al., 2005). The stent is placed over a balloon catheter, guided into position, and expanded as the balloon is inflated. It then remains in the artery as a prop after the balloon is removed. Endothelial cells will completely line the inner wall of the stent to produce a smooth inner lining. Antiplatelet medications (aspirin and ticlopidine) are given following stent insertion to reduce the risk of thrombus formation at the site.

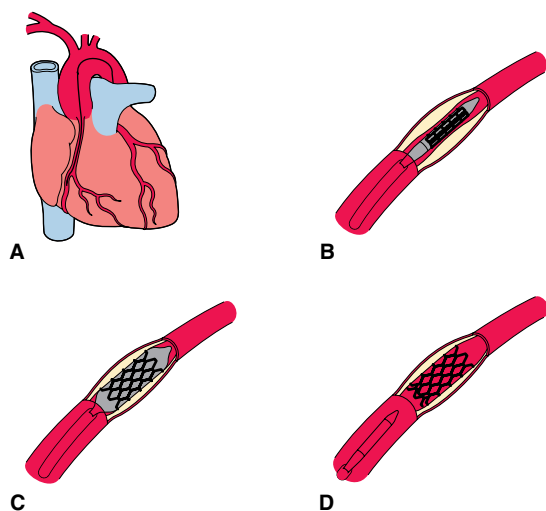
In contrast to stent procedures, which enlarge the artery by displacing plaque, *atherectomy* procedures remove plaque from the identified lesion. The directional atherectomy catheter shaves the plaque off vessel walls using a rotary cutting head, retaining the fragments in its housing and removing them from the vessel. Rotational atherectomy catheters pulverize plaque into particles small enough to pass through the coronary microcirculation. Laser atherectomy devices use laser energy to remove plaque.

Complications following PCR procedures include hematoma at the catheter insertion site, pseudoaneurysm, embolism, hypersensitivity to contrast dye, dysrhythmias, bleeding, vessel perforation, and restenosis, or reocclusion of the treated vessel.

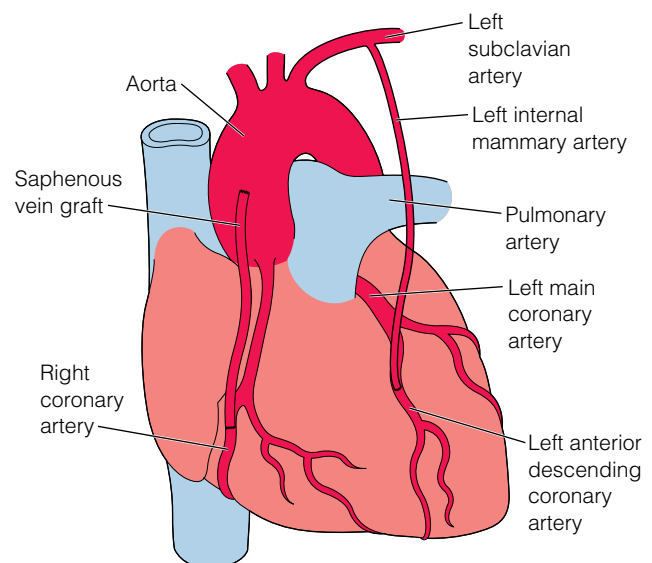
Nursing care of the client undergoing PCR is outlined in the box on the next page.

**CORONARY ARTERY BYPASS GRAFTING** Surgery for coronary heart disease involves using a section of a vein or an artery to create a connection (or bypass) between the aorta and the coronary artery beyond the obstruction (Figure 31–3 ■). This then allows blood to perfuse the ischemic portion of the heart. The internal mammary artery in the chest and the saphenous vein from the leg are the vessels most commonly used for CABG.

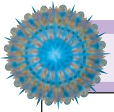
Bypass grafts are safe and effective. Angina is totally relieved or significantly reduced in 90% of clients who undergo



**Figure 31–2** ■ Percutaneous coronary revascularization. *A*, The balloon catheter with the stent is threaded into the affected coronary artery. *B*, *C*, The stent is positioned across the blockage and expanded. *D*, The balloon is deflated and removed, leaving the stent in place.



**Figure 31–3** ■ Coronary artery bypass grafting using the internal mammary artery and a saphenous vein graft.



## NURSING CARE OF THE CLIENT HAVING PCR

### BEFORE THE PROCEDURE

- Assess knowledge of the procedure and expectations of treatment. *This allows information to be tailored to the client's needs and provides an opportunity to clarify misconceptions.*
- Describe the cardiac catheterization laboratory and the planned PCR procedure, including:
  - Preoperative preparation (see Chapter 4 ∞)
  - Planned anesthesia or sedation to be used.
  - Drugs that may be given during the procedure, such as anticoagulants to reduce the risk of thrombus formation, and intravenous nitroglycerin and a calcium channel blocker to dilate coronary arteries and prevent anginal pain.
- Discuss possible sensations during the procedure, including flushing or warmth and a metallic taste in the mouth as the contrast dye is injected, and a feeling of pressure or chest pain during balloon inflation. *Advanced preparation for expected sensations reduces anxiety and improves outcomes.*
- Perform a comprehensive assessment, including hydration status (skin and mucous membrane moisture, turgor) and peripheral circulation (color, warmth, sensation, pulses, and capillary refill).

### AFTER THE PROCEDURE

- Complete a head-to-toe assessment. Note any complaints of chest pain, or evidence of decreased cardiac output or myocardial infarction. *Assessment provides a baseline for subsequent assessments and allows early identification of possible complications.*
- Monitor vital signs and cardiac rhythm continuously. Treat dysrhythmias as ordered. Obtain a 12-lead ECG if signs of ischemia develop, and notify physician. *Vital signs reflect cardiac output. Dysrhythmias may develop with reperfusion of the ischemic myocardium. ECG changes may indicate infarction or restenosis of the affected vessel.*
- Maintain intravenous nitroglycerin infusion. Administer anticoagulant and antiplatelet medications, nitrates, and calcium

channel blockers as ordered. *These drugs decrease oxygen demand and increase oxygen supply by dilating the coronary arteries and systemic vasculature. They also reduce the risk of thrombus formation.*

- Monitor for and treat or report chest pain as indicated. *Chest pain may indicate ischemia and possible myocardial infarction.*
- Maintain bed rest as ordered with the head of the bed at 30 degrees or less. Prevent flexion of the leg on the affected side. Following sheath removal, follow protocol for pressure dressing or device or sandbag placement. *A large puncture wound occurs at the insertion site. Immobilization allows the wound to seal; a pressure dressing helps prevent bleeding.*
- Monitor distal pulses, color, movement, sensation, and temperature of the affected leg, and insertion site every 15 minutes for the first hour, every 30 minutes for the next hour, every hour for the next 8 hours, then every 4 hours. *A clot may form at the site, reducing perfusion of the affected leg. The site and dressing are monitored for excessive bleeding, hematoma formation, or pseudoaneurysm. Pseudoaneurysm occurs as a result of inadequate hemostasis after catheter removal.*
- Monitor intake and output, serum electrolytes, blood urea nitrogen (BUN), creatinine, complete blood count (CBC), partial thromboplastin time (PTT), and cardiac enzymes. Report abnormal results to the physician. *Contrast dye causes osmotic diuresis and may cause renal damage or a hypersensitivity reaction. Electrolyte imbalances increase the risk of dysrhythmias. Cardiac enzymes are monitored for indications of possible myocardial damage during the procedure. The PTT monitors the effectiveness of heparin therapy.*
- Monitor for bradycardia, light-headedness, hypotension, diaphoresis, and loss of consciousness during sheath removal. Keep atropine at bedside during sheath removal. *Bradycardia and signs of decreased cardiac output may occur during sheath removal because of a vasovagal reaction. Atropine decreases vagal tone and increases heart rate.*

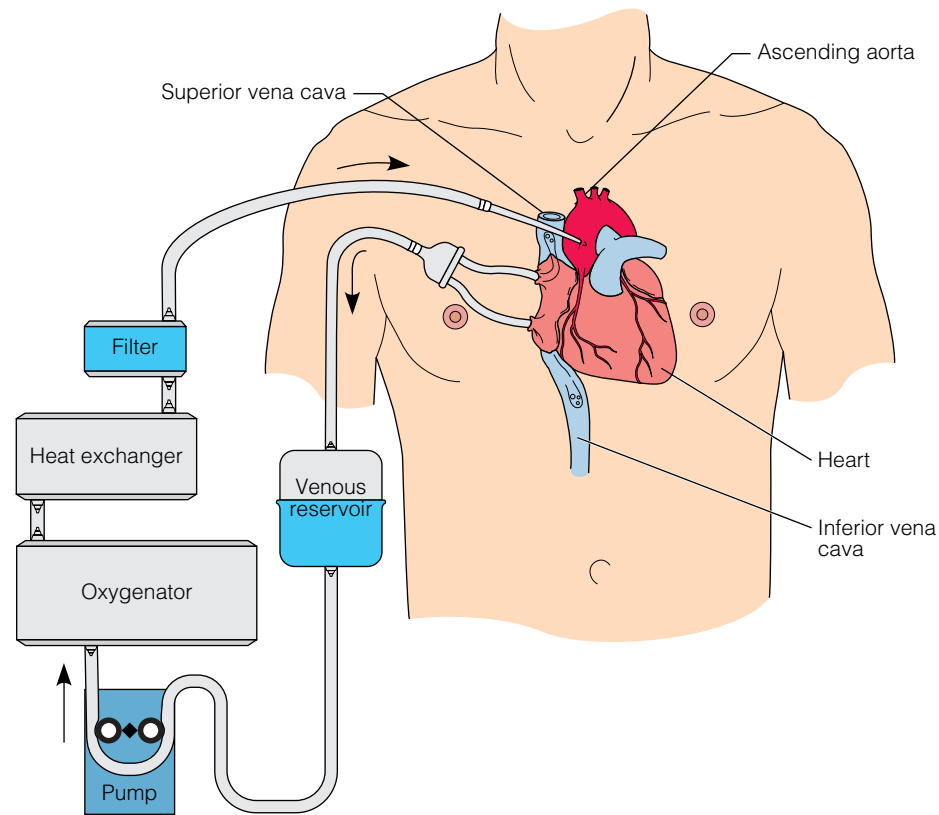
complete revascularization. While anginal pain may recur within 3 years, it rarely is as severe as before surgery. Coronary artery bypass graft has a positive effect on mortality in many cases. It is recommended for clients who have multiple vessel disease and impaired left ventricular function or diabetes, and for clients who have significant obstruction of the left main coronary artery (Kasper et al., 2005).

A median sternotomy commonly is used to access the heart. The heart is usually stopped during surgery. The *cardiopulmonary bypass (CPB) pump* is used to maintain perfusion to the rest of the organs during open-heart surgery. Venous blood is removed from the body through a cannula placed in the right atrium or the superior and inferior venae cavae. Blood then circulates through the CPB pump, where it is oxygenated, its temperature regulated, and it is filtered. Oxygenated blood is returned to the body through a cannula in the ascending aorta (Figure 31-4 ■). Cardiopulmonary bypass enables surgeons to

operate on a quiet heart and a relatively bloodless field. Hypothermia can be maintained to reduce the metabolic rate and decrease oxygen demand during surgery.

Newer techniques have been developed that allow surgeons to perform CABG without cardioplegia (stopping the heart) and CPB. Off-pump coronary artery bypass (OPCAB) allows use of a smaller incision for access. Although cardiopulmonary bypass is employed for the majority of coronary artery bypass procedures, OPCAB is a promising alternative. Controlled studies demonstrate lower mortality and morbidity rates and faster recovery for clients undergoing OPCAB as compared to CABG with cardiopulmonary bypass (Eagle et al., 2004).

When the saphenous vein is used, it is excised from its normal attachments in the leg, flushed with a cold heparinized saline solution, and then reversed so that its valves do not interfere with blood flow. When appropriate, a laparoscopic approach may be used to remove the vein. The vein is



**Figure 31–4** ■ A diagrammatic representation of cardiopulmonary bypass. A cannula in the superior and inferior venae cavae removes venous blood, which is then pumped through an oxygenator and heat exchanger. After filtering, oxygenated blood is returned to the ascending aorta.

*anastomosed* (grafted) to the aorta and the coronary artery, distal to the occlusion (see Figure 31–3). This provides a bridge or conduit for blood flow past the obstruction. If the internal mammary artery (IMA) is used, its distal end is excised and anastomosed to the coronary artery distal to the obstruction. The IMA often is used to revascularize the left coronary artery because of the greater oxygen demand of the left ventricle.

Once grafting is completed, cardiopulmonary bypass is discontinued and the client is rewarmed. Rewarming stimulates the heart to resume beating. Temporary pacing wires are sutured in place and passed through the chest wall in case temporary pacing is necessary. Chest tubes are placed in the pleural space and mediastinum to drain blood and reestablish negative pressure in the thoracic cavity. The sternum is closed using heavy wires and bone wax, the skin is closed with sutures or staples, and sterile dressings are applied over sternal and leg incisions.

Pre- and postoperative nursing care and teaching for the client having a coronary artery bypass graft or other open-heart surgery are outlined on pages 980–982.

**MINIMALLY INVASIVE CORONARY ARTERY SURGERY** *Minimally invasive coronary artery surgery* is a potential future alternative to CABG. Two approaches may be used: *Port-access coronary artery bypass* uses several small holes, or “ports,” in

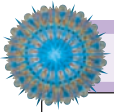
the chest wall to access vessels for connection to the CPB pump and the surgical site. Alternatively, the femoral artery and femoral vein may be used for CPB (Eagle et al., 2004). CPB is avoided altogether using the *minimally invasive direct coronary artery bypass (MIDCAB)* approach. With MIDCAB, a small surgical incision and several chest wall ports are used to graft a chest wall artery to the affected coronary vessel while the heart continues to beat.

**TRANSMYOCARDIAL LASER REVASCLARIZATION** A new development in myocardial revascularization techniques is called *transmyocardial laser revascularization (TMLR)*. In this procedure, a laser is used to drill tiny holes into the myocardial muscle itself to provide collateral blood flow to ischemic muscle. Clients whose coronary artery obstructions are too diffuse to bypass are candidates for this new surgical treatment.



## NURSING CARE

Health promotion, assessment, nursing diagnoses and interventions for the client with ACS are similar to those identified for clients with angina and with acute myocardial infarction. See the preceding and subsequent sections of this chapter for specific nursing care activities, as well as the Nursing Care Plan that follows.



## NURSING CARE OF THE CLIENT HAVING A Coronary Artery Bypass Graft

### PREOPERATIVE CARE

- Provide routine preoperative care and teaching as outlined in Chapter 4 ∞.
- Verify presence of laboratory and diagnostic test results in the chart, including CBC, coagulation profile, urinalysis, chest x-ray, and coronary angiogram. *These baseline data are important for comparison of postoperative results and values.*
- Type and crossmatch four or more units of blood as ordered. *Blood is made available for use during and after surgery as needed.*
- Provide specific client and family teaching related to procedure and postoperative care. Include the following topics.
  - Cardiac recovery unit; sensory stimuli, personnel; noise and alarms; visiting policies
  - Tubes, drains, and general appearance
  - Monitoring equipment, including cardiac and hemodynamic monitoring systems
  - Respiratory support: ventilator, endotracheal tube, suctioning; communication while intubated
  - Incisions and dressings
  - Pain management

*Preoperative teaching reduces anxiety and prepares the client and family for the postoperative environment and expected sensations.*

### POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 4 ∞. In addition to the care needs of all clients having major surgery, the cardiac surgery client has specific care needs related to open-heart and thoracic surgery. These are outlined under the nursing diagnoses identified below.

#### Decreased Cardiac Output

Cardiac output may be compromised postoperatively due to bleeding and fluid loss; depression of myocardial function by drugs, hypothermia, and surgical manipulation; dysrhythmias; increased vascular resistance; and a potential complication, *cardiac tamponade*, compression of the heart due to collected blood or fluid in the pericardium.

- Monitor vital signs, oxygen saturation, and hemodynamic parameters every 15 minutes. Note trends and report significant changes to the physician. *Initial hypothermia and bradycardia are expected; the heart rate should return to the normal range with rewarming. The blood pressure may fall during rewarming as vasodilation occurs. Hypotension and tachycardia, however, may indicate low cardiac output. Pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), cardiac output, and oxygen saturation are monitored to evaluate fluid volume, cardiac function, and gas exchange.* Hemodynamic monitoring is further discussed in Chapter 32 ∞.
- Auscultate heart and breath sounds on admission and at least every 4 hours. *A ventricular gallop, or S<sub>3</sub>, is an early sign of heart failure; an S<sub>4</sub> may indicate decreased ventricular compliance. Muffled heart sounds may be an early indication of cardiac tamponade. Adventitious breath sounds (wheezes, crackles, or rales) may be a manifestation of heart failure or respiratory compromise.*
- Assess skin color and temperature, peripheral pulses, and level of consciousness with vital signs. *Pale, mottled, or cyanotic*

*coloring, cool and clammy skin, and diminished pulse amplitude are indicators of decreased cardiac output.*

- Continuously monitor and document cardiac rhythm. *Dysrhythmias are common, and may interfere with cardiac filling and contractility, decreasing the cardiac output.*
- Measure intake and output hourly. Report urine output less than 30 mL/h for 2 consecutive hours. *Intake and output measurements help evaluate fluid volume status. A fall in urine output may be an early indicator of decreased cardiac output.*
- Record chest tube output hourly. *Chest tube drainage greater than 70 mL/h or that is warm, red, and free flowing indicates hemorrhage and may necessitate a return to surgery. A sudden drop in chest tube output may indicate impending cardiac tamponade.*
- Monitor hemoglobin, hematocrit, and serum electrolytes. *A drop in hemoglobin and hematocrit may indicate hemorrhage that is not otherwise obvious. Electrolyte imbalances, potassium, calcium, and magnesium in particular, affect cardiac rhythm and contractility.*
- Administer intravenous fluids, fluid boluses, and blood transfusions as ordered. *Fluid and blood replacement helps ensure adequate blood volume and oxygen-carrying capacity.*
- Administer medications as ordered. *Medications ordered in the early postoperative period to maintain the cardiac output include inotropic drugs (e.g., dopamine, dobutamine) to increase the force of myocardial contractions; vasodilators (e.g., nitroprusside or nitroglycerin) to decrease vascular resistance and after-load; and antidysrhythmics to correct dysrhythmias that affect cardiac output.*
- Keep a temporary pacemaker at the bedside; initiate pacing as indicated. *Temporary pacing may be needed to maintain the cardiac output with bradydysrhythmias, such as high-level AV blocks.*

### PRACTICE ALERT

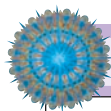
Assess for signs of cardiac tamponade: increased heart rate, decreased BP, decreased urine output, increased central venous pressure, a sudden decrease in chest tube output, muffled/distant heart sounds, and diminished peripheral pulses. Notify physician immediately. Cardiac tamponade is a life-threatening complication that may develop postoperatively. Cardiac tamponade interferes with ventricular filling and contraction, decreasing cardiac output. Untreated, cardiac tamponade leads to cardiogenic shock and possible cardiac arrest.

#### Hypothermia

Hypothermia is maintained during cardiac surgery to reduce the metabolic rate and protect vital organs from ischemic damage. Although rewarming is instituted on completion of the surgery, the client often remains hypothermic on admission to cardiac recovery. Gradual rewarming is necessary to prevent peripheral vasodilation and hypotension.

- Monitor core body temperature (e.g., tympanic membrane, pulmonary artery, bladder) for the first 8 hours following surgery. *Oral and rectal temperature measurements are not reliable indicators of core body temperature during this period.*





## NURSING CARE OF THE CLIENT HAVING A Coronary Artery Bypass Graft (continued)

- Institute rewarming measures (e.g., warmed intravenous solutions or blood transfusion, warm blankets, warm inspired gases, radiant heat lamps) as needed to maintain a temperature above 96.8°F (36°C). Administer Thorazine, morphine, or diltiazem as ordered to relieve shivering. *Low body temperature may cause shivering, increasing oxygen demand and consumption. Hypothermia also increases the risk for hypoxia, metabolic acidosis, vasoconstriction and increased cardiac work, altered clotting, and dysrhythmias.*

### Acute Pain

Following a CABG, pain is experienced due to both the thoracic incision and removal of the saphenous vein from the leg. Dissection of the internal mammary artery (usually the left IMA) from the chest wall also causes chest pain on the affected side. Chest tube sites are also uncomfortable. The leg from which the saphenous vein graft was obtained may be more painful than the chest incision.

- Frequently assess for pain, including its location and character. Document its intensity using a standard pain scale. Assess for verbal and nonverbal indicators of pain. Validate pain cues with the client. *Pain is subjective, and differs among individuals. Incisional pain is expected; however, anginal pain also may develop. It is important to differentiate the type of pain.*

### PRACTICE ALERT

Promptly report anginal or cardiac pain. Cardiac pain may indicate a perioperative or postoperative myocardial infarction.

- Administer analgesics on a scheduled basis, by PCA, or by continuous infusion for the first 24 to 48 hours. *Research demonstrates that adequate pain management in the immediate postoperative period reduces complications from sympathetic stimulation and allows faster recovery. Pain causes muscle tension and vasoconstriction, impairing circulation and tissue perfusion, slowing wound healing, and increasing cardiac work.*
- Premedicate 30 minutes before activities or planned procedures. *Premedication and the subsequent reduction of pain improves client participation and cooperation with care.*

### Ineffective Airway Clearance/Impaired Gas Exchange

Atelectasis due to impaired ventilation and airway clearance is a common pulmonary complication of cardiac surgery. Gas exchange may also be affected by blood loss and decreased oxygen-carrying capacity following surgery. Phrenic nerve paralysis is a potential complication of cardiac surgery which may also contribute to impaired ventilation and gas exchange.

- Evaluate respiratory rate, depth, effort, symmetry of chest expansion, and breath sounds frequently. *Pain, anxiety, excess fluid volume, surgical injury, narcotics and anesthesia, and altered homeostasis can affect respiratory rate, depth, and effort postoperatively. Decreased chest expansion or asymmetrical movement may indicate impaired ventilation of one lung, and needs further evaluation.*

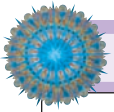
- Note endotracheal tube (ETT) placement on chest x-ray. Mark tube position and secure in place. Insert an oral airway if an oral ETT is used. *The chest x-ray documents correct ETT placement above the bifurcation to the right and left mainstem bronchus. Marking its appropriate placement allows evaluation of potential tube movement. Secure the tube firmly in place to prevent slippage or inadvertent removal. An oral airway helps prevent obstruction of an oral ETT by biting.*
- Maintain ventilator settings as ordered. Monitor arterial blood gases (ABGs) as ordered. *Mechanical ventilation promotes optimal lung expansion and oxygenation postoperatively. ABGs are used to evaluate oxygenation and acid-base balance.*
- Suction as needed. *Suctioning is performed only as indicated to clear airway secretions.*
- Prepare for ventilator weaning and extubation, as appropriate. *The client is removed from the ventilator and extubated as soon as possible to reduce complications associated with mechanical ventilation and intubation.*
- After extubation, teach use of the incentive spirometer, and encourage use every 2 hours. Encourage deep breathing; advise against vigorous coughing. Teach use of a “cough pillow” to splint chest incision and decrease pain. Frequently turn and encourage movement. Dangle on postoperative day 1. *Deep breathing, controlled coughing, and position changes improve ventilation and airway clearance and help prevent complications. Vigorous coughing may excessively increase intrathoracic pressure and cause sternal instability.*

### Risk for Infection

Following an open chest procedure, a sternal infection may develop that can progress to involve the mediastinum. Incisions for removal of the saphenous vein also may become infected. Clients with IMA grafts, who are diabetic, older, or malnourished, are at high risk: Harvesting of IMA disrupts blood supply to the sternum, and these clients have impaired immune responses and healing.

- Assess sternal incision and leg wounds every shift. Document redness, warmth, swelling, and/or drainage from the site. Note wound approximation. *These assessments provide indicators of inflammation and healing.*
- Maintain a sterile dressing for the first 48 hours, then leave the incision open to air. Use Steri-Strips as needed to maintain approximation of the wound edges. *The sterile dressing prevents early contamination of the wound, whereas exposing the incision after 48 hours promotes healing.*
- Report signs of wound infection: a swollen, reddened area that is hot and painful to the touch; drainage from the wound; impaired healing, or healed areas that reopen. *Evidence of infection or impaired healing requires further evaluation and treatment.*
- Culture wound drainage as indicated. *Identifying the infective organism facilitates appropriate antibiotic therapy.*
- Collaborate with the dietitian to promote nutrition and fluid intake. *Good nutritional status is vital to healing and immune function.*

(continued)



## NURSING CARE OF THE CLIENT HAVING A Coronary Artery Bypass Graft (continued)

### Disturbed Thought Processes

Many factors affect neuropsychologic function after CABG, including the length of cardiopulmonary bypass, age, presurgery organic brain dysfunction, severity of illness, and decreased cardiac output. Sensory overload and deprivation, sleep disruption, and numerous drugs also affect thinking and mental clarity.

- Frequently reorient during initial recovery period. State that surgery is over and that the client is in the recovery area. *Frequent reorientation provides emotional support and reality checks.*
- Explain all procedures before performing them. Speak in a clear, calm voice. Encourage questions, and give honest answers. *These measures provide information, decrease anxiety, and establish trust.*
- Secure all intravenous lines and invasive catheters/tubes (e.g., ETT, Foley catheter, nasogastric tube). *Disoriented clients may tug or pull at invasive equipment, disrupting them and increasing the risk of injury.*
- Note verbal responses to questions. Correct misconceptions immediately (e.g., “Mr. Snow, look at all the special equipment in this room. Does this room look like your bedroom at

home?”). *Helping the client recognize differences in the hospital environment offers a basis for continual reality checks.*

- Maintain a calendar and clock within the client’s view. *This provides current information regarding day, date, and time.*
- Involve family members in providing reorientation. Place familiar objects and photographs within view. Encourage family presence. *The family provides reassurance and contact with the familiar, assisting with orientation.*
- Promote client participation in care and decision making as appropriate. *This allows the client to maintain a degree of power and control and enables the client to take an active role in recovery.*
- Report signs of hallucinations, delusions, depression, or agitation. *These may indicate progressive deterioration of mental status.*
- Administer sedatives cautiously. *Mild sedation may help prevent injury. Some sedatives may, however, have adverse effects, increasing confusion and disorientation.*
- Reevaluate neurologic status every shift. *These data allow evaluation of the effect of interventions.*

## THE CLIENT WITH ACUTE MYOCARDIAL INFARCTION

An **acute myocardial infarction (AMI)**, necrosis (death) of myocardial cells, is a life-threatening event. If circulation to the affected myocardium is not promptly restored, loss of functional myocardium affects the heart’s ability to maintain an effective cardiac output. This may ultimately lead to cardiogenic shock and death.

Heart disease remains the leading cause of death in the United States. Of the major heart diseases, myocardial infarction or *heart attack*, and other forms of ischemic heart disease cause the majority of deaths. Annually, approximately 700,000 people in the United States experience their first MI; another 500,000 suffer an MI subsequent to the initial one. Nearly 492,000 people died of coronary heart disease in 2002, with most of these deaths related to MI (NHLBI, 2004).

The majority of deaths from MI occur during the initial period after symptoms begin: approximately 60% within the first hour, and 40% prior to hospitalization. Heightening public awareness of the manifestations of MI, the importance of seeking immediate medical assistance, and training in cardiopulmonary resuscitation (CPR) techniques are vital to decrease deaths due to MI.

Myocardial infarction rarely occurs in clients without pre-existing coronary heart disease. While no specific cause has been identified, the risk factors for MI are those for coronary heart disease: age, gender, heredity, race; smoking, obesity, hyperlipidemia, hypertension, diabetes, sedentary lifestyle, diet, and others. See the previous section of this chapter on coronary heart disease for further discussion of these risk factors.

### Pathophysiology

Atherosclerotic plaque may form stable or unstable lesions. *Stable* lesions progress by gradually occluding the vessel lu-

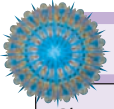
men, whereas *unstable* (or *complicated*) lesions are prone to rupture and thrombus formation. Stable lesions often cause angina (discussed in the previous sections); unstable lesions often lead to acute coronary syndromes, or acute ischemic heart diseases. Acute coronary syndromes include unstable angina, myocardial infarction, and sudden cardiac death (Braunwald et al., 2002).

Myocardial infarction occurs when blood flow to a portion of cardiac muscle is completely blocked, resulting in prolonged tissue ischemia and irreversible cell damage. Coronary occlusion is usually caused by ulceration or rupture of a complicated atherosclerotic lesion. When an atherosclerotic lesion ruptures or ulcerates, substances are released that stimulate platelet aggregation, thrombin generation, and local vasomotor tone. As a result, the vessel constricts and a thrombus (clot) forms, occluding the vessel and interrupting blood flow to the myocardium distal to the obstruction.

Cellular injury occurs when the cells are denied adequate oxygen and nutrients. When ischemia is prolonged, lasting more than 20 to 45 minutes, irreversible hypoxic damage causes cellular death and tissue necrosis. Oxygen, glycogen, and ATP stores of ischemic cells are rapidly depleted. Cellular metabolism shifts to an anaerobic process, producing hydrogen ions and lactic acid. Cellular acidosis increases cells’ vulnerability to further damage. Intracellular enzymes are released through damaged cell membranes into interstitial spaces.

Cellular acidosis, electrolyte imbalances, and hormones released in response to cellular ischemia affect impulse conduction and myocardial contractility. The risk for dysrhythmias increases, and myocardial contractility decreases, reducing stroke volume, cardiac output, blood pressure, and tissue perfusion.

The subendocardium suffers the initial damage, within 20 minutes of injury, because this area is the most susceptible to



## NURSING CARE PLAN A Client with Coronary Artery Bypass Surgery

Six weeks ago, John Clements, age 50, was discharged from the hospital after emergency triple bypass surgery. Despite having emergency surgery, his postoperative recovery was uneventful, and he was discharged 6 days after admission. He returns to the clinic for a postoperative stress test and to discuss his cardiac rehabilitation program. Anne Wagner, RN, CNS, a cardiac clinical nurse specialist and the program coordinator, meets Mr. Clements to obtain specific information regarding his medical status.

### ASSESSMENT

Mr. Clements's medical history reveals significant CHD, an anterior wall myocardial infarction that led to his emergency triple bypass, and hyperlipidemia. Current medications include Cardizem, Isordil, Ecotrin, and Transderm-Nitro 5. The ECG reveals sinus rhythm with some ST segment and T wave flattening. Resting heart rate 68, and blood pressure 136/84.

Mr. Clements has a strong family history of CHD. He does not smoke and uses alcohol occasionally in social situations. He enjoys "good Southern-style cooking" and watching television. Mr. Clements states his only regular exercise used to be an evening of dancing with his wife and friends about once a month, "But I get short of breath walking around the block now, so I guess I can't go dancing anymore!"

Mr. Clements owns his own contracting business and states that he typically works about 50 to 60 hours per week. He tells Ms. Wagner, "I don't know what this program is supposed to do for me. I have got to get back to work! You just can't sit around in my business—you have to make sure that the work is getting done on time, and you have to check on supplies and equipment and the like. But I feel like a weakling—I need to get my energy back!"

### DIAGNOSES

- *Activity Intolerance* related to general weakness and fatigue
- *Ineffective Role Performance* related to health crisis

### EXPECTED OUTCOMES

- Verbalize an understanding of the definition and components of his structured cardiac rehabilitation program.
- Verbalize a desire to make lifestyle changes.
- Identify resources available in the community to assist with lifestyle changes.
- Participate in his activity program without suffering any complications.
- Verbalize an increase in energy after 6 weeks on the program.
- Accept the reality of the temporary change in his usual work responsibilities.

### PLANNING AND IMPLEMENTATION

- Define the purpose and components of a cardiac rehabilitation program.
- Enroll in "heart health" classes, including cardiac anatomy, physiology, and coronary heart disease; exercise and activity prescriptions; lifestyle modifications, including diet counseling and stress management; emotional reactions to CAD; sexual activity; use of cardiac medications; and self-responsibility for health.
- Plan an exercise program based on stress test results, physical examination, and interview.
- Encourage to schedule rest periods before and after activity/exercise.
- Review signs and symptoms of overexertion.
- Provide information about community resources for emotional and educational support.
- Assist to identify strategies for dealing with concerns about his business role.

### EVALUATION

Mr. Clements decides to "give the rehab program a try." Ms. Wagner and an exercise physiologist work with him to plan an individualized exercise/activity program. A registered dietitian provides dietary counseling. Ms. Wagner emphasizes stress management strategies. Mr. Clements is able to list manifestations of overexertion and states that he realizes the need for gradual activity progression.

After 6 weeks, Mr. Clements has reported a significant increase in energy and strength. "I am feeling much stronger, and have been sleeping better. Mary and I are taking evening walks around the neighborhood. My chest soreness is also gone." He has completed the 12-week cardiac rehabilitation program, and another stress test indicates that his cardiac function is adequate. Mr. Clements has joined the local Mended Hearts support group and states that he is now incorporating "heart-healthy" considerations into his daily routines.

### CRITICAL THINKING IN THE NURSING PROCESS

1. Develop a personalized risk factor reduction plan for Mr. Clements.
2. How might denial affect Mr. Clements's ability to (a) accept the need for cardiac rehabilitation, (b) comply with the proposed lifestyle changes, and (c) make permanent adjustments to his daily life?
3. How does spousal support influence a client's compliance with a structured cardiac rehabilitation program?
4. Mr. Clements tells you that since the surgery, his wife has been afraid that sexual activity will induce another heart attack. How would you respond to these concerns?

*See Evaluating Your Response in Appendix C.*

changes in coronary blood flow. If blood flow is restored at this point, the infarction is limited to subendocardial tissue (a *subendocardial* or *non-Q-wave infarction*). The damage progresses to the epicardium within 1 to 6 hours. When all layers of the myocardium are affected, it is known as a *transmural infarction*. A significant Q wave develops with a transmural infarction, so this also may be called a *Q-wave MI*.

Complications such as heart failure are more frequently associated with Q-wave MIs; however, clients with non-Q-wave MIs frequently experience recurrent ischemia or subsequent MI within weeks or months of the event (Woods et al., 2004).

The necrotic, infarcted tissue is surrounded by regions of injured and ischemic tissues. Tissue in this ischemic area is potentially viable; restoration of blood flow minimizes the

amount of tissue lost. This surrounding tissue also undergoes metabolic changes. It may be *stunned*, its contractility impaired for hours to days following reperfusion, or *hibernating*, a process that protects myocytes until perfusion is restored. *Myocardial remodeling* also may occur, with cellular hypertrophy and loss of contractility in regions distant from the infarction. Rapid restoration of blood flow limits these changes (McCance & Huether, 2006).

When a larger artery is compromised, *collateral vessels* connecting smaller arteries in the coronary system dilate to maintain blood flow to the cardiac muscle. The degree of collateral circulation helps determine the extent of myocardial damage from ischemia. Acute occlusion of a coronary artery without any collateral flow results in massive tissue damage and possible death. Progressive narrowing of the larger coronary arteries allows collateral vessels to develop and enlarge, meeting the demand for blood flow. Good collateral circulation can limit the size of an MI.

Myocardial infarctions are described by the damaged area of the heart. The coronary artery that is occluded determines the area of damage. Myocardial infarction usually affects the left ventricle because it is the major “workhorse” of the heart; its muscle mass is greater, as are its oxygen demands. Occlusion of the left anterior descending (LAD) artery affects blood flow to the anterior wall of the left ventricle (an *anterior MI*) and part of the interventricular septum. Occlusion of the left circumflex artery (LCA) causes a *lateral MI*. *Right ventricular, inferior, and posterior infarcts* involve occlusions of the right coronary artery (RCA) and posterior descending artery (PDA). Occlusion of the left main coronary artery is the most devastating, causing ischemia of the entire left ventricle, and a grave prognosis. Identifying the infarct site helps predict possible complications and determine appropriate therapy.

### Cocaine-Induced MI

Acute myocardial infarction may develop due to cocaine intoxication. Cocaine increases sympathetic nervous system activity by both increasing the release of catecholamines from central and peripheral stores and interfering with the reuptake

of catecholamines. This increased catecholamine concentration stimulates the heart rate and increases its contractility, increases the automaticity of cardiac tissues and the risk of dysrhythmias, and causes vasoconstriction and hypertension. The client with cocaine-induced MI may present with an altered level of consciousness, confusion and restlessness, seizure activity, tachycardia, hypotension, increased respiratory rate, and respiratory crackles.

## Manifestations

Pain is a classic manifestation of myocardial infarction. Chest pain due to MI is more severe than anginal pain. However, it is not the intensity of the chest pain that distinguishes MI from angina or acute coronary syndrome, but its duration and its continuous nature. The onset of pain is sudden and usually is not associated with activity. In fact, most MIs occur in the early morning. Clients with a history of angina may have more frequent anginal attacks in the days or weeks prior to an MI (unstable angina or ACS). Chest pain may be described as crushing and severe; as a pressure, heavy, or squeezing sensation; or as chest tightness or burning. The pain often begins in the center of the chest (*substernal*), and may radiate to the shoulders, neck, jaw, or arms. It lasts more than 15 to 20 minutes and is not relieved by rest or nitroglycerin.

Women and older adults often experience atypical chest pain, presenting with complaints of indigestion, heartburn, nausea, and vomiting (see the box below). Up to 25% of clients with acute MI deny chest discomfort (Woods et al., 2004).

Compensatory mechanisms cause many of the other symptoms of MI. Sympathetic nervous system stimulation causes anxiety, tachycardia, and vasoconstriction. This results in cool, clammy, mottled skin. Pain and blood chemistry changes stimulate the respiratory center, causing tachypnea. The client often has a sense of impending doom and death. Tissue necrosis causes an inflammatory reaction that increases the white blood cell count and elevates the temperature. Serum cardiac enzyme levels rise as enzymes are released from necrotic cardiac cells.

### MEETING INDIVIDUALIZED NEEDS

#### Recognizing Acute Myocardial Infarction in Women and Older Adults

Women and older adults often present with atypical manifestations of MI. However, heart disease is the number one cause of death in both groups, making early recognition and aggressive treatment vital.

Women are more likely than men to have a “silent” or unrecognized heart attack, or to present in cardiac arrest or with cardiogenic shock. Women often experience epigastric pain and nausea, causing them to blame their discomfort on heartburn. Shortness of breath is common, as is fatigue and weakness of the shoulders and upper arms.

Older people often seek treatment for vague complaints of difficulty breathing, confusion, fainting, dizziness, abdominal pain, or

cough. They often attribute their symptoms to a stroke. The prevalence of silent ischemia is greater in older adults.

Stress the importance of seeking medical help promptly for atypical manifestations of MI. Prompt diagnosis and intervention reduces the mortality and morbidity of MI in women and older adults, just as it does in men. Despite this fact, both women and older adults are more likely to delay seeking treatment and are less likely to be accurately diagnosed and aggressively treated for CHD. Younger women are a particularly important group to reach; their mortality rate when MI occurs is twice that of men (Kasper et al., 2005).

Other manifestations may vary, depending on the location and amount of infarcted tissue. Hypertension, hypotension, or signs of heart failure may develop. Vagal stimulation may cause nausea and vomiting, bradycardia, and hypotension. Hiccups may develop due to diaphragmatic irritation. If a large vessel is occluded, the first sign of MI may be sudden death. Typical manifestations of MI are listed in the box below.

## Complications

The risk of complications associated with myocardial infarction is related to the size and location of the MI.

### Dysrhythmias

**Dysrhythmias**, disturbances or irregularities of heart rhythm, are the most frequent complication of MI. Dysrhythmias are discussed in detail in the next section of this chapter.

Infarcted tissue is *arrhythmogenic*; that is, it affects the generation and conduction of electrical impulses in the heart, increasing the risk of dysrhythmias. Premature ventricular contractions (PVCs) are common following an MI, developing in more than 90% of clients with an acute MI. While not dangerous in themselves, they may be predictive of more dangerous dysrhythmias such as ventricular tachycardia or ventricular fibrillation (Woods et al., 2004). The risk of ventricular fibrillation is greatest the first hour after MI; it is a frequent cause of sudden cardiac death associated with acute MI. Its incidence declines with time. If the infarct affects a conduction pathway, electrical conduction may be affected. Any degree of atrioventricular (AV) block may occur following MI, especially when the anterior wall is infarcted. First-degree and Mobitz I (Wenckebach) blocks are most common, although complete heart block may develop. Bradydysrhythmias (abnormal slow rhythms) also may develop, particularly when the inferior wall of the ventricle is affected.

### Pump Failure

Myocardial infarction reduces myocardial contractility, ventricular wall motion, and compliance. Impaired contractility

and filling may produce heart failure. The risk of heart failure is greatest when large portions of the left ventricle are infarcted. Heart failure may be more severe with an anterior infarction. Loss of 20% to 30% of the left ventricular muscle mass may cause manifestations of left-sided heart failure, including dyspnea, fatigue, weakness, and respiratory crackles on auscultation. Inferior or right ventricular MI may lead to right-sided heart failure with manifestations such as neck vein distention and peripheral edema. Hemodynamic monitoring is often initiated for clients with evidence of heart failure. Heart failure and its manifestations are discussed in greater depth in Chapter 32 ∞.

**CARDIOGENIC SHOCK** *Cardiogenic shock*, impaired tissue perfusion due to pump failure, results when functioning myocardial muscle mass decreases by more than 40%. The heart is unable to pump enough blood to meet the needs of the body and maintain organ function. Low cardiac output due to cardiogenic shock also impairs perfusion of the coronary arteries and myocardium, further increasing tissue damage. Mortality from cardiogenic shock is greater than 70%, although this can be reduced by prompt intervention with revascularization procedures. See Chapter 11 ∞ for a more extensive discussion of cardiogenic shock.

### Infarct Extension

Approximately 10% of clients experience extension or reinfarction in the area of the original infarction during the first 10 to 14 days after an MI. *Extension* of the MI is characterized by increased myocardial necrosis from continued blood flow impairment and ongoing injury. *Expansion* of the MI is described as a permanent expansion of the infarcted area from thinning and dilation of the muscle. Infarct extension and expansion may cause manifestations such as continuing chest pain, hemodynamic compromise, and worsening heart failure.

### Structural Defects

Necrotic muscle is replaced by scar tissue that is thinner than the ventricular muscle mass. This can lead to such complications as ventricular aneurysm, rupture of the interventricular septum or papillary muscle, and myocardial rupture. A *ventricular aneurysm* is an outpouching of the ventricular wall. It may develop when a large section of the ventricle is replaced by scar tissue. Because it does not contract during systole, stroke volume decreases. Blood may pool within the aneurysm, causing clots to form. Ischemia of the papillary muscle or chordae tendineae may cause structural damage leading to papillary muscle dysfunction or rupture. This affects AV valve function (usually the mitral valve), causing *regurgitation*, backflow of blood into the atria during systole. The interventricular septum may perforate or rupture due to ischemia and infarction. Myocardial rupture is a risk between days 4 and 7 after MI, when the injured tissue is soft and weak. This potential complication of MI is often fatal.

### Pericarditis

Tissue necrosis prompts an inflammatory response. *Pericarditis*, inflammation of the pericardial tissue surrounding the heart, may



## MANIFESTATIONS of Acute Myocardial Infarction

- Chest pain: substernal or precordial (across the entire chest wall); may radiate to neck, jaw, shoulder(s), or left arm
- Tachycardia, tachypnea
- Dyspnea, shortness of breath
- Nausea and vomiting
- Anxiety, sense of impending doom
- Diaphoresis
- Cool, mottled skin; diminished peripheral pulses
- Hypotension or hypertension
- Palpitations, dysrhythmias
- Signs of left heart failure
- Decreased level of consciousness

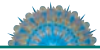
complicate AMI, usually within 2 to 3 days. Pericarditis causes chest pain that may be aching or sharp and stabbing, aggravated by movement or deep breathing. A *pericardial friction rub* may be heard on auscultation of heart sounds.

*Dressler's syndrome*, thought to be a hypersensitivity response to necrotic tissue or an autoimmune disorder, may develop days to weeks after AMI. It is a symptom complex characterized by fever, chest pain, and dyspnea. Dressler's syndrome may spontaneously resolve or recur over several months, causing significant discomfort and distress.

### FAST FACTS

- Dysrhythmias are the most common complication of AMI.
- Heart failure also is a common complication or consequence of myocardial infarction, developing due to loss of functional muscle tissue.

## INTERDISCIPLINARY CARE



Immediate treatment goals for the MI client are to:

- Relieve chest pain.
- Reduce the extent of myocardial damage.
- Maintain cardiovascular stability.
- Decrease cardiac workload.
- Prevent complications.

Slowing the process of coronary heart disease and reducing the risk of future MI is a major long-term management goal for the client.

Rapid assessment and early diagnosis is important in treating AMI. “Time is muscle” is a medical truism for the client with AMI. The evolution of an AMI is dynamic: The quicker the artery is reopened (medically, surgically, or spontaneously), the more myocardium can be salvaged. Survival and long-term outcomes following AMI are improved by rapidly restoring blood flow to the “stunned” myocardium surrounding the infarcted tissue, reducing myocardial oxygen demand and limiting the accumulation of toxic by-products of necrosis and reperfusion (Kasper et al., 2005). The AHA recommends initiation of definitive treatment within 1 hour of entry into the healthcare system. A recent study by De Luca et al. (2004) showed that every minute of delay in treating clients with AMI affects the mortality risk during the first year.

The major problem interfering with timely reperfusion is delay in seeking medical care following the onset of symptoms. Up to 44% of clients with symptoms of chest discomfort or pain wait more than 4 hours before seeking treatment. Many factors are cited as reasons for treatment delay, including advanced age, the perception of the seriousness of symptoms, denial, access to medical care, the availability of an emergency response system, and in-hospital delays. Immediate evaluation of the client presenting with manifestations of myocardial infarction is essential to early diagnosis and treatment.

### Diagnosis

Diagnostic testing is used to establish the diagnosis of AMI.

*Serum cardiac markers* are proteins released from necrotic heart muscle. The proteins most specific for diagnosis of MI

are the creatine kinase (CK, or creatine phosphokinase, CPK) and cardiac-specific troponins (Table 31–6).

- *Creatine kinase* is an important enzyme for cellular function found principally in cardiac and skeletal muscle and the brain. CK levels rise rapidly with damage to these tissues, appearing in the serum 4 to 6 hours after AMI, peaking within 12 to 24 hours, and then declining over the next 48 to 72 hours. The CK level correlates with the size of the infarction; the greater the amount of infarcted tissue, the higher the serum CK level.
- *CK-MB* (also called MB-bands) is a subset of CK specific to cardiac muscle. This isoenzyme of CK is considered the most sensitive indicator of MI. Elevated CK alone is not specific for MI; elevated CK-MB greater than 5% is considered a positive indicator of MI. CK-MB levels do not normally rise with chest pain from angina or causes other than MI.
- Cardiac muscle troponins, *cardiac-specific troponin T* ( $cT_nT$ ) and *cardiac-specific troponin I* ( $cT_nI$ ), are proteins released during myocardial infarction that are sensitive indicators of myocardial damage. These proteins are part of the actin-myoclin unit in cardiac muscle and normally are not detectable in the blood. With necrosis of cardiac muscle, troponins are released and blood levels rise. The specificity of  $cT_nT$  and  $cT_nI$  to cardiac muscle necrosis makes these markers particularly useful when skeletal muscle trauma contributes to elevated CK levels (e.g., when CPR has been performed or traumatic injury occurred at the time of the MI). They are sensitive enough to detect very small infarctions that do not cause significant CK elevation. Both  $cT_nT$  and  $cT_nI$  remain in the blood for 10 to 14 days after an MI, making them useful to diagnose MI when medical treatment is delayed.

Serum levels of cardiac markers are ordered on admission and for 3 succeeding days. Serial blood levels help establish the diagnosis and determine the extent of myocardial damage.

Other laboratory tests may include the following:

- *Myoglobin* is one of the first cardiac markers to be detectable in the blood after an MI. It is released within a few hours of symptom onset. Its lack of specificity to cardiac muscle and rapid excretion (blood levels return to normal within 24 hours) limit its use, however (Kasper et al., 2005).
- *Complete blood count (CBC)* shows an elevated white blood cell (WBC) count due to inflammation of the injured myocardium. The *erythrocyte sedimentation rate (ESR)* also rises because of inflammation.
- *Arterial blood gases (ABGs)* may be ordered to assess blood oxygen levels and acid–base balance.

Electrocardiography, echocardiography, and myocardial nuclear scans are the most common diagnostic tests performed when AMI is suspected. With the exception of the ECG, the timing of these tests depends on the client's immediate condition. Hemodynamic monitoring may be initiated in the unstable client following MI.

- The *electrocardiogram* reflects changes in conduction due to myocardial ischemia and necrosis. Classic ECG changes

TABLE 31–6 Cardiac Markers

MARKER	NORMAL LEVEL	PRIMARY TISSUE LOCATION	SIGNIFICANCE OF ELEVATION	Changes Occurring with MI		
				APPEARS	PEAKS	DURATION
CK (CPK)	Male: 12 to 80 unit/L Female: 10 to 70 unit/L	Cardiac muscle, skeletal muscle, brain	Injury to muscle cells	3 to 6 hours	12 to 24 hours	24 to 48 hours
CK-MB	0% to 3% of total CK	Cardiac muscle	MI, cardiac ischemia, myocarditis, cardiac contusion, defibrillation	4 to 8 hours	18 to 24 hours	72 hours
cT <sub>n</sub> T	<0.2 mcg/L	Cardiac muscle	Acute MI, unstable angina	2 to 4 hours	24 to 36 hours	10 to 14 days
cT <sub>n</sub> I	<3.1 mcg/L	Cardiac muscle	Acute MI, unstable angina	2 to 4 hours	24 to 36 hours	7 to 10 days

seen in AMI include T-wave inversion, ST-segment elevation, and formation of a Q wave. Ischemic changes in the heart are seen as depression of the ST segment or inversion of the T wave (see Figure 31–2). With myocardial injury, elevation of the ST segment occurs (Figure 31–5A ■). Significant Q-wave development (Figure 31–5B) indicates a transmural, or full-thickness infarction. Myocardial damage can be localized using the 12-lead ECG. See Chapter 30 ∞ for more information about ECGs.

- *Echocardiography* is done to evaluate cardiac wall motion and left ventricular function. Stunned and infarcted tissue does not contract as effectively (if at all) as healthy myocardium.
- *Radionuclide imaging* may be done to evaluate myocardial perfusion. These studies cannot differentiate between an acute MI and old scar tissue, but do help identify the specific area of myocardial ischemia and damage.
- *Hemodynamic monitoring* may be initiated when AMI significantly affects cardiac output and hemodynamic status. These invasive procedures are described in Chapter 32 ∞.

### Medications

Aspirin, a platelet inhibitor, is now considered an essential part of treating AMI. A 160- to 325-mg aspirin tablet is given by emergency personnel, with the instruction that it is to be chewed (for buccal absorption). This initial dose is followed by a daily oral dose of 160 to 325 mg of aspirin.

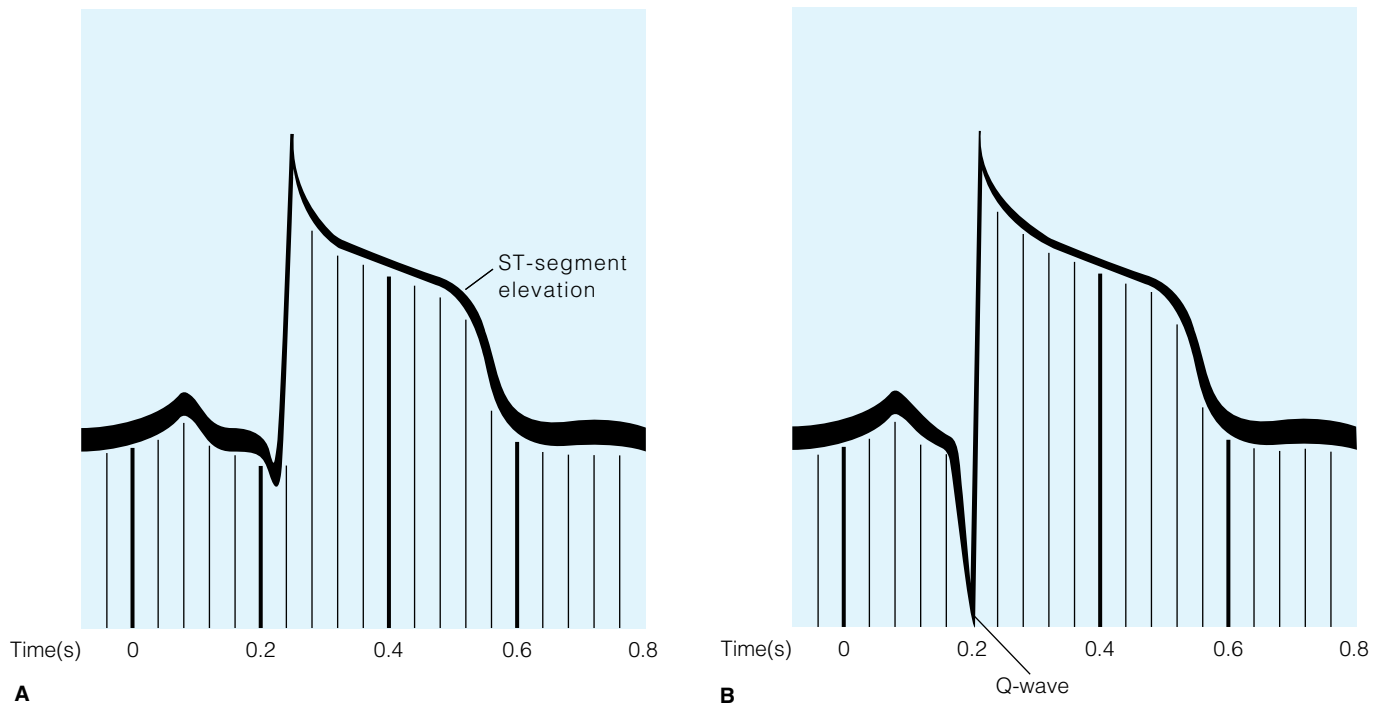
Fibrinolytic agents, analgesics, and antidysrhythmic agents are among the principal classes of drugs used in treating AMI.

**ANALGESIA** Pain relief is vital in treating the client with AMI. Pain stimulates the sympathetic nervous system, increasing the heart rate and blood pressure and, in turn, myocardial workload. Sublingual nitroglycerin may be given (up to three 0.4-mg doses at 5-minute intervals). Intravenous nitroglycerin may be continued for the first 24 to 48 hours to reduce myocardial work. In addition to pain relief, nitroglycerin decreases myocardial oxygen demand and may increase the

supply of oxygen to the myocardium. Nitroglycerin is a peripheral and arterial vasodilator that reduces afterload. It dilates coronary arteries and collateral channels in the heart, increasing coronary blood flow to save myocardial tissue at risk. Nitrates may, however, cause reflex tachycardia or excessive hypotension, so close monitoring is necessary during administration. It also is important to ask the client about use of sildenafil (Viagra) within the previous 24 hours before administering nitroglycerin, as the combination can precipitate a significant drop in blood pressure. See the Medication Administration box on page 973 for the nursing implications of nitroglycerin and other drugs given to reduce myocardial work following AMI.

Morphine sulfate is the drug of choice for pain unrelieved by nitroglycerin and for sedation. Following an initial intravenous dose of 4 to 8 mg, small doses (2 to 4 mg) may be repeated intravenously every 5 minutes until pain is relieved. It is important to assess frequently for pain relief and possible adverse effects of analgesia, such as excessive sedation. Pain unrelieved by expected or usual doses should be reported to the physician as it may indicate a complication such as extension of the infarct. See Chapter 9 ∞ for more details about morphine administration. Antianxiety agents such as diazepam (Valium) may also be administered to promote rest.

**FIBRINOLYTIC THERAPY** Fibrinolytic agents, drugs that dissolve or break up blood clots, are first-line drugs used to treat acute MI when access to a cardiac catheterization lab for revascularization procedures is not immediately available. Fibrinolytic drugs activate the fibrinolytic system to *lyse* or destroy the clot, restoring blood flow to the obstructed artery. Early fibrinolytic administration (within the first 6 hours of MI onset) limits infarct size, reduces heart damage, and improves outcomes. Activation of the fibrinolytic system can cause multiple complications; approximately 0.5% to 5% of clients receiving fibrinolytic drugs experience serious bleeding complications. Not every client is a candidate for fibrinolytic therapy; for example, it is contraindicated in clients



**Figure 31-5** ■ ECG changes characteristic of MI. *A*, ST-segment elevation characteristic of myocardial injury. *B*, Clinically significant Q-wave characteristic of a transmural infarction.

with known bleeding disorders, history of cerebrovascular disease, uncontrolled hypertension, pregnancy, or recent trauma or surgery of the head or spine (Tierney et al., 2005).

Several fibrinolytic agents are commonly used today. Among these, little difference in effectiveness has been demonstrated; there are, however, big differences in cost. Streptokinase, a biologic agent derived from group C *Streptococcus* organisms, is the least expensive of the drugs. Its primary drawback is the risk of a severe hypersensitivity reaction, including anaphylaxis. Streptokinase is administered by intravenous infusion. Anisoylated plasminogen streptokinase activator complex (APSAC) is a related drug that can be administered by bolus over 2 to 5 minutes. It has many of the same effects as streptokinase, but is considerably more expensive. Tissue plasminogen activator (t-PA), tenecteplase (TNK), and reteplase (rPA) are more effective in reestablishing myocardial perfusion, especially when the pain developed more than 3 hours previously. These drugs, however, are the most expensive. Nursing care of the client receiving a fibrinolytic agent is outlined on the next page.

**ANTIDYSRHYTHMICS** Dysrhythmias are a common complication of AMI, particularly in the first 12 to 24 hours. Antidysrhythmic medications are used as needed to treat dysrhythmias. They also may be given prophylactically to prevent dysrhythmias. Ventricular dysrhythmias are treated with a class I or class III antidysrhythmic drug (see the Medication Administration box on page 1006). Symptomatic bradycardia (bradycardia with associated hypotension and other signs of low cardiac output) is treated with intravenous atropine, 0.5 to 1 mg. Intra-

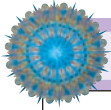
venous verapamil or the short-acting beta blocker esmolol (Brevibloc) may be ordered to treat atrial fibrillation or other supraventricular tachydysrhythmias.

**OTHER MEDICATIONS** Beta blockers such as propranolol (Inderal), atenolol (Tenormin), and metoprolol (Lopressor) reduce pain, limit infarct size, and decrease the incidence of serious ventricular dysrhythmias in AMI. These drugs decrease the heart rate, reducing cardiac work and myocardial oxygen demand. Initial doses are given intravenously. Oral beta blocker therapy is continued to reduce the risk of reinfarction and death related to cardiovascular causes (Kasper et al., 2005).

ACE inhibitors also reduce mortality associated with AMI. These drugs reduce ventricular remodeling following an MI, reducing the risk for subsequent heart failure. They also may reduce the risk of reinfarction (Kasper et al., 2002).

Anticoagulants and antiplatelet medications often are prescribed to maintain coronary artery patency following thrombolysis or a revascularization procedure. Abciximab (ReoPro) suppresses platelet aggregation and reduces the risk of reocclusion following angioplasty. It also improves vessel opening with fibrinolytic therapy, permitting lower doses of fibrinolytic drugs. Standard or low-molecular-weight heparin preparations often are given to clients with AMI. Heparin helps establish and maintain patency of the affected coronary artery. It also is used, along with long-term warfarin, to prevent systemic or pulmonary embolism in clients with significant left ventricular impairment or atrial fibrillation following AMI. See the Medication Administration box on page 976 for the nursing implications of an-





## NURSING CARE OF THE CLIENT RECEIVING Fibrinolytic Therapy

### PREINFUSION CARE


- Obtain nursing history, and perform a physical assessment. *Information obtained from the history and physical exam helps determine whether fibrinolytic therapy is appropriate. The goal is to initiate fibrinolytic therapy within 30 minutes of arrival.*
- Evaluate for contraindications to fibrinolytic therapy: recent surgery or trauma (including prolonged CPR), bleeding disorders or active bleeding, cerebral vascular accident, neurosurgery within the last 2 months, gastrointestinal ulcers, diabetic hemorrhagic retinopathy, and uncontrolled hypertension. *Fibrinolytic agents dissolve clots and therefore may precipitate intracranial, internal, or peripheral bleeding.*
- Inform the client of the purpose of the therapy. Discuss the risk of bleeding and the need to keep the extremity immobile during and after the infusion. *Minimal movement of the extremity is necessary to prevent bleeding from the infusion site.*


### DURING THE INFUSION

- Assess and record vital signs and the infusion site for hematoma or bleeding every 15 minutes for the first hour, every 30 minutes for the next 2 hours, and then hourly until the intravenous catheter is discontinued. Assess pulses, color, sensation, and temperature of both extremities with each vital sign check. *Vital signs and the site are frequently assessed to detect possible complications.*
- Remind the client to keep the extremity still and straight. Do not elevate head of bed above 15 degrees. *Extremity immobilization helps prevent infusion site trauma and bleeding. Hypotension may develop; keeping the bed flat helps maintain cerebral perfusion.*
- Maintain continuous cardiac monitoring during the infusion. Keep antidysrhythmic drugs and the emergency cart readily available for treatment of significant dysrhythmias. *Ventricular dysrhythmias commonly occur with reperfusion of the ischemic myocardium.*

### POSTINFUSION CARE

- Assess vital signs, distal pulses, and infusion site frequently as needed. *The client remains at high risk for bleeding following fibrinolytic therapy.*
- Evaluate response to therapy: normalization of ST segment, relief of chest pain, reperfusion dysrhythmias, early peaking of the CK and CK-MB. *These are signs that the clot has been dissolved and the myocardium is being reperfused.*
- Maintain bed rest for 6 hours. Keep the head of the bed at or below 15 degrees. Reinforce the need to keep the extremity straight and immobile. Avoid any injections for 24 hours after catheter removal. *Precautions such as these are important to prevent bleeding.*
- Assess puncture sites for bleeding. On catheter removal hold direct pressure over the site for at least 30 minutes. Apply a pressure dressing to any venous or arterial sites as needed. Perform routine care in a gentle manner to avoid bruising or injury. *Fibrinolytic therapy disrupts normal coagulation. Peripheral bleeding may occur at puncture sites, and there may not be sufficient fibrin to form a clot. Direct or indirect pressure may be needed to control the bleeding.*
- Assess body fluids, including urine, vomitus, and feces, for evidence of bleeding; frequently assess for changes in level of consciousness and manifestations of increased intracranial pressure, which may indicate intracranial bleeding. Assess surgical sites for bleeding. Monitor hemoglobin and hematocrit levels, prothrombin time (PT), and partial thromboplastin time (PTT). *These provide additional means of assessing for bleeding.*
- Administer platelet-modifying drugs (e.g., aspirin, dipyridamole) as ordered. *Platelet inhibitors decrease platelet aggregation and adhesion and are used to prevent reocclusion of the artery.*
- Report manifestations of reocclusion, including changes in the ST segment, chest pain, or dysrhythmias. *Early recognition of reocclusion is vital to save myocardial tissue.*

tiplatelet drugs, and Chapter 34  for more information about anticoagulant therapy.

Clients with pump failure and hypotension may receive intravenous dopamine, a vasopressor. At low doses (less than 5 mg/kg/min), it improves blood flow to the kidneys, preventing renal ischemia and possible acute renal failure (see Chapter 29 ). With increasing doses, dopamine increases myocardial contractility and causes vasoconstriction, improving blood pressure and cardiac output.

Antilipemic agents are used for the client with hyperlipidemia. A stool softener such as docusate sodium is prescribed to maintain normal bowel function and reduce straining.

### Treatments

The client with a suspected or confirmed MI is monitored continuously. Care is provided in the intensive coronary care unit for the first 24 to 48 hours, after which time less intensive monitoring (e.g., telemetry) may be required. An intra-

venous line is established to allow rapid administration of emergency medications.

Bed rest is prescribed for the first 12 hours to reduce the cardiac workload. The bedside commode generally is allowed; studies have shown this to be less stressful than using a bedpan. If the client's condition is stable, sitting in a chair at the bedside is permitted after 12 hours. Activities are gradually increased as tolerated. A quiet, calm environment with limited outside stimuli is preferred. Visitors are limited to promote rest. Oxygen is administered by nasal cannula at 2 to 5 L/min to improve oxygenation of the myocardium and other tissues.

A liquid diet may be prescribed for the first 4 to 12 hours to reduce gastric distention and myocardial work. Following that, a low-fat, low-cholesterol, reduced-sodium diet is allowed. Sodium restrictions may be lifted after 2 to 3 days if no evidence of heart failure is present. Small, frequent feedings are often recommended. Drinks containing caffeine, and very hot and cold foods, may also be limited.

## Revascularization Procedures

Many clients with AMI are treated with immediate or early percutaneous coronary revascularization such as angioplasty and stent placement. PCR may follow fibrinolytic therapy or be used in place of fibrinolytic therapy to restore blood flow to ischemic myocardium. When compared with fibrinolytic therapy, prompt PCR reduces hospital mortality (Kasper et al., 2005). In some cases, CABG surgery may be performed. The choice of procedure depends on the client's age and immediate condition, the time elapsed from the onset of manifestations, and the extent of myocardial disease and damage. These procedures and related nursing care are covered in more depth in the preceding section on acute coronary syndrome.

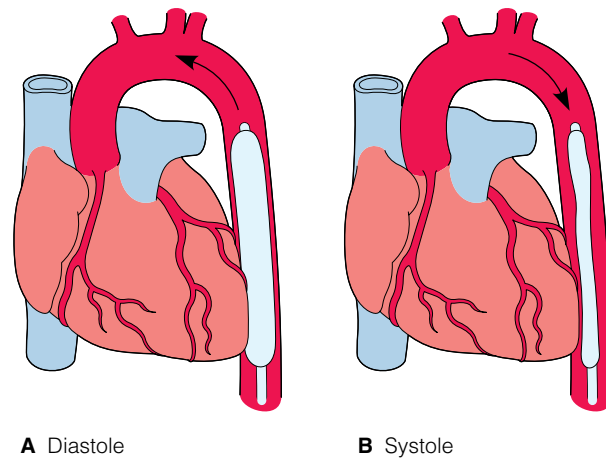
## Other Invasive Procedures

For clients with large MIs and evidence of pump failure, invasive devices may be used to temporarily take over the function of the heart, allowing the injured myocardium to heal. The intra-aortic balloon pump is widely used to augment cardiac output. Ventricular assist devices are indicated for clients requiring more or longer term artificial support than the intra-aortic balloon pump provides.

**INTRA-AORTIC BALLOON PUMP** The *intra-aortic balloon pump (IABP)*, also called intra-aortic balloon counterpulsation, is a mechanical circulatory support device that may be used after cardiac surgery or to treat cardiogenic shock following AMI. The IABP temporarily supports cardiac function, allowing the heart gradually to recover by decreasing myocardial workload and oxygen demand and increasing perfusion of the coronary arteries.

A catheter with a 30- to 40-mL balloon is introduced into the aorta, usually via the femoral artery. The balloon catheter is connected to a console that regulates the inflation and deflation of the balloon. The IABP catheter inflates during diastole, increasing perfusion of the coronary and renal arteries, and deflates just prior to systole, decreasing afterload and cardiac workload (Figure 31–6 ■). The inflation–deflation sequence is triggered by the ECG pattern. During the most acute period, the balloon inflates and deflates with each heartbeat (1:1 ratio), providing maximal assistance to the heart. As the client's condition improves, the IABP is weaned to inflate–deflate at varying intervals (e.g., 1:2, 1:4, 1:8). This provides a continually decreasing amount of support as the heart muscle recovers. When mechanical assistance is no longer required, the IABP catheter is removed.

**VENTRICULAR ASSIST DEVICES** Use of *ventricular assist devices (VADs)* to aid the failing heart is becoming more common with advances in technology. Whereas the IABP can supplement cardiac output by approximately 10% to 15%, the VAD temporarily takes partial or complete control of cardiac function, depending on the type of device used. VADs may be used as temporary or complete assist in AMI and cardiogenic shock when there is a chance for recovery of normal heart function after a period of cardiac rest. The device also may be

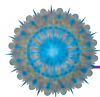


**Figure 31–6 ■** The intra-aortic balloon pump. *A*, When inflated during diastole, the balloon supports cerebral, renal, and coronary artery perfusion. *B*, The balloon deflates during systole, so cardiac output is unimpeded.

used as a bridge to heart transplant. Nursing care for the client with a VAD is supportive and includes assessing hemodynamic status and for complications associated with the device. Clients with VAD are at considerable risk for infection; strict aseptic technique is used with all invasive catheters and dressing changes. Pneumonia also is a risk due to immobility and ventilatory support. Mechanical failure of the VAD is a life-threatening event that requires immediate intervention (Urden et al., 2006).

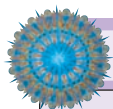
## Cardiac Rehabilitation

**Cardiac rehabilitation** is a long-term program of medical evaluation, exercise, risk factor modification, education, and counseling designed to limit the physical and psychologic effects of cardiac illness and improve the client's quality of life (Woods et al., 2004). Cardiac rehabilitation begins with admission for a cardiac event such as AMI or a revascularization procedure. Phase 1 of the program is the inpatient phase. A thorough assessment of the client's history, current status, risk factors, and motivation is obtained. During this phase, activity progresses from bed rest to independent performance of activities of daily living (ADLs) and ambulation within the facility. Both subjective and objective responses to increasing activity levels are evaluated. Excess fatigue, shortness of breath, chest pain, tachypnea, tachycardia, or cool, clammy skin indicate activity intolerance. Phase 2, immediate outpatient cardiac rehabilitation, begins within 3 weeks of the cardiac event. The goals for the outpatient program are to increase activity level, participation, and capacity; improve psychosocial status and treat anxiety or depression; and provide education and support for risk factor reduction. Continuation programs, phase 3 of cardiac rehabilitation, are directed at providing a transition to independent exercise and exercise maintenance. During this final phase, the client may “check in” every 3 months to evaluate risk factors, quality of life, and exercise habits (Woods et al., 2004).



## NURSING CARE

Nursing care of the client with an acute myocardial infarction focuses on reducing cardiac work, identifying and treating complications in a timely manner, and preparing the client for rehabilitation. See the following Nursing Care Plan for a client with an AMI.



### NURSING CARE PLAN A Client with Acute Myocardial Infarction

Betty Williams, a 62-year-old psychologist, is admitted to the emergency department with complaints of severe substernal chest pain. Mrs. Williams states that the pain began after lunch, about 4 hours ago. She initially attributed the pain to indigestion. She described the pain, which now radiates to her jaw and left arm, as “really severe heartburn.” It is accompanied by a “choking feeling,” severe shortness of breath, and diaphoresis. The pain is unrelieved by rest, antacids, or three sublingual nitroglycerin tablets (0.4 mg).

Oxygen is started per nasal cannula at 5 L/min. Central and peripheral intravenous lines are inserted. A 12-lead ECG and the following labwork are obtained: cardiac troponins, CK and CK isoenzymes, ABGs, CBC, and a chemistry panel. Morphine sulfate relieves Mrs. Williams’s pain.

Mrs. Williams’s medical history includes type 2 diabetes, angina, and hypertension. She has a 45-year history of cigarette smoking, averaging 1.5 to 2 packs per day. Family history reveals that Mrs. Williams’s father died at age 42 of AMI, and her paternal grandfather died at age 65 of AMI. Mrs. Williams is taking the following medications: tolbutamide (Orinase), hydrochlorothiazide, and isosorbide (Isordil).

Based on ECG changes and cardiac markers, an acute anterior MI is diagnosed. Mrs. Williams has no contraindications to fibrinolytic therapy and is deemed a good candidate. Intravenous alteplase (t-PA, Activase) is given by bolus followed by intravenous infusions of alteplase and heparin. She is transferred to the coronary care unit (CCU).

#### ASSESSMENT

Dan Morales, RN, is Mrs. Williams’s primary care nurse. Mrs. Williams is alert and oriented to person, place, and time. Vital signs are T 99.6°F (37.5°C), P 118, R 24 with adequate depth, and BP 172/92. Auscultation reveals an S<sub>4</sub> and fine crackles in the bases of both lungs. The ECG shows sinus tachycardia with occasional PVCs. Her skin is cool and slightly diaphoretic. Capillary refill is less than 3 seconds, and peripheral pulses are strong and equal. Her nail beds are pink.

A triple-lumen central line is in place. Nitroglycerin is infusing at 200 mcg/min in the distal lumen; the alteplase infusion is in the middle lumen; and a heparin infusion is in the proximal lumen. The peripheral intravenous line has a saline lock. Mrs. Williams states, “The pain is better since the nurse in the ER gave me a shot. But it has been coming and going. I would rate it a 4 right now, but it was terrible before. The doctor told me that this drug I’m getting will quickly open up the artery that is blocked. I hope it works! Do many people get this drug?”

## Health Promotion

Health promotion activities to prevent acute myocardial infarction are those outlined for coronary heart disease and angina in previous sections of this chapter. In addition, discuss risk factor management, use of prescribed medications, and cardiac rehabilitation to reduce the risk of complications or future infarctions.

#### DIAGNOSES

- *Acute Pain* related to ischemic myocardial tissue
- *Anxiety and Fear* related to change in health status
- *Ineffective Protection* related to the risk of bleeding secondary to fibrinolytic therapy
- *Risk for Decreased Cardiac Output* related to altered cardiac rate and rhythm

#### EXPECTED OUTCOMES

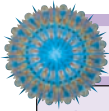
- Rate chest pain as 2 or lower on a pain scale of 0 to 10.
- Verbalize reduced anxiety and fear.
- Demonstrate no signs of internal or external bleeding.
- Maintain an adequate cardiac output during and following reperfusion therapy.

#### PLANNING AND IMPLEMENTATION

The following interventions are planned and implemented during the immediate phase of Mrs. Williams’s hospitalization.

- Instruct to report all chest pain. Monitor and evaluate pain using a scale of 0 to 10. Titrate intravenous nitroglycerin infusion for chest pain; stop infusion if systolic BP is below 100 mmHg. Administer 2 to 4 mg morphine intravenously for chest pain unrelieved by nitroglycerin infusion.
- Encourage verbalization of fears and concerns. Respond honestly, and correct misconceptions about the disease, therapeutic interventions, or prognosis.
- Assess knowledge of CHD. Explain the purpose of fibrinolytic therapy to dissolve the fresh clot and reperfuse the heart muscle, limiting heart damage.
- Explain the need for frequent monitoring of vital signs and potential bleeding.
- Assess for manifestations of internal or intracranial bleeding: complaints of back or abdominal pain, headache, decreased level of consciousness, dizziness, bloody secretions or excretions, or pallor. Test all stools, urine, and vomitus for occult blood. Notify physician immediately of any abnormal findings.
- Monitor for signs of reperfusion: decreased chest pain, return of ST segment to baseline, reperfusion dysrhythmias (e.g., PVCs, bradycardia, and heart block).
- Continuously monitor ECG for changes in cardiac rate, rhythm, and conduction. Assess vital signs.
- Treat dangerous dysrhythmias or other cardiac events per protocol. Notify the physician.
- Discuss continuing cardiac care and rehabilitation.

(continued)



## NURSING CARE PLAN A Client with Acute Myocardial Infarction (continued)

### EVALUATION

The initial morphine dose reduces Mrs. Williams's chest pain from a rating of 8 to 4. The nitroglycerin infusion and fibrinolytic therapy further reduce her pain to 2. The nitroglycerin infusion is gradually discontinued after 24 hours. As her pain subsides, Mrs. Williams states that she feels "much better now that the pain is gone. I was afraid it would just get worse." She verbalizes an understanding of fibrinolytic therapy to limit myocardial damage. No indication of bleeding problems are noted. Reperfusion is indicated by relief of chest pain, return of the ST segment to baseline on the ECG, early peaking of CK levels, and increased frequency of PVCs but no significant dysrhythmias. Mrs. Williams remains in CCU for 36 hours and is transferred to the floor.

### CRITICAL THINKING IN THE NURSING PROCESS

1. How would the initial plan of care have changed if Mrs. Williams were not a candidate for fibrinolytic therapy?
2. Two days after her initial therapy, Mrs. Williams complains of palpitations. You notice frequent PVCs on the ECG monitor. What do you do?
3. What health promotion topics would you teach Mrs. Williams before discharge?
4. Mrs. Williams states, "I've been smoking for over 45 years, and I'm not going to stop now! Besides, it calms me down when I'm anxious." How would you respond to this statement?

*See Evaluating Your Response in Appendix C.*

### Assessment

Nursing assessment for the client with AMI must be both timely and ongoing. Assessment data related to AMI include the following:

- **Health history:** Complaints of chest pain, including its location, intensity, character, radiation, and timing; associated symptoms such as nausea, heartburn, shortness of breath, and anxiety; treatment measures taken since onset of pain; past medical history, especially cardiac related; chronic diseases; current medications and any known allergies to medications; smoking history and use of recreational drugs and alcohol.
- **Physical examination:** General appearance including obvious signs of distress; vital signs; peripheral pulses; skin color, temperature, moisture; level of consciousness; heart and breath sounds; cardiac rhythm (on bedside monitor); bowel sounds, abdominal tenderness.

### Nursing Diagnoses and Interventions

Priorities of nursing care include relieving chest pain, reducing cardiac work, and promoting oxygenation. Psychosocial support is especially important, because an acute myocardial infarction can be devastating, bringing the client face to face with his or her own mortality for the first time.

#### Acute Pain

Chest pain occurs when the oxygen supply to the heart muscle does not meet the demand. Myocardial ischemia and infarction cause pain, as does reperfusion of an ischemic area following fibrinolytic therapy or emergent PTCA. Pain stimulates the sympathetic nervous system, increasing cardiac work. Pain relief is a priority of care for the client with AMI.

- Assess for verbal and nonverbal signs of pain. Document characteristics and the intensity of the pain, using a standard pain scale. Verify nonverbal indicators of pain with the client. *Frequent, careful pain assessment allows early intervention to reduce the risk of further damage. Pain is a subjective experience; its expression may vary with location and intensity, previous experiences, and cultural and social background.*

*Pain scales provide an objective tool for measuring pain and a way to assess pain relief or reduction.*

- Administer oxygen at 2 to 5 L/min per nasal cannula. *Supplemental oxygen increases oxygen supply to the myocardium, decreasing ischemia and pain.*
- Promote physical and psychological rest. Provide information and emotional support. *Rest decreases cardiac workload and sympathetic nervous system stimulation, promoting comfort. Information and emotional support help decrease anxiety and provide psychological rest.*
- Titrate intravenous nitroglycerin as ordered to relieve chest pain, maintaining a systolic blood pressure greater than 100 mmHg. *Nitroglycerin decreases chest pain by dilating peripheral vessels, reducing cardiac work, and dilating coronary vessels, including collateral channels, improving blood flow to ischemic tissue.*

#### PRACTICE ALERT

Intravenous nitroglycerin causes peripheral vasodilation, which may lead to hypotension, reduced coronary blood flow, and tachycardia. Reduce the nitro flow rate and notify the physician if this occurs.

- Administer 2 to 4 mg morphine by intravenous push for chest pain as needed. *Morphine is an effective narcotic analgesic for chest pain. It decreases pain and anxiety, acts as a venodilator, and decreases the respiratory rate. The resulting reduction in preload and sympathetic nervous system stimulation reduces cardiac work and oxygen consumption.*

#### PRACTICE ALERT

Reassess for relief of chest pain. The goal of care is to achieve pain relief, not simply a reduction in pain to a "manageable" level.

### Ineffective Tissue Perfusion

Cardiac muscle damage affects compliance, contractility, and cardiac output. The extent of the effect on tissue perfusion depends on the location and amount of damage. Anterior wall infarcts have a greater effect on cardiac output than do right ventricular infarcts. Infarcted muscle also increases the risk for

cardiac dysrhythmias, which can also affect the delivery of blood and oxygen to the tissues.

- Assess and document vital signs. Report increases in heart rate and changes in rhythm, blood pressure, and respiratory rate. *Decreased cardiac output activates compensatory mechanisms that may cause tachycardia and vasoconstriction, increasing cardiac work.*
- Assess for changes in level of consciousness (LOC); decreased urine output; moist, cool, pale, mottled, or cyanotic skin; dusky or cyanotic mucous membranes and nail beds; diminished to absent peripheral pulses; delayed capillary refill. *These are manifestations of impaired tissue perfusion. A change in LOC is often the first manifestation of altered perfusion because brain tissue and cerebral function depend on a continuous supply of oxygen.*
- Auscultate heart and breath sounds. Note abnormal heart sounds (e.g., an S<sub>3</sub> or S<sub>4</sub> gallop or a murmur) or adventitious lung sounds. *Abnormal heart sounds or adventitious lung sounds may indicate impaired cardiac filling or output, increasing the risk for decreased tissue perfusion.*
- Monitor ECG rhythm continuously. *Dysrhythmias can further impair cardiac output and tissue perfusion.*

### PRACTICE ALERT

Obtain a 12-lead ECG to assess complaints of chest pain. Report marked changes to the physician. Continued or unrelieved chest pain may indicate further myocardial ischemia and extension of the infarct; an ECG during episodes of chest pain provides a valuable diagnostic tool to assess myocardial perfusion.

- Monitor oxygen saturation levels. Administer oxygen as ordered. Obtain and assess ABGs as indicated. *Oxygen saturation is an indicator of gas exchange, tissue perfusion, and the effectiveness of oxygen administration. ABGs provide a more precise measurement of blood oxygen levels and allow assessment of acid–base balance.*
- Administer antidysrhythmic medications as needed. *Dysrhythmias affect tissue perfusion by altering cardiac output.*
- Obtain serial CK, isoenzyme, and troponin levels as ordered. *Levels of cardiac markers, CK isoenzymes in particular, correlate with the extent of myocardial damage.*
- Plan for invasive hemodynamic monitoring. *Hemodynamic monitoring facilitates AMI management and treatment evaluation by providing a means of assessing pressures in the systemic and pulmonary arteries, the relationship between oxygen supply and demand, cardiac output, and cardiac index.*

### PRACTICE ALERT

Continuously evaluate the response to interventions such as fibrinolytic therapy, drugs to improve cardiac output and tissue perfusion, and drugs to reduce cardiac work. Adverse effects of therapy may reduce the effectiveness of treatment. Bleeding due to fibrinolytic therapy may affect vascular volume and cardiac output; reperfusion dysrhythmias also may affect cardiac output. Drugs used to improve cardiac output may also increase cardiac work, whereas those given to reduce cardiac work may significantly affect contractility and cardiac output.

## Ineffective Coping

Coping mechanisms help a person deal with a life-threatening event or with acute changes in health. However, certain coping mechanisms may be detrimental to restoring health, particularly if the client relies on them for a prolonged period. Denial, for example, is a common coping mechanism among post-MI clients. In the initial stages, denial can reduce anxiety. Continued denial, however, can interfere with learning and compliance with treatment.

- Establish an environment of caring and trust. Encourage the client to express feelings. *Establishing a trusting nurse–client relationship provides a safe environment for the client to discuss feelings of helplessness, powerlessness, anxiety, and hopelessness. The nurse may then be able to provide additional resources to meet the client’s needs.*
- Accept denial as a coping mechanism, but do not reinforce it. *Denial may initially help by diminishing the psychologic threat to health, decreasing anxiety. However, its prolonged use can interfere with acceptance of reality and cooperation, possibly delaying treatment and hindering recovery.*
- Note aggressive behaviors, hostility, or anger. Document any failure to comply with treatments. *These signs can indicate anxiety and denial.*
- Help the client identify positive coping skills used in the past (e.g., problem-solving skills, verbalization of feelings, asking for help, prayer). Reinforce use of positive coping behaviors. *Coping behaviors that have been successful in the past can help the client deal with the current situation. These familiar methods can decrease feelings of powerlessness.*
- Provide opportunities for the client to make decisions about the plan of care, as possible. *This promotes self-confidence and independence. Participating in care planning gives the client a sense of control and the opportunity to use positive coping skills.*
- Provide privacy for the client and significant other to share their questions and concerns. *Privacy provides an opportunity for the client and partner to share their feelings and fears, offer support and encouragement to one another, relieve anxiety, and establish effective coping methods.*

## Fear

The fear of death and disability can be a paralyzing emotion that adversely affects the client’s recovery from acute myocardial infarction.

- Identify the client’s level of fear, noting verbal and nonverbal signs. *This information enables the nurse to plan appropriate interventions. Clients may not voice concerns; attention to nonverbal indicators is important. Controlling fear helps decrease sympathetic nervous system responses and catecholamine release that may increase feelings of fear and anxiety.*
- Acknowledge the client’s perception of the situation. Allow to verbalize concerns. *A sudden change in health status causes anxiety and fear of the unknown. Verbalizing these fears may help the client cope with change and allow the healthcare team to provide information and correct misconceptions.*
- Encourage questions and provide consistent, factual answers. Repeat information as needed. *Accurate and consistent information can reduce fear. Honest explanations help*

strengthen the client–nurse relationship and help the client develop realistic expectations. Anxiety and fear decrease the ability to concentrate and retain information; therefore, information may need to be repeated.

- Encourage self-care. Allow the client to make decisions regarding the plan of care. *This promotes personal responsibility for health and allows some control over the situation. Clients' confidence increases as their dependence decreases.*
- Administer antianxiety medications as ordered. *These medications promote rest and relaxation and decrease feelings of anxiety, which may act as barriers to health restoration.*
- Teach nonpharmacologic methods of stress reduction (e.g., relaxation techniques, mental imagery, music therapy, breathing exercises, meditation, massage). *Stress management techniques can help reduce tension and anxiety, provide a sense of control, and enhance coping skills.*

## NANDA, NIC, and NOC Linkages

Chart 31–1 shows links between NANDA nursing diagnoses, NIC, and NOC for the client with acute myocardial infarction.

### Community-Based Care

Cardiac rehabilitation begins with admission to the healthcare facility and continues through the inpatient stay and after discharge into the rehabilitative period. The emphasis is on realistic application of information to maintain lifestyle changes.

Assessing readiness to learn is an important first step in preparing for home care. The client in strong denial may not identify any relevance to the information being taught. Evaluate ability to learn, assessing physiologic and psychologic health, beliefs regarding personal responsibility for health, and expectations of the healthcare system. Also assess developmental level, ability to perform psychomotor skills, cognitive function, learning disabilities, existing knowledge base, and the influence of previous learning experiences. Provide written material to supplement teaching and encourage questions.

Include the following topics in teaching for home care:

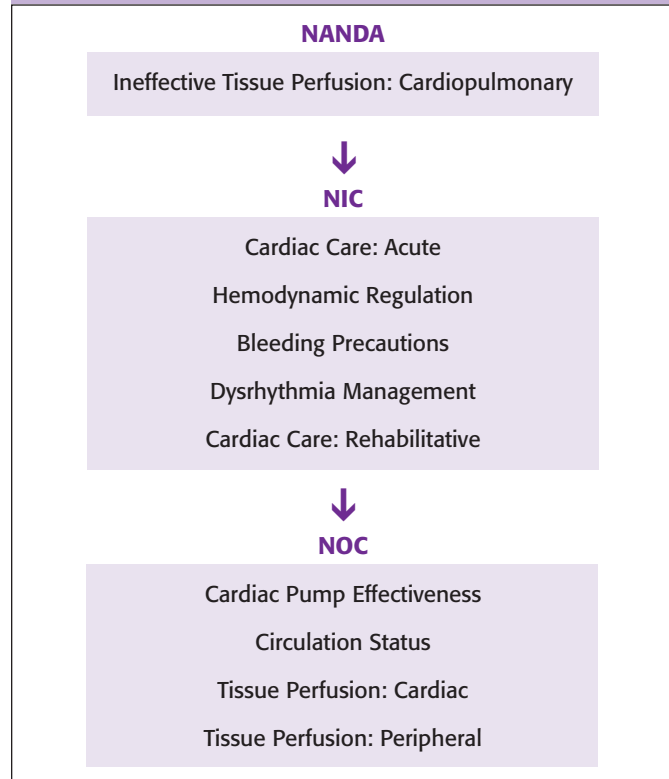
- The normal anatomy and physiology of the heart, and the specific area of heart damage
- The process of CHD and implications of MI
- Purposes and side effects of prescribed medications
- The importance of complying with the medical regimen and cardiac rehabilitation program and of keeping follow-up appointments

## CARDIAC RHYTHM DISORDERS

Heart muscle contracts in response to electrical stimulation. In the normal heart, electrical stimulation produces a synchronized, rhythmic heart muscle contraction that propels blood into the vascular system. Changes in cardiac rhythm affect this synchronized activity and the heart's ability to effectively pump blood to body tissues.

### NANDA, NIC, AND NOC LINKAGES

#### CHART 31–1 The Client with Acute Myocardial Infarction



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.

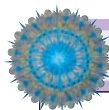
- Information about community resources, such as the local chapter of the American Heart Association.

After discharge, follow up by telephone within 1 week and periodically thereafter during the recovery period. Provide telephone numbers of resource personnel who are available to respond to questions and concerns after discharge. Research demonstrates the value of motivational and social support in adopting healthier behaviors after AMI (see the research box on the next page).

Because the client who has had an MI is at high risk for sudden cardiac death; encourage family members to learn CPR and provide information about community resources for CPR training.

## THE CLIENT WITH A CARDIAC DYSRHYTHMIA

A cardiac dysrhythmia is a disturbance or irregularity in the electrical system of the heart. Cardiac dysrhythmias may be benign or have lethal consequences. Prompt recognition of a lethal dysrhythmia and quick action can be lifesaving.



## NURSING RESEARCH Evidence-Based Practice: Women Following Myocardial Infarction

Coronary heart disease is the leading killer of women in the United States. The risk of death and disability resulting from acute myocardial infarction is higher in women than in men. Although clients often can significantly reduce their risk for subsequent cardiac events by making behavioral changes, the success of efforts to promote these changes has been limited.

In a study of behavioral changes in women following AMI, McSweeney and Coon (2004) identified six inhibitors and four facilitators to change. Financial concerns presented the major barrier to the desired behavior changes, from taking prescribed medications and complying with recommendations for follow-up care, to eating a low-fat diet, exercise, and smoking cessation. Environmental factors and physical problems or symptoms also presented major barriers to dietary changes, exercise, and smoking cessation. Other inhibitors included social support, reduced quality of life, and motivation. Many of the same factors identified as barriers for some women were perceived as enablers of change by others. Social support and financial stability or support were the major factors enabling change, along with motivators and environmental factors.

### IMPLICATIONS FOR NURSING

The nurse needs to look holistically at women when addressing lifestyle changes following AMI. Because the very same factors were perceived as barriers by some women and facilitators by others, it is important for the nurse to work collaboratively with the client to develop successful strategies for behavior change.

Nurses are in a unique position to advocate for the client when medication samples or lower cost alternative drugs can help the client adhere to prescribed treatment, and to suggest referral to cardiac rehabilitation when a structured exercise program will benefit the client (e.g., when the client lives in an area in which she does not feel safe walking). Nurses also often are much more aware of the unique stressors in the woman's life, such as caring for grandchildren, aging parents, or a disabled spouse. Tailoring strategies for change to the individual and his or her circumstances increases the probability of success and continuation of the adopted healthy lifestyle habit.

### CRITICAL THINKING IN CLIENT CARE

1. Why might the same factors (finances, social support, environment, and motivation) be perceived as barriers to changing eating, exercise, and smoking behaviors in some women and as facilitators of change in others?
2. Identify some questions the nurse might ask to help ascertain both barriers to and facilitators of change related to smoking cessation, exercise, and eating habits.
3. How would you intervene when a woman tells you she is not taking the beta blocker and cholesterol-lowering statin prescribed by her physician?
4. Identify some strategies for incorporating daily exercise into the life of a woman who is raising her two pre-teen grandchildren and caring for her aging mother who has Alzheimer's disease.

Source: Adapted from "Women's Inhibitors and Facilitators Associated with Making Behavioral Changes after Myocardial Infarction," by J. C. McSweeney & S. Coon (2004), *Medsurg Nursing*, 13(1), 49–56.

Dysrhythmias develop for many reasons. Not all are pathologic; some alterations in cardiac rhythm occur in response to events such as exercise or fear. For example, a rapid heart rate due to exercise, fever, or excitement is a normal response to the body's demand for oxygen or to stimulation of the sympathetic nervous system. Slow heart rates also may be normal. *Athletic heart syndrome*, which results from long-term training on the heart muscle, allows the heart to beat more slowly and forcefully while maintaining cardiac output and tissue perfusion. Many athletes have a heart rate of less than 60 beats per minute. Aging affects cardiac rhythm as well (see the box on page 996).

Regardless of cause, a dysrhythmia can significantly affect cardiac performance, depending on heart muscle health. The client's response to the dysrhythmia is key in determining the urgency and type of treatment needed.

## Physiology Review

The unique properties of cardiac cells allow effective heart function. Four properties are electrical; the fifth is cardiac muscle's mechanical response to electrical stimulation.

- **Automaticity** is the ability of pacemaker cells to spontaneously initiate an electrical impulse (action potential). The SA node is the dominant pacemaker, generating impulses at 60 to 100 times a minute. Myocardial muscle cells do not possess this ability.

- **Excitability** is the ability of myocardial cells to respond to stimuli generated by pacemaker cells.
- **Conductivity** is the ability to transmit an impulse from cell to cell. When one cell is stimulated, the impulse rapidly spreads throughout the heart muscle.
- **Refractoriness** is the inability of cardiac cells to respond to additional stimuli immediately following depolarization. In the absolute refractory period, depolarization will not occur in response to any stimulus. A stronger than normal stimulus is required to initiate depolarization during the relative refractory period. This is followed by the supernormal period, during which a mild stimulus will cause depolarization.
- **Contractility** is the ability of myocardial fibers to shorten in response to a stimulus. Heart muscle responds in an *all-or-nothing* manner: Stimulation of one muscle fiber causes the entire muscle mass to contract to its fullest extent as one unit.

Electrical activity of the heart is normally controlled by the cardiac conduction system (see Figure 30–7). The sinoatrial (SA) node, the primary pacemaker of the heart, usually generates impulses at a regular rate of 60 to 100 beats/min. The impulse spreads through the atria, is briefly delayed at the AV node, then spreads through conduction pathways of the ventricles and to ventricular muscle. The AV nodal delay allows the

## NURSING CARE OF THE OLDER ADULT

## Cardiac Dysrhythmias

Aging affects the heart and the cardiac conduction system, increasing the incidence of dysrhythmias and conduction defects. Older adults may experience dysrhythmias even when no evidence of heart disease is found.

Older adults have a higher incidence of both ventricular and supraventricular dysrhythmias without detrimental effects than younger people. Ectopic beats, including short runs of ventricular tachycardia, occur more commonly during exercise in older adults. These dysrhythmias do not affect cardiac morbidity or mortality. Fibrosis of the bundle branches can lead to atrioventricular blocks; a prolonged PR interval is common in clients over the age of 65. Older adults also have a higher incidence of diseases that may affect heart rhythm. An elderly client with hyperthyroidism, for example, may present with atrial fibrillation, syncope, and confusion instead of the usual manifestations of goiter, tremor, and exophthalmos.

### Assessing for Home Care

Assessing older adults for problems related to cardiac dysrhythmias focuses on the effect of the dysrhythmia on functional health status.

- Ask about a history of cardiovascular disease and current medications.
- Inquire about symptoms such as episodes of dizziness, lightheadedness, fainting, palpitations, chest pain, or shortness of breath.

- Ask about relationship of symptoms such as palpitations to intake of certain foods and caffeine-containing beverages.
- Evaluate for other contributing factors such as smoking or alcohol intake.
- Inquire about a history of falls, particularly those occurring without apparent reason.

### Teaching for Home Care

Teach measures to reduce the risk of cardiac dysrhythmias and potential adverse consequences of dysrhythmias.

- Emphasize the importance of taking medications as prescribed. Discuss possible effects of over-the-counter medications on the heart.
- Encourage reducing or eliminating caffeine intake. Caffeine increases the risk of ectopic beats and rapid heart rates.
- Encourage participation in a smoking cessation program and to reduce or eliminate alcohol intake if appropriate.
- Encourage engaging in regular exercise. Discuss the beneficial effects of exercise to maintain muscle mass, including cardiac muscle, and cardiovascular health.
- Instruct to contact primary care provider for evaluation of symptoms such as dizziness, fainting, frequent palpitations, shortness of breath, unexplained falls, or chest pain.

atria to contract, delivering an extra bolus of blood to the ventricles before they contract (the **atrial kick**). The AV node also controls the number of impulses that reach the ventricles, preventing extremely rapid heart rates.

## Pathophysiology

Dysrhythmias arise through disruption of the very properties that stimulate and control the heartbeat: automaticity, excitability, conductivity, and refractoriness.

Dysrhythmias due to altered impulse formation include changes in rate and rhythm and the development of ectopic beats. This category includes *tachydysrhythmias* (rapid heart rates), *bradydysrhythmias* (slow heart rates), and ectopic rhythms. These dysrhythmias result from a change in the automaticity of cardiac cells. The rate of impulse formation may abnormally increase or decrease. Aberrant (abnormal) impulses may originate outside normal conduction pathways, causing **ectopic beats**. Ectopic beats interrupt the normal conduction sequence and may not initiate a normal muscle contraction. Depending on the site and timing of abnormal impulses, they may have little effect on the client or pose a significant threat.

Ischemia, injury, and infarction of myocardial tissue affect its excitability and ability to conduct and respond to an electrical stimulus. Conduction abnormalities cause varying degrees of **heart block**, a block in the normal conduction pathways. Myocardial injury or infarction can obstruct or delay impulse conduction. Bundle branch blocks are common in acute myocardial infarction.


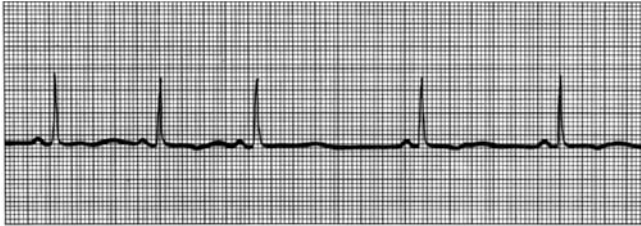
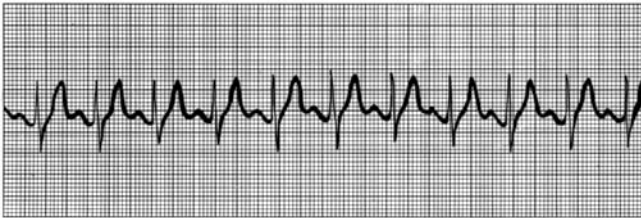
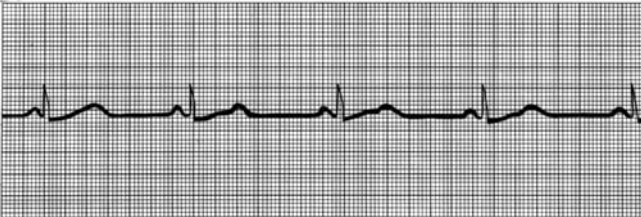

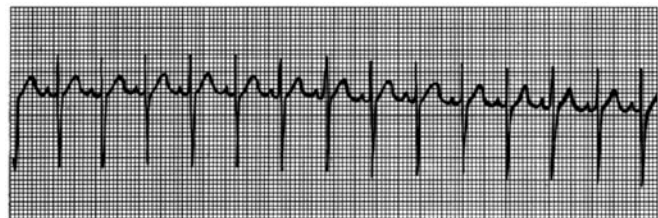
The *reentry phenomenon*, a phenomenon of normal and slow conduction, is a major cause of tachydysrhythmias. A stimulus such as an ectopic beat triggers the reentry phenomenon. The impulse is delayed in one area of the heart (e.g., an area of ischemia or injury) but conducted normally through the rest. Muscle that has been depolarized by the normally conducted impulse is repolarized by the time the impulse traveling through the area of slow conduction reaches it, thus initiating another cycle of depolarization (Porth, 2005). The result is a dysrhythmia that propagates itself.

Several forms of reentry may occur. The impulse may travel through a set pathway to reenter repolarized tissue. Many atrial dysrhythmias follow this pattern, including atrial flutter. In functional reentry, local differences in the conduction of an impulse interrupt the normal wave of depolarization, sending it back upon itself in a spiral pattern and setting up a permanent rotation. This type of pattern suppresses normal pacemaker activity and can lead to atrial fibrillation (Porth, 2005).

Cardiac rhythms are classified according to the site of impulse formation or the site and degree of conduction block. *Supraventricular rhythms* arise above the ventricles. These rhythms usually produce a QRS complex within the normal range. Sinus rhythms, atrial rhythms, and junctional (arising from the AV junction) rhythms are all supraventricular rhythms. *Ventricular rhythms* originate in the ventricles and may prove fatal if left untreated. *AV conduction blocks* result from a defect in impulse transmission from the atria to the ventricles. The major normal and abnormal cardiac rhythms are summarized in Table 31–7.



TABLE 31-7 Characteristics of Selected Cardiac Rhythms and Dysrhythmias

RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
<p><b>Supraventricular Rhythms</b></p> <p><i>Normal sinus rhythm (NSR)</i></p> 	<p>Rate: 60 to 100 beats/min Rhythm: Regular P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec</p>	<p>None; normal heart rhythm.</p>
<p><i>Sinus arrhythmia</i></p> 	<p>Rate: 60 to 100 beats/min Rhythm: Irregular, varying with respirations P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec</p>	<p>Generally none; considered a normal rhythm in the very young and very old.</p>
<p><i>Sinus tachycardia</i></p> 	<p>Rate: 101 to 150 beats/min Rhythm: Regular P:QRS: 1:1 (With very fast rates, P wave may be hidden in preceding T wave) PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec</p>	<p>Treated only if symptomatic or client is at risk for myocardial damage. Treat underlying cause (e.g., hypovolemia, fever, pain). Beta blockers or verapamil may be used.</p>
<p><i>Sinus bradycardia</i></p> 	<p>Rate: &lt; 60 beats/min Rhythm: Regular P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec</p>	<p>Treated only if symptomatic. Intravenous atropine or isoproterenol, and/or pacemaker therapy may be used.</p>
<p><i>Premature atrial contractions (PAC)</i></p> 	<p>Rate: Variable Rhythm: Irregular, with normal rhythm interrupted by early beats arising in the atria P:QRS: 1:1 PR interval: 0.12 to 0.20 sec, but may be prolonged QRS complex: 0.6 to 0.10 sec</p>	<p>Usually require no treatment. Advise to reduce alcohol and caffeine intake, to reduce stress, and to stop smoking. Beta blocker may be prescribed</p>
<p><i>Paroxysmal supraventricular tachycardia (PSVT)</i></p> 	<p>Rate: 100 to 280 beats/min (usually 150 to 200 beats/min) Rhythm: Regular P:QRS: P waves often not identifiable PR interval: Not measured QRS complex: 0.6 to 0.10 sec</p>	<p>Treat if symptomatic. Treatment may include vagal maneuvers (Valsalva, carotid sinus massage); oxygen therapy; adenosine or a beta blocker; temporary pacing, or synchronized cardioversion.</p>

(continued)

TABLE 31-7 Characteristics of Selected Cardiac Rhythms and Dysrhythmias (continued)

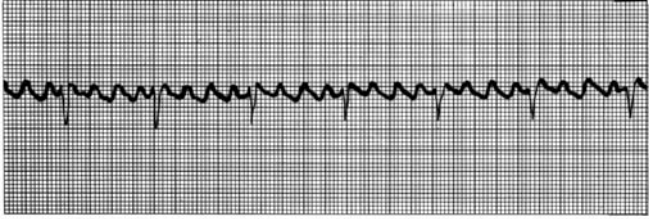

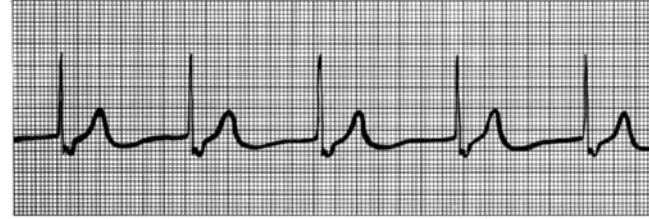

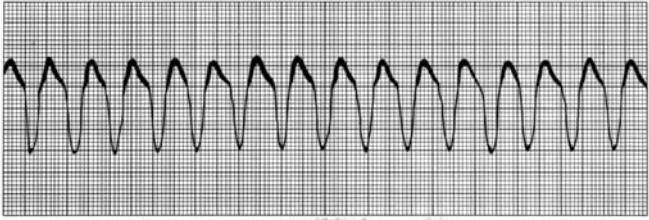


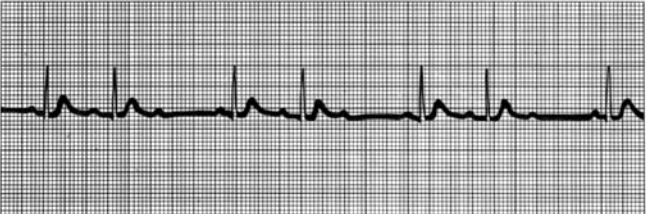
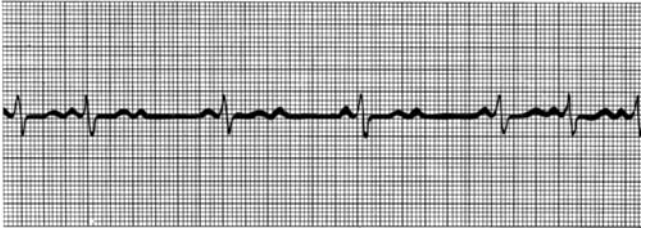

RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
<p data-bbox="115 310 228 338"><i>Atrial flutter</i></p> 	<p data-bbox="805 310 1130 562">Rate: Atrial 240 to 360 beats/min, ventricular rate depends on degree of AV block and usually is &lt;150 beats/min Rhythm: Atrial regular; ventricular usually regular P:QRS: 2:1, 4:1, 6:1; may vary PR interval: Not measured QRS complex: 0.6 to 0.10 sec</p>	<p data-bbox="1159 310 1455 562">Synchronized cardioversion; medications to slow ventricular response such as a beta blocker or calcium channel blocker, followed by a class I antidysrhythmic agent or amiodarone.</p>
<p data-bbox="115 583 261 611"><i>Atrial fibrillation</i></p> 	<p data-bbox="805 583 1130 835">Rate: Atrial 300 to 600 beats/min (too rapid to count); ventricular 100 to 180 beats/min in untreated clients Rhythm: Irregularly irregular P:QRS: Variable PR interval: Not measured QRS complex: 0.06 to 0.10 sec</p>	<p data-bbox="1159 583 1455 835">Synchronized cardioversion; medications to reduce ventricular response rate: metoprolol, diltiazem, or digoxin; anticoagulant therapy to reduce risk of clot formation and stroke.</p>
<p data-bbox="115 856 354 884"><i>Junctional escape rhythm</i></p> 	<p data-bbox="805 856 1130 1108">Rate: 40 to 60 beats/min; junctional tachycardia 60 to 140 BPM Rhythm: Regular P:QRS: P waves may be absent, inverted and immediately preceding or succeeding QRS complex, or hidden in QRS complex PR interval: &lt;0.10 sec QRS complex: 0.06 to 0.10 sec</p>	<p data-bbox="1159 856 1455 1108">Treat cause if symptomatic.</p>
<p data-bbox="115 1150 321 1178"><b>Ventricular Rhythms</b></p> <p data-bbox="115 1182 496 1209"><i>Premature ventricular contractions (PVC)</i></p> 	<p data-bbox="805 1182 1130 1444">Rate: Variable Rhythm: Irregular, with PVC interrupting underlying rhythm and followed by a compensatory pause P:QRS: No P wave noted before PVC PR interval: Absent with PVC QRS complex: Wide (&gt;0.12 sec) and bizarre in appearance; differs from normal QRS complex</p>	<p data-bbox="1159 1182 1455 1444">Treat if symptomatic or in presence of severe heart disease. Advise against stimulant use (caffeine, nicotine). Beta blockers, or class I or III antidysrhythmic agents (see the box on page 1006) may be used in clients with severe heart disease who are symptomatic</p>
<p data-bbox="115 1528 440 1556"><i>Ventricular tachycardia (VT, V tach)</i></p> 	<p data-bbox="805 1528 1130 1780">Rate: 100 to 250 beats/min Rhythm: Regular P:QRS: P waves usually not identifiable PR interval: Not measured QRS complex: 0.12 sec or greater; bizarre shape</p>	<p data-bbox="1159 1528 1455 1780">Treat if VT is sustained, symptomatic, or associated with organic heart disease. Treatment includes DC cardioversion or intravenous procainamide, lidocaine, or a class III antidysrhythmic agent if hemodynamic instability accompanies. Surgical ablation or antitachycardia pacing with an implanted cardioverter/defibrillator (ICD) for repeated episodes.</p>

TABLE 31-7 Characteristics of Selected Cardiac Rhythms and Dysrhythmias (continued)

RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
<p data-bbox="180 306 469 333"><i>Ventricular fibrillation (VF, V fib)</i></p> 	<p data-bbox="867 306 1154 474">Rate: Too rapid to count Rhythm: Grossly irregular P:QRS: No identifiable P waves PR interval: None QRS: Bizarre, varying in shape and direction</p>	<p data-bbox="1222 306 1463 363">Immediate cardioversion/defibrillation.</p>
<p data-bbox="180 579 526 606"><b>Atrioventricular Conduction Blocks</b></p> <p data-bbox="180 611 375 638"><i>First-degree AV block</i></p> 	<p data-bbox="867 611 1187 751">Rate: Usually 60 to 100 beats/min Rhythm: Regular P:QRS: 1:1 PR interval: &gt;0.21 sec QRS complex: 0.06 to 0.10 sec</p>	<p data-bbox="1222 611 1365 638">None required.</p>
<p data-bbox="180 894 691 921"><i>Second-degree AV block, type I (Mobitz I, Wenckebach)</i></p> 	<p data-bbox="867 894 1166 1203">Rate: 60 to 100 beats/min Rhythm: Atrial regular; ventricular irregular P:QRS: 1:1 until P wave blocked with no subsequent QRS complex PR interval: Progressively lengthens in a regular pattern QRS complex: 0.06 to 0.10 sec; sudden absence of QRS complex</p>	<p data-bbox="1222 894 1487 1010">Monitoring and observation; rarely progresses to a higher degree of block or requires treatment.</p>
<p data-bbox="180 1209 574 1236"><i>Second-degree AV block, type II (Mobitz II)</i></p> 	<p data-bbox="867 1209 1175 1465">Rate: Atrial 60 to 100 beats/min; Ventricular &lt;60 beats/min Rhythm: Atrial regular; ventricular irregular P:QRS: Typically 2:1, may vary PR interval: Constant PR interval for each conducted QRS complex QRS complex: 0.06 to 0.10 sec</p>	<p data-bbox="1222 1209 1463 1266">Atropine or isoproterenol; pacemaker therapy.</p>
<p data-bbox="180 1503 607 1530"><i>Third-degree AV block (Complete heart block)</i></p> 	<p data-bbox="867 1503 1179 1839">Rate: Atrial 60 to 100 beats/min; ventricular 15 to 60 beats/min Rhythm: Atrial regular; ventricular regular P:QRS: No relationship between P waves and QRS complexes; independent rhythms PR interval: Not measured QRS complex: 0.06 to 0.10 sec if junctional escape rhythm; &gt;0.12 sec if ventricular escape rhythm</p>	<p data-bbox="1222 1503 1435 1560">Immediate pacemaker therapy.</p>

**FAST FACTS**

- The normal sinus rate is 60 to 100 beats per minute. Each complex includes a P wave, QRS, and T wave.
- Supraventricular dysrhythmias arise in the sinus node or the atria. A P wave may be present; the QRS appears normal, and a T wave may be seen.
- Junctional dysrhythmias arise in tissue just above or just below the AV node. The P wave may be inverted, and may precede, follow, or be buried in the QRS complex. The QRS usually appears normal and is followed by a T wave.
- Ventricular dysrhythmias arise in ventricular myocardium. They do not reset the SA node or activate the atria. QRS complexes are wide and bizarre.

**Supraventricular Rhythms**

**NORMAL SINUS RHYTHM** Normal sinus rhythm (NSR) is the normal heart rhythm, in which impulses originate in the SA (sinus) node and travel through all normal conduction pathways without delay. All waveforms are of normal configuration, look alike, and have consistent (fixed) durations. The rate is between 60 and 100 bpm.

**SINUS NODE DYSRHYTHMIAS** Sinus node dysrhythmias may occur as a normal compensatory response (e.g., to exercise) or because of altered automaticity. In these rhythms, as in NSR, the initiating impulse is from the sinus node. They differ from NSR in rate or regularity of the rhythm. Sinus dysrhythmias include sinus arrhythmia, sinus tachycardia, and sinus bradycardia.

**Sinus Arrhythmia** *Sinus arrhythmia* is a sinus rhythm in which the rate varies with respirations, causing an irregular rhythm. The rate increases during inspiration and decreases with expiration. Sinus arrhythmia is common in the very young and the very old. It can be caused by an increase in vagal tone, by digitalis toxicity, or by morphine administration.

**Sinus Tachycardia** *Sinus tachycardia* has all of the characteristics of NSR, except that the rate is greater than 100 bpm. Tachycardia arises from enhanced automaticity in response to changes in the internal environment. Sympathetic nervous system stimulation or blocked vagal (parasympathetic) activity increases the heart rate. Tachycardia is a normal response to any condition or event that increases the body's demand for oxygen and nutrients, such as exercise or hypoxia. In the client on bed rest, tachycardia is an ominous sign. Sinus tachycardia may be an early sign of cardiac dysfunction, such as heart failure. Tachycardia is detrimental in clients with cardiac disease because it increases cardiac work and oxygen use.

Common causes of sinus tachycardia include exercise, excitement, anxiety, pain, fever, hypoxia, hypovolemia, anemia, hyperthyroidism, myocardial infarction, heart failure, cardiogenic shock, pulmonary embolism, caffeine intake, and certain drugs, such as atropine, epinephrine (Adrenalin), or isoproterenol (Isuprel).

Manifestations of sinus tachycardia include a rapid pulse rate. The client may complain of feeling that the heart is “racing,” shortness of breath, and dizziness. In the presence of heart disease, sinus tachycardia may precipitate chest pain.

**Sinus Bradycardia** *Sinus bradycardia* has all of the characteristics of NSR, but the rate is less than 60 beats/min. Sinus bradycardia may result from increased vagal (parasympathetic) activity or from depressed automaticity due to injury or ischemia to the sinus node. Sinus bradycardia may be normal (e.g., in clients with athletic heart syndrome). The heart rate also normally slows during sleep because the parasympathetic nervous system is dominant at this time. Other causes of sinus bradycardia include pain, increased intracranial pressure, sinus node disease, AMI (especially with inferior wall damage), hypothermia, acidosis, and certain drugs.

Sinus bradycardia may be asymptomatic; it is important to assess the client before treating the rhythm. Manifestations of decreased cardiac output, such as decreased level of consciousness, syncope (faintness), or hypotension, indicate a need for intervention.

**Sick Sinus Syndrome** *Sick sinus syndrome (SSS)* results from sinus node disease or dysfunction that causes problems with impulse formation, transmission, and conduction. Sick sinus syndrome is often found in older adults. It may be caused by direct injury to sinus tissue, fibrosis of conduction fibers associated with aging, and such drugs as digitalis, beta blockers, and calcium channel blockers.

ECG characteristics of SSS include sinus bradycardia, sinus arrhythmia, sinus pauses or arrest, and atrial tachydysrhythmias such as atrial fibrillation, atrial flutter, or atrial tachycardia. Bradycardia-tachycardia syndrome, characterized either by **paroxysmal** (abrupt onset and termination) atrial tachycardia followed by prolonged sinus pauses or alternating periods of bradycardia and tachycardia also may indicate sinus node dysfunction.

Manifestations of sinus node dysfunction often are intermittent, related to a drop in cardiac output caused by the irregular rhythm. Fatigue, dizziness, light-headedness, and syncope are common. The heart rate may not increase in response to stressors such as exercise or fever.

**SUPRAVENTRICULAR DYSRHYTHMIAS** When an action potential originates in atrial tissue outside the sinus node, the resulting rhythm is classified as a *supraventricular rhythm*. In these dysrhythmias, an ectopic pacemaker takes over, or overrides, the SA node. They may also occur when the SA node fails; an *escape rhythm* develops as a fail-safe mechanism to maintain the heart rate. The most common supraventricular dysrhythmias are premature atrial contractions, paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation. These rhythms may be paroxysmal, that is, occur in bursts with an abrupt beginning and end.

**Premature Atrial Contractions** A *premature atrial contraction (PAC)* is an ectopic atrial beat that occurs earlier than the next expected sinus beat. PACs can arise anywhere in the atria. They are usually asymptomatic and benign, but they may initiate paroxysmal supraventricular tachycardia in susceptible individuals. PACs are common in older adults, often occurring without an obvious cause. Strong emotions, excessive alcohol intake, tobacco, and stimulants such as caffeine can precipitate PACs. They also may be associated with myocardial infarction, heart failure and other cardiac disorders, hypoxemia, pulmonary embolism, digi-

talis toxicity, and electrolyte or acid–base imbalances. In clients with underlying heart disease, PACs may precede a more serious dysrhythmia.

The ECG tracing shows interruption of the underlying rhythm by a premature complex that looks similar to the underlying beats. The ectopic impulse of the PAC is usually conducted normally, leading to depolarization of cardiac muscle and a normal QRS complex. Because the impulse arises above the ventricles, it follows normal conduction pathways through the ventricles. The QRS complex is narrow or matches those of the underlying rhythm. The shape of the P wave of a PAC differs from normal P waves because its impulse arises outside the sinus node. A *non-compensatory pause* usually follows, as the PAC resets the SA node rhythm. Occasionally, the ectopic impulse may not be conducted through the heart, resulting in a lone P wave without a QRS, or a nonconducted PAC.

PACs cause few manifestations. If frequent, they may cause palpitations or a fluttering sensation in the chest. Early beats may be noted on auscultating or palpating the pulse.

**Paroxysmal Supraventricular Tachycardia** *Paroxysmal supraventricular tachycardia (PSVT)* is tachycardia of sudden onset and termination. PSVT is usually initiated by a reentry loop in or around the AV node; that is, an impulse reenters the same section of tissue over and over, causing repeated depolarizations.

PSVT occurs more frequently in women. Sympathetic nervous system stimulation and stressors such as fever, sepsis, and hyperthyroidism may precipitate PSVT. It also may be associated with heart diseases such as CHD, myocardial infarction, rheumatic heart disease, myocarditis, or acute pericarditis. Abnormal conduction pathways associated with Wolff-Parkinson-White (WPW) syndrome may account for PSVT.

PSVT affects ventricular filling and cardiac output, and decreases coronary artery perfusion. Its manifestations include complaints of palpitations and a “racing” heart, anxiety, dizziness, dyspnea, anginal pain, diaphoresis, extreme fatigue, and polyuria (urine output may reach up to 3 L in the first few hours after PSVT onset).

**Atrial Flutter** *Atrial flutter* is a rapid and regular atrial rhythm thought to result from an intra-atrial reentry mechanism. Causes include sympathetic nervous system stimulation due to anxiety or caffeine and alcohol intake; thyrotoxicosis; coronary heart disease or myocardial infarction; pulmonary embolism; and abnormal conduction syndromes, such as WPW syndrome. Older persons with rheumatic heart disease and/or valvular disease are especially vulnerable.

Two types of atrial flutter have been identified. Type I atrial flutter has an atrial rate of 240 to 340 beats per minute. It develops due to a reentry mechanism in the right atrium. The mechanism leading to type II atrial flutter has not been identified. In this type of flutter, the atrial rate is faster, to 350 beats per minute.

Clients with atrial flutter may complain of palpitations or a fluttering sensation in the chest or throat. If the ventricular rate is rapid, manifestations of decreased cardiac output, such as decreased level of consciousness, hypotension, decreased urinary output, and cool clammy skin, may be noted. The atrial kick

(additional ventricular filling with atrial contraction) is lost because of inadequate atrial filling.

ECG characteristics include a “sawtooth” or “picket fence” appearance of P waves, which are labeled flutter (F) waves. The atrial rate is rapid, often around 300 beats/min. As a protective mechanism, many impulses are blocked at the AV node, and the ventricular rate is rarely greater than 150 to 170 beats/min. Usually, atrial impulses are evenly conducted through the AV node, for example, two impulses to one QRS complex (2:1), four impulses to one QRS complex (4:1), or six impulses to one QRS complex (6:1). A constant conduction ratio results in a regular ventricular rhythm; the ventricular rhythm is irregular if the conduction ratio varies. The ventricular rate usually ranges from 150 to 170 beats/min in 2:1 conduction and 60 to 75 beats/min for lower conduction ratios. The T wave is usually hidden by overriding F waves; some F waves may be hidden in the QRS complex.

**Atrial Fibrillation** *Atrial fibrillation* is a common dysrhythmia characterized by disorganized atrial activity without discrete atrial contractions. Multiple small reentry circuits develop in the atria. Atrial cells cannot repolarize in time to respond to the next stimulus (Porth, 2005). Extremely rapid atrial impulses bombard the AV node, resulting in an irregularly irregular ventricular response. Atrial fibrillation may occur suddenly and recur, or it may persist as a chronic dysrhythmia. Atrial fibrillation is commonly associated with heart failure, rheumatic heart disease, coronary heart disease, hypertension, and hyperthyroidism.

Manifestations of atrial fibrillation relate to the rate of the ventricular response. With rapid response rates, manifestations of decreased cardiac output such as hypotension, shortness of breath, fatigue, and angina may develop. Clients with extensive heart disease may develop syncope or heart failure. Peripheral pulses are irregular and of variable amplitude (strength).

The specific ECG characteristics of atrial fibrillation include an irregularly irregular rhythm and the absence of identifiable P waves. The atrial rate is so rapid that it is not measurable. The ventricular rate varies.

Atrial fibrillation increases the risk for formation of thromboemboli. Organ infarction may occur as a result; the incidence of stroke is high.

### Junctional Dysrhythmias

Rhythms that originate in AV nodal tissue are termed *junctional*. The AV junction includes the AV node and the bundle of His, which branches into the right and left bundle branches. An impulse arising from the AV junction may occur in response to failure of higher pacemakers, as in a *junctional escape rhythm*, or it may result from an abnormal mechanism, such as altered automaticity. An impulse arising from the AV junction may or may not be conducted back up to the atria. This conduction against the normal flow or pattern is called *retrograde conduction*. The resulting atrial wave, called a P' wave, may be found before, during, or after the QRS complex, depending on the speed of conduction. The P' wave is inverted in some ECG leads because the impulse moves from the AV node up to the atria instead of from the SA node down toward

the AV node. In addition, the P'R interval is shorter than normal (less than 0.12 sec). The QRS complex is typically narrow.

A junctional rhythm may be due to drug toxicity (e.g., digitalis, beta blockers, or calcium channel blockers) or other causes such as hypoxemia, hyperkalemia, increased vagal tone or damage to the AV node, myocardial infarction, and heart failure. Loss of synchronized atrial contraction and the atrial kick may affect cardiac output, leading to manifestations of decreased cardiac output and impaired myocardial tissue perfusion. Heart failure may develop.

*Premature junctional contractions (PJC)s* occur before the next expected beat of the underlying rhythm. Isolated PJC's may occur in healthy people and are insignificant. *Junctional tachycardia* is a junctional rhythm with a rate greater than 60 beats/min. It is caused by increased automaticity of AV nodal tissue. The ventricular rate is usually less than 140 beats/min. Both rhythms are most commonly associated with digitalis toxicity, hypoxia, ischemia, or electrolyte imbalances.

### Ventricular Dysrhythmias

Ventricular dysrhythmias originate in the ventricles. Because the ventricles pump blood into the pulmonary and systemic vasculature, any disruption of their rhythm can affect cardiac output and tissue perfusion. A wide and bizarre QRS complex (greater than 0.12 sec) is a characteristic feature of ventricular dysrhythmias. This occurs because ventricular ectopic impulses begin and travel outside normal conduction pathways. Other characteristics include no relationship of the QRS complex to a P wave, increased amplitude of the QRS complex, an abnormal ST segment, and a T wave deflected in the opposite direction from the QRS complex.

**PREMATURE VENTRICULAR CONTRACTIONS** *Premature ventricular contractions (PVCs)* are ectopic ventricular beats that occur before the next expected beat of the underlying rhythm. They usually do not reset the atrial rhythm and are followed by a full compensatory pause. PVCs often have no significance in people without heart disease. Frequent, recurrent, or multifocal PVCs may be associated with an increased risk for lethal dysrhythmias. PVCs result from either enhanced automaticity or a reentry phenomenon. They may be triggered by anxiety or stress; tobacco, alcohol, or caffeine use; hypoxia, acidosis, and electrolyte imbalances; sympathomimetic drugs; coronary heart disease; heart failure; and mechanical stimulation of the heart (e.g., the insertion of a cardiac catheter); or reperfusion after fibrinolytic therapy. The incidence and significance of PVCs is greatest after myocardial infarction.

PVCs may be isolated or occur in a specific pattern. Two PVCs in a row are called a *couplet* or *paired* PVCs. Three consecutive PVCs (a *triplet* or *salvo*) is a short run of ventricular tachycardia. *Ventricular bigeminy* is characterized by a PVC following each normal beat; a PVC noted every third beat is called *ventricular trigeminy*. When the ventricular impulse arises from one ectopic site, all PVCs look the same (*monomorphic*) and are called *unifocal* PVCs. *Multifocal* PVCs arise from different ectopic sites and appear different from one another on the ECG (*polymorphic*).

The frequency and patterns of PVCs can be indicative of myocardial irritability and the risk for a lethal dysrhythmia.

The following are considered warning signs in the client with acute heart disease (e.g., an acute MI).

- PVCs that develop within the first 4 hours of an MI
- Frequent PVCs (six or more per minute)
- Couplets or triplets
- Multifocal PVCs
- R-on-T phenomenon (PVCs falling on the T wave).

In people without heart disease, isolated PVCs usually are insignificant and do not require treatment. Clients may complain of feeling their hearts “skip a beat” or of palpitations. In clients with preexisting heart disease, PVCs may indicate a drug toxicity or an increased risk for lethal dysrhythmias and cardiac arrest. The risk is greatest following acute MI.

**VENTRICULAR TACHYCARDIA** *Ventricular tachycardia (VT, V tach)* is a rapid ventricular rhythm defined as three or more consecutive PVCs. Ventricular tachycardia may occur in short bursts, or “runs,” or may persist for more than 30 seconds (sustained ventricular tachycardia). The rate is greater than 100 beats/min, and the rhythm is usually regular. Reentry is the usual electrophysiologic mechanism responsible for VT. Myocardial ischemia and infarction are the most common predisposing factors for VT. It also is associated with cardiac structural disorders such as valvular disease, rheumatic heart disease, or cardiomyopathy. It may occur in the absence of heart disease, and with anorexia nervosa, metabolic disorders, and drug toxicity.

Nonsustained VT may occur paroxysmally and convert back to an effective rhythm spontaneously. The client may experience a fluttering sensation in the chest or complain of palpitations and brief shortness of breath. Clients in sustained VT generally develop signs and symptoms of decreased cardiac output and hemodynamic instability, including severe hypotension, a weak or nonpalpable pulse, and loss of consciousness. Allowed to continue, VT can deteriorate into ventricular fibrillation. Sustained ventricular tachycardia is a medical emergency that requires immediate intervention, particularly in clients with cardiac disease.

*Toursades de pointes* is a type of ventricular tachycardia associated with *long QT syndrome*, a prolongation of the QT interval. Long QT syndrome may be genetic or acquired, occurring secondarily to electrolyte disruptions, myocardial infarction, cocaine use, liquid protein diets, medications, or other conditions. In *toursades de pointes*, the QRS complexes vary in size, shape, and amplitude (Figure 31–7 ■). Clients with *toursades de pointes* may have multiple bursts or episodes of ventricular tachycardia or may develop ventricular fibrillation and sudden cardiac death (Kasper et al., 2005; Porth, 2005).

**VENTRICULAR FIBRILLATION** *Ventricular fibrillation (VF, V fib)* is extremely rapid, chaotic ventricular depolarization causing the ventricles to quiver and cease contracting; the heart does not pump. This is known as **cardiac arrest**; it is a medical emergency requiring immediate intervention with cardiopulmonary resuscitation (CPR). Death will follow the onset of VF within 4 minutes if the rhythm is not recognized and terminated and an effective perfusing rhythm reestablished.

Ventricular fibrillation is usually triggered by severe myocardial ischemia or infarction. It occurs without warning 50%



**Figure 31-7** ■ Torsades de pointes. Note the wide and bizarre QRS complexes of varying size, shape (morphology), and amplitude.

of the time. It is the terminal event in many disease processes or traumatic conditions. Ventricular fibrillation may be precipitated by a single PVC or may follow VT. Other causes of VF include digitalis toxicity, reperfusion therapy, antidysrhythmic drugs, hypokalemia and hyperkalemia, hypothermia, metabolic acidosis, mechanical stimulation (as with the insertion of cardiac catheters or pacing wires), and electric shock.

Clinically, loss of ventricular contractions results in absence of a palpable or audible pulse. The client loses consciousness and stops breathing as perfusion ceases. The ECG shows grossly irregular, bizarre complexes with no discernible rate or rhythm.

### Atrioventricular Conduction Blocks

Conduction defects that delay or block transmission of the sinus impulse through the AV node are called *atrioventricular conduction blocks*. Impaired conduction may result from tissue injury or disease, increased vagal (parasympathetic) tone, drug effects, or a congenital defect. AV conduction blocks vary in severity from benign to severe.

#### FAST FACTS

- First-degree AV block = delayed conduction through the AV node and a long PR interval.
- Second-degree AV block = complete blockage of *some* impulses through the AV node; some P waves are not followed by a QRS complex.
- Third-degree AV block = complete blockage of *all* impulses through the AV node; no relationship between P waves and QRS complexes.

**FIRST-DEGREE AV BLOCK** *First-degree AV block* is a benign conduction delay that generally poses no threat, has no symptoms, and requires no treatment. Impulse conduction through the AV node is slowed, but all atrial impulses are conducted to the ventricles. It may result from injury or infarct of the AV node, other cardiac diseases, or drug effects. The ECG shows all characteristics of NSR, except the PR interval is greater than 0.20 second.

**SECOND-DEGREE AV BLOCK** *Second-degree AV block* is characterized by failure to conduct one or more impulses from the atria to the ventricles. Two patterns of second-degree AV block are seen, identified as type I and type II.

**Second-Degree AV Block—Type I** *Type I second-degree AV block (Mobitz type I or Wenckebach phenomenon)* is characterized by a repeating pattern of increasing AV conduction delays until an impulse fails to conduct to the ventricles. On the ECG, PR intervals progressively lengthen until one QRS complex is not conducted, or dropped. The ventricular rate remains adequate to maintain cardiac output, and the client usually is asymptomatic. Mobitz type I AV block usually is transient, associated with acute MI or drug intoxication (e.g., digitalis, beta blockers, or calcium channel blockers). It rarely progresses to complete heart block.

**Second-Degree AV Block—Type II** *Type II second-degree AV block (Mobitz type II)* involves intermittent failure of the AV node to conduct an impulse to the ventricles without preceding delays in conduction. The PR interval remains constant, but not all P waves are followed by QRS complexes (e.g., there may be two P waves for every QRS). Conduction through the His-Purkinje system usually is delayed as well, causing a widened QRS complex (Braunwald et al., 2002). Mobitz type II block is frequently associated with acute anterior wall MI and a high rate of mortality (Porth, 2005). Manifestations of Mobitz type II block depend on the ventricular rate. Pacemaker therapy may be required to maintain the cardiac output.

**THIRD-DEGREE AV BLOCK** *Third-degree AV block (complete heart block)* occurs when atrial impulses are completely blocked at the AV node, and fail to reach the ventricles. As a result, the atria and ventricles are controlled by different and independent pacemakers, with separate rates and rhythms. The ventricular impulse arises from either junctional fibers (with a rate of 40 to 60 beats/min) or a ventricular pacemaker at a rate of less than 40 beats/min. The width of the QRS complex depends on the location of the escape pacemaker. The QRS is wide and the rate is slow when the rhythm arises distal to the bundle of His.

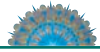
Third-degree block is frequently associated with an inferior or anteroseptal myocardial infarction. Other causes include congenital conditions, acute or degenerative cardiac disease or damage, drug effects, and electrolyte imbalances. The slow escape rhythm significantly affects cardiac output, causing manifestations such as syncope (known as a *Stokes-Adams attack*), dizziness, fatigue, exercise intolerance, and heart failure. Third-degree AV block is life threatening and requires immediate intervention to maintain adequate cardiac output.

**AV DISSOCIATION** Complete dissociation of atrial and ventricular rhythms can occur in conditions other than third-degree AV block. The two primary factors leading to AV dissociation are severe sinus bradycardia and a lower pacemaker (junctional or ventricular) that competes with or exceeds the normal sinus rhythm (Braunwald et al., 2002). AV dissociation may result from acute myocardial ischemia or infarction, cardiac surgery, or drug effects. The ECG shows separate and competing atrial (P waves) and ventricular (QRS complexes) rhythms.

## Intraventricular Conduction Blocks

Once the impulse enters the ventricles, its conduction through the right and left bundle branches may be impaired (*bundle branch block*). As a result, the impulse is conducted more slowly than normal through the ventricles. On the ECG, the QRS complex is prolonged. Its appearance varies, depending on the affected bundle (right or left). Typically, no clinical manifestations are associated with bundle branch block unless it occurs in conjunction with an AV block.


## INTERDISCIPLINARY CARE



Cardiac dysrhythmias may be either benign or critical: Recognizing lethal dysrhythmias is a matter of life and death. Major goals of care include identifying the dysrhythmia, evaluating its effect on physical and psychosocial well-being, and treating underlying causes. This may involve correcting fluid and electrolyte or acid–base imbalances; treating hypoxia, pain, or anxiety; administering antidysrhythmic medications; or mechanical and surgical interventions.

### Diagnosis

Diagnostic tests for dysrhythmias include the electrocardiogram, cardiac monitoring, and electrophysiology studies. Laboratory tests such as serum electrolytes, drug levels, and ABGs may be done to help identify the cause of the dysrhythmia.

**ELECTROCARDIOGRAM** The 12-lead ECG may be required to accurately diagnose a dysrhythmia. It also provides information about underlying disease processes, such as myocardial infarction or other cardiac disease. The ECG may also be used to monitor the effects of treatment. See Chapter 30  for more information about the 12-lead ECG.

**CARDIAC MONITORING** Cardiac monitoring allows continuous observation of the cardiac rhythm. It is used in many different circumstances (Box 31–4). Different types of ECG monitoring are employed for different situations.

**Continuous Cardiac Monitoring** Continuous monitoring of the cardiac rhythm is provided by bedside and central monitoring stations. Electrodes placed on the client’s chest attach to cables connected to a monitor. The heart rate and rhythm is visually displayed on a bedside monitor connected to a central monitoring station. The central station allows simultaneous monitoring of multiple clients within a nursing unit. Alarms on both bedside and central monitors warn of potential problems such as very rapid or very slow heart rates. Alarm limits are preset by the nurse for the individual client. Procedure 31–1 describes how to place a client on cardiac monitoring.

*Telemetry* may be used in acute care settings when the client is ambulatory. Chest electrodes are connected to a portable transmitter worn around the neck or waist; the ECG is transmitted electronically to a central monitoring station for continuous monitoring.

**Home Monitoring** Clients often complain of palpitations or other heart symptoms but are asymptomatic during evaluation in a hospital or community-based setting. Ambulatory or Holter monitoring may be used to identify intermittent dys-

### BOX 31–4 Indications for Cardiac Monitoring

- Perioperative monitoring of heart rate and rhythm
- Detecting and identifying dysrhythmias
- Monitoring the effects of cardiac and noncardiac diseases on the heart
- Monitoring clients with potentially life-threatening conditions:
  - a. Major trauma (especially cardiac trauma)
  - b. Dissecting aneurysm
  - c. Acute myocardial infarction
  - d. Heart failure
  - e. Shock
  - f. Other emergency conditions
- Evaluating responses to procedures and interventions:
  - a. Drug therapies
  - b. Diagnostic procedures
  - c. Ablative techniques
  - d. Angioplasty or cardiac catheterization
  - e. Cardiac surgery
  - f. Pacemaker function
  - g. Automatic implantable cardioverter-defibrillator function

rhythmias, to detect silent ischemia, to monitor the effects of treatment, and to assess pacemaker or automatic cardioverter-defibrillator function. Electrodes are applied and the leads attached to the portable telemetry monitor that records and stores all electrical activity. Clients are instructed to leave the electrode pads in place during monitoring, record any cardiac symptoms or events in a journal (such as chest pain, palpitations, syncope), and are told when to return to the clinic. After the prescribed period, usually 48 to 72 hours, the client returns and the monitor is removed. Diary entries are compared to the recorded heart rhythms to identify the effects of dysrhythmias.

**ELECTROPHYSIOLOGY STUDIES** Diagnostic cardiac *electrophysiology (EP) procedures* are used to identify dysrhythmias and their causes. EP studies are used to analyze components of the conduction system, identify sites of ectopic stimulation, and evaluate the effectiveness of treatment. EP procedures can be used for both diagnosis and as a therapeutic intervention.

In the electrophysiology laboratory, electrode catheters are guided by fluoroscopy into the heart through the femoral or brachial vein. The timing and sequence of electrical activation during normal and abnormal (aberrant) rhythms is observed and measured. Electrical stimulation may be used to induce dysrhythmias similar to the client’s clinical dysrhythmia (Woods et al., 2004). Following diagnosis, an EP procedure may be used to treat the dysrhythmia, for example, by overdrive pacing (stimulating the client’s heart rate to a rate faster than that of the tachydysrhythmia) to break the dysrhythmia’s cycle, or to perform ablative therapy to destroy the ectopic site. See the section on ablative techniques for further information.

Nursing care for the client undergoing an EP procedure is similar to that for a percutaneous coronary revascularization (see the box on page 978). The procedure and expected sensations are explained. The client remains awake during the procedure; antianxiety medications or sedatives are given to reduce



## PROCEDURE 31-1 INITIATING CARDIAC MONITORING



### GATHER SUPPLIES

- Bedside monitor and cable or telemetry unit with fresh battery
- Electrodes—self-adherent, pregelled, disposable
- Lead wires
- Washcloth, soap, and towel
- Alcohol prep pads
- Dry gauze pads or ECG prep pads

### BEFORE THE PROCEDURE

Explain the reason for ECG monitoring. Reassure client that changes in heart rhythm can be noted and immediately treated if necessary. Explain that loose or disconnected lead wires, poor electrode contact, excessive movement, electrical interference, or equipment malfunc-

tion may trigger alarms and alert the staff, allowing correction of the problem. Reassure that movement is allowed, within activity restrictions, while on the monitor. Explain skin preparation procedure. Provide for privacy, and drape appropriately.

### PROCEDURE

1. Follow standard precautions.
2. Check equipment for damage (i.e., fraying, bent, or broken wires). Connect lead wires to cable, and secure connections.
3. Select electrode sites on the chest wall, avoiding areas of excessive movement, joints, skin creases, scar tissue, or other lesions.
4. Clean sites with soap and water, and dry thoroughly. Alcohol may be used to remove skin oils; allow the skin to dry for 60 seconds after use.
5. Gently rub the site with a dry gauze pad or ECG prep pad to remove dead skin cells, debris, and residue.
6. Open the electrode package; peel the backing from the electrode, and check to ensure that the center of the pad is moist with conductive gel.
7. Apply electrode pads, pressing firmly to ensure contact (see figure).
8. Attach leads and position cable with sufficient slack for comfort. Place the telemetry unit (if used) in gown pouch or pocket.
9. Assess ECG tracing on the monitor, adjusting settings as needed.
10. Set monitor alarm limits typically at 20 bpm higher and lower than the client's baseline rate. Turn alarms on, and leave on at all times. Assess immediately if an alarm is triggered.
11. Time and date pads with every change.



### AFTER THE PROCEDURE

Monitor periodically for comfort. Assess electrode and lead wire connections as needed. Remove and apply new pads every 24 to 48 hours or whenever the pad becomes dislodged or nonadherent. Clean gel residue from previous site, and document skin condition under the pads. Choose an alternative site if the skin appears irritated or blistered. Document ECG strips according to

unit policy and/or physician's order, as well when the cardiac rhythm or the client's condition changes (especially with complaints of chest pain, decreased level of consciousness, or changes in vital signs). Note the date, time, client identification, monitor lead, duration of PR and QT intervals, and rhythm interpretation on each ECG strip.

apprehension. Intravenous heparin may be given during the procedure to reduce the risk of thromboembolism.

Complications of EP procedures are infrequent, but include fatal ventricular fibrillation, cardiac perforation, and major venous thrombosis (Woods et al., 2004). Careful postprocedure monitoring is vital.

### Medications

The goal of drug therapy is to suppress dysrhythmia formation. No drug has been found to be completely safe and effective. Antidysrhythmic drugs are primarily used for acute treatment of dysrhythmias, although they may also be used to manage

chronic conditions. The overall goal of therapy is to maintain an effective cardiac output by stabilizing cardiac rhythm.

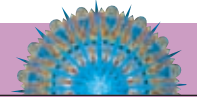
It is important to remember that virtually all antidysrhythmic drugs also have *prodyrhythmic* effects; that is, they can worsen existing dysrhythmias and precipitate new ones. Because of this tendency, studies that demonstrate higher mortality rates in clients receiving antidysrhythmic medications, and the increasing safety and availability of interventional techniques, antidysrhythmic medications are used sparingly.

Most antidysrhythmic drugs are classified by their effects on the cardiac action potential. Most are class I drugs, or fast sodium channel blockers. By blocking sodium channels, these

drugs slow impulse conduction in the atria and ventricles. This class is further divided into subclasses A, B, and C. Class II drugs are beta blockers, which decrease SA node automaticity, AV conduction velocity, and myocardial contractility. Class III agents block potassium channels, delaying repolarization and prolonging the relative refractory period. Class IV drugs are calcium channel blockers. Their effect is similar to that of beta blockers. Adenosine and digoxin do not fit within the major classes. Both drugs reduce SA node automaticity and slow AV

conduction. Ibutilide and magnesium also fall outside the major classes, but are used to treat dysrhythmias. The Medication Administration box below identifies common antidysrhythmic drugs within each class and the nursing implications in caring for clients receiving these drugs.

Drugs that affect the autonomic nervous system may also be used to treat dysrhythmias. Sympathomimetics, such as epinephrine, stimulate the heart, increasing both heart rate and contractility. Anticholinergic agents such as atropine are used



## MEDICATION ADMINISTRATION Antidysrhythmic Drugs

### CLASS I DRUGS: SODIUM CHANNEL BLOCKERS

#### Class IA

**Quinidine (Cardioquin, Quinidex, Quinaglute)**

**Procainamide (Pronestyl, Procan SR)**

**Disopyramide (Norpace, Norpace CR)**

**Moricizine (Ethmozine)**

Class IA drugs decrease the flow of sodium into the cell and prolong the action potential. This decreases automaticity, slows the rate of impulse conduction, and prolongs refractoriness. They are used to treat both supraventricular and ventricular tachycardias.

#### Class IB

**Lidocaine (Xylocaine)**

**Tocainide (Tonocard)**

**Mexiletine (Mexitil)**

**Phenytoin (Dilantin)**

Class IB, or lidocaine-like, drugs decrease the refractory period but have little effect on automaticity. Drugs in this class are used primarily to treat ventricular dysrhythmias, including PVCs and ventricular tachycardia.

#### Class IC

**Flecainide (Tambocor)**

**Propafenone (Rythmol)**

Class IC drugs slow impulse conduction velocity but have little effect on refractoriness. They are used to reduce or eliminate tachydysrhythmias associated with reentry. Their significant prodysrhythmic effects limit their usefulness, but they may be used to treat supraventricular tachycardia.

### CLASS II DRUGS: BETA-BLOCKERS

**Esmolol (Brevibloc)**

**Propranolol (Inderal)**

**Metoprolol (Toprol)**

Class II drugs are beta blockers that decrease automaticity and conduction through the AV node. They also reduce the heart rate and myocardial contractility. They are used to treat supraventricular tachycardia and to slow the ventricular response rate to atrial fibrillation. These drugs may cause bronchospasm and are contraindicated for clients with asthma, chronic obstructive pulmonary disease (COPD), or other restrictive or obstructive lung diseases.

### CLASS III DRUGS: POTASSIUM CHANNEL BLOCKERS

**Sotalol (Betapace)**

**Bretylum (Bretylol)**

**Amiodarone (Cordarone)**

**Ibutilide (Corvert)**

**Dofetilide (Tikosyn)**

Class III drugs block potassium channels, prolonging repolarization and the refractory period. Drugs in this class are used primarily to treat ventricular tachycardia and ventricular fibrillation. Amiodarone may also be used for supraventricular tachycardias.

### CLASS IV DRUGS: CALCIUM CHANNEL BLOCKERS

**Verapamil (Calan, Isoptin, Verelan)**

**Diltiazem (Cardizem, Dilacor XR)**

Calcium channel blockers decrease automaticity and AV nodal conduction. They are used to manage supraventricular tachycardias. Like the beta blockers, calcium channel blockers reduce myocardial contractility.

#### OTHER DRUGS

**Adenosine (Adenocard) Digoxin**

Adenosine and digoxin decrease conduction through the AV node and are used to treat supraventricular tachycardias.

#### Nursing Responsibilities

- Obtain baseline data including vital signs, cardiac rhythm (including rate, PR and QT intervals, and QRS duration), and physical assessment (especially cardiac, neurologic, and respiratory status).
- Assess medication regimen to identify drugs that may interfere with antidysrhythmic therapy.
- Monitor ECG to evaluate the effectiveness of therapy and to assess for possible dysrhythmias precipitated by treatment.
- Immediately report manifestations of drug toxicity:
  - Procainamide—signs of heart failure; conduction delays or ventricular dysrhythmias; skin rash, myalgias or arthralgias, flulike symptoms.
  - Disopyramide—urinary retention, heart failure, eye pain
  - Lidocaine—changes in neurologic status, such as agitation, confusion, dizziness, nervousness
  - Amiodarone—pulmonary fibrosis (increasing dyspnea, cough, hepatic dysfunction—changes in liver function tests, jaundice); vision changes, photosensitivity
  - Digoxin—anorexia, nausea, vomiting; blurred or double vision; yellow green halos; new-onset dysrhythmias
- Use an infusion pump to administer intravenous infusions. Monitor the dose and assess its appropriateness (in mg/min or mcg/kg/min).

#### Health Education for the Client and Family

- Take the drug exactly as prescribed. Do not skip or double doses. Check with your physician if a dose is missed.
- Take your pulse and record the rate daily before rising. Count the pulse for 1 full minute. Bring the record with you to each office or clinic visit
- Report the following to the physician: irregular pulse rate or rhythm, dizziness, eye pain, changes in vision, skin rashes or color changes, wheezing or other respiratory problems, changes in behavior.

to decrease vagal tone and increase the heart rate. Magnesium sulfate is an unclassified drug that has been shown to be safe and effective in treating ventricular tachycardias.

### Countershock

*Countershock* is used to interrupt cardiac rhythms that compromise cardiac output and the client's welfare. Delivery of a direct current charge depolarizes all cardiac cells at the same time. This simultaneous depolarization may stop a tachydysrhythmia and allow the sinus node to recover control of impulse formation. There are two types of countershock: synchronized cardioversion and defibrillation.

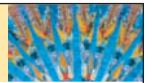
**SYNCHRONIZED CARDIOVERSION** *Synchronized cardioversion* delivers direct electrical current synchronized with the client's heart rhythm. Synchronization of the shock with the QRS complex prevents ventricular fibrillation by avoiding current delivery during the vulnerable period of repolarization.

Cardioversion is usually done as an elective procedure to treat supraventricular tachycardia, atrial fibrillation, atrial flutter, or hemodynamically stable ventricular tachycardia.

The nurse assists with cardioversion by preparing the client before the procedure; obtaining any laboratory tests ordered; obtaining and documenting ECG strips prior to, during, and after treatment; setting up the equipment; and monitoring the client's response. Procedure 31–2 describes synchronized cardioversion.

Clients in atrial fibrillation are at high risk for thromboembolism following cardioversion. Loss of atrial contractions with atrial fibrillation leads to blood pooling in the atria, increasing the risk of clot formation. When the atria begin to contract following successful cardioversion, clots may be dislodged, embolizing to the pulmonary or systemic circulation. If possible, anticoagulants are given for several weeks before cardioversion is attempted.

## PROCEDURE 31–2 ELECTIVE SYNCHRONIZED CARDIOVERSION



### GATHER SUPPLIES

- Cardioverter-defibrillator with ECG cable and monitor
- Conductive gel pads or paste
- Dry gauze pads
- Emergency drug kit and resuscitation equipment
- IV supplies (catheter, solution, administration set)

### BEFORE THE PROCEDURE

Explain the purpose of the procedure (to restore an effective cardiac rhythm). Describe the procedure in simple, nonthreatening terms. Advise that some discomfort may be felt with each countershock, but a sedative will be given to minimize discomfort. Witness the signature on an informed consent form for this procedure. Document preprocedure rhythm on an ECG strip. Ensure a patent intravenous access site for emergency drug administration. Keep NPO as specified prior

to the procedure. Assess acid-base and electrolyte levels (especially potassium, magnesium, and calcium) and drug levels if appropriate. Report abnormalities to the physician prior to the procedure. Document vital signs, level of consciousness, and peripheral pulses. Administer the prescribed sedative, and provide for safety. Remove any medication patches from the chest and all metallic objects. Place in supine position, and provide for privacy.

### PROCEDURE

1. Use standard precautions.
2. Turn on the cardioverter-defibrillator and ECG monitor.
3. Connect the client's ECG cable to the cardioverter. Select a lead with prominent R waves for monitoring.
4. Set cardioverter to "synchronize" mode. Observe the ECG waveform on the monitor for indications of synchronization, such as a flashing bold line or a blip. Many units also display the message "synchronized mode" on the monitor.
5. Place conductive pads on the chest below the right clavicle to the right of the sternum and in the midaxillary line on the left. If using conductive paste, spread it evenly on the defibrillator paddles.
6. Turn on the ECG recording strip for a continuous printout during the procedure.
7. Charge the paddles to the prescribed energy dose. The machine will beep to indicate that the selected energy level has been reached and that the paddles are ready for discharge.
8. The paddles are applied firmly to the chest over the conductive pads by the physician.
9. Turn oxygen off and remove it.
10. Ensure that no one is touching the client or the bed prior to discharge of the electrical shock. There may be a slight delay in shock delivery as the machine synchronizes with the R wave.
11. Assess client status and ECG rhythm. Assure a patent airway and the presence of a pulse.
12. The procedure may be repeated if unsuccessful. The energy level may be increased with each attempt.
13. Remove conductive pads. Using a dry gauze pad, clean paste from the chest and the paddles.

### AFTER THE PROCEDURE

Assess client for return of consciousness from sedative or cardioversion. Evaluate neurologic, cardiovascular, and respiratory status. Assess for possible complications, including emboli (especially cerebral), respira-

tory depression, and dysrhythmias. Document postcardioversion rhythm strip. Assess skin for burns. Document the procedure and the client's response in the medical record.

**DEFIBRILLATION** Unlike carefully synchronized cardioversion, *defibrillation* is an emergency procedure that delivers direct current without regard to the cardiac cycle. Ventricular fibrillation is immediately treated as soon as the dysrhythmia is recognized. Early defibrillation has been shown to improve survival in clients experiencing VF.

Defibrillation can be delivered by external or internal paddles or pads. Conductive gel pads or paste is applied, and external paddles or pads are placed on the chest wall at the apex and base of the heart (Figure 31–8 ■). Internal paddles are applied directly on the heart, and may be used in surgery, the emergency department, or critical care. Internal defibrillation is done only by a physician; external defibrillation may be performed by any healthcare provider who has been trained in the procedure. Automatic external defibrillators (AEDs) are available on most hospital units to allow early defibrillation for cardiac arrest. (See Procedure 31–3.)

### Pacemaker Therapy

A **pacemaker** is a pulse generator used to provide an electrical stimulus to the heart when the heart fails to generate or conduct its own at a rate that maintains the cardiac output. The pulse generator is connected to *leads* (insulated wires) passed intravenously into the heart or sutured directly to the epicardium. The leads sense intrinsic electrical activity of the heart and provide an electrical stimulus to the heart when necessary (pacing).



**Figure 31–8 ■** Placement of paddles for defibrillation.

Source: Floyd Jackson

Pacemakers are used to treat both acute and chronic conduction defects such as third-degree AV block. They also may be used to treat bradydysrhythmias and tachydysrhythmias.

*Temporary pacemakers* use an external pulse generator (Figure 31–9 ■) attached to a lead threaded intravenously into the right ventricle, to temporary pacing wires implanted during cardiac surgery, or to external conductive pads placed on the chest wall for emergency pacing.

## PROCEDURE 31-3 EMERGENCY EXTERNAL DEFIBRILLATION

### GATHER SUPPLIES

- Automatic external defibrillator or defibrillator with ECG cable and monitor
- Conductive gel pads or paste
- Dry gauze pads
- Emergency medications and cart with pacemaker, airway management equipment, and oxygen supplies.

### BEFORE THE PROCEDURE

Verify the lethal dysrhythmia, such as pulseless VT, VF, or asystole. Initiate the cardiac arrest (code) procedure, and obtain the defibrillator. If one is not immediately available, begin CPR until the emergency

cart and defibrillator are brought to the bedside. Place client in supine position on a firm surface.

### PROCEDURE

1. Turn on the defibrillator. Set it in *defibrillation* mode.
2. Turn ECG recording on for a continuous printout of events during the procedure.
3. Set the energy level and charge the paddles. Initial defibrillation is usually performed at 200 joules.
4. Place conductive pads on the chest, or spread conductive paste evenly on the paddles.
5. Position the paddles, holding them firmly on the chest wall.
6. **Ensure that no one is touching the client or the bed. State, "All clear."**
7. Depress the button on each paddle simultaneously to discharge the energy.
8. Immediately resume CPR.
9. Evaluate cardiac rhythm and for a pulse after approximately 2 minutes.
10. If the first attempt is unsuccessful, repeat the procedure, increasing the energy level to 300 joules and 360 joules for successive attempts. Reapply conductive paste as necessary.
11. Implement ACLS protocols.

### AFTER THE PROCEDURE

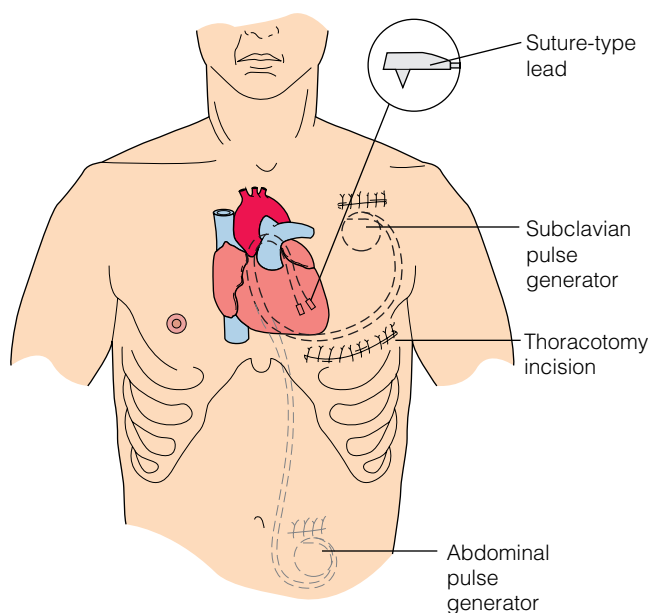
If the dysrhythmia is successfully converted, evaluate and support neurologic, cardiovascular, and respiratory status. Monitor and titrate any intravenous infusions as ordered. Maintain ventilatory support as needed. Evaluate skin for burns. Obtain blood for laboratory analysis

as ordered. Monitor vital signs and ECG continuously. Transfer to the intensive care unit (ICU) as indicated. Provide support and information to the client and family.



**Figure 31-9** ■ Programmable settings on a temporary pacemaker.  
Courtesy of Medtronic, Inc.

*Permanent pacemakers* use an internal pulse generator placed in a subcutaneous pocket in the subclavian space or abdominal wall. The generator connects to leads sewn directly onto the heart (*epicardial*) or passed transvenously into the heart (*endocardial*). Epicardial pacemakers (Figure 31-10 ■) require surgical exposure of the heart. Leads may be placed during cardiac surgery, or using a small subxiphoid incision to expose on the heart. Transvenous pacemaker leads are positioned in the right heart via the cephalic, subclavian, or jugular



**Figure 31-10** ■ A permanent epicardial pacemaker. The pulse generator may be placed in subcutaneous pockets in the subclavian or abdominal regions.

vein (Figure 31-11 ■). Local anesthesia can be used for permanent pacemaker insertion.

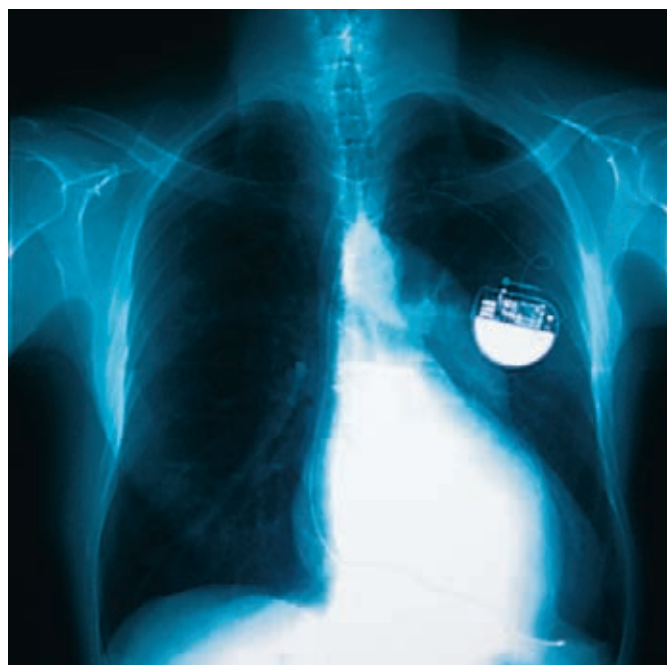
Pacemakers are programmed to stimulate the atria or the ventricles (*single-chamber pacing*), or both (*dual-chamber pacing*). Table 31-8 defines terms used to describe pacemaker modes and functions. The most commonly used pacemakers either (1) sense activity in and pace the ventricles only; or (2) sense activity in and pace both the atria and the ventricles. Dual-chamber or *atrioventricular sequential pacing* stimulates both chambers of the heart in sequence. AV pacing imitates the normal sequence of atrial contraction followed by ventricular contraction, improving cardiac output.

Pacing is detected on the ECG strip by the presence of pacing artifact (Figure 31-12 ■). A sharp spike is noted before the P wave with atrial pacing, and before the QRS complex with ventricular pacing. Pacing spikes are seen before both the P wave and QRS complex in AV sequential pacing. Capture is noted if there is a contraction of the chamber immediately following the pacer spike. Problems in sensing, pacing, and capture are noted in Table 31-9.

Care of the client with a temporary or permanent pacemaker focuses on monitoring for pacemaker malfunctioning, maintaining safety (Box 31-5), and preventing infection and post-operative complications. Nursing care for the client having a pacemaker implant is outlined on page 1012.

### Implantable Cardioverter-Defibrillator

Sudden cardiac death claims more than 300,000 lives per year in the United States (Woods et al., 2004). The *implantable cardioverter-defibrillator (ICD)* detects life-threatening changes in the cardiac rhythm and automatically delivers an electric shock to convert the dysrhythmia back into a normal



**Figure 31-11** ■ A permanent transvenous (endocardial) pacemaker with the lead placed in the right ventricle via the subclavian vein.

Source: Phototake NYC

TABLE 31–8 Terms Used to Describe Pacemaker Functions

TERM	DEFINITION
Asynchronous pacing	Pacemaker delivers a pacing stimulus at a set rate regardless of intrinsic cardiac activity.
Base rate	Rate at which the pacemaker paces when no cardiac activity is sensed.
Capture	The ability of the pacing stimulus to generate a cardiac depolarization.
Demand pacing	Pacemaker delivers a pacing stimulus only when the intrinsic rate falls below the pacemaker's base rate.
Dual-chamber pacing	Allows both the atria and the ventricles to be paced; most frequently used permanent pacing mode.
Lead	An insulated wire that senses intrinsic cardiac activity and delivers a pacing stimulus as programmed.
Output	The electrical stimulus delivered by the pulse generator.
Pacing spike	A small vertical spike noted on the ECG with every pacemaker stimulus.
Sensing	The pacemaker's ability to identify and respond to intrinsic cardiac activity.
Single-chamber pacing	Pacing of only the atria or the ventricles, not both; most common temporary pacing mode used.

Source: Adapted from *Cardiac Nursing* (5th ed.) by S. L. Woods, E. S. Froelicher, S. A. Motzer, & E. Bridges, 2004, Philadelphia: Lippincott.

rhythm. ICDs are used for sudden death survivors, clients with recurrent ventricular tachycardia, and clients with demonstrated risk factors for sudden death. ICDs can deliver a shock as needed, provide pacing on demand, and can store ECG records of tachycardic episodes (Woods et al., 2004).

A pulse generator connected to lead electrodes for rhythm detection and current delivery is implanted in the left pectoral

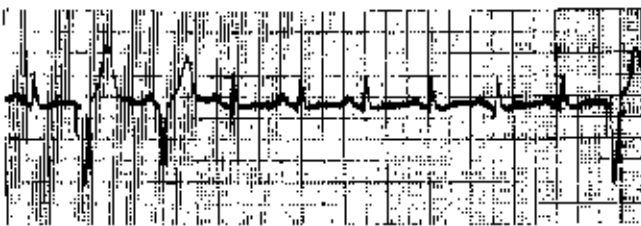
region. The lead is threaded transvenously to the apex of the right ventricle. The ICD is programmed to sense a change in heart rate or rhythm. When it detects a potentially lethal rhythm, it shocks the heart to convert the rhythm. The device can be programmed or reprogrammed at the bedside as necessary. The ICD may be tested prior to discharge.

Local or general anesthesia is used, and the client may be discharged within 24 hours. The lithium-powered battery must be surgically replaced every 5 years. Complications and nursing care are similar to that for a client having a permanent pacemaker implant (see the Nursing Care box on page 1012).

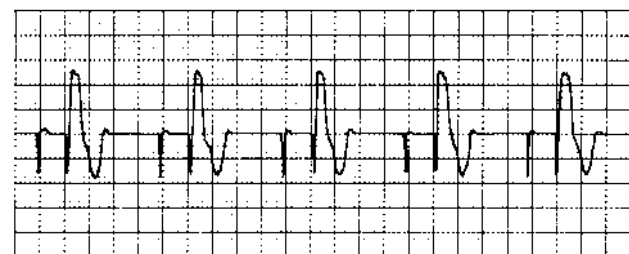
The client may briefly lose consciousness before the device discharges, typically regaining consciousness quickly after the episode. Some clients report significant discomfort with ICD



A



B




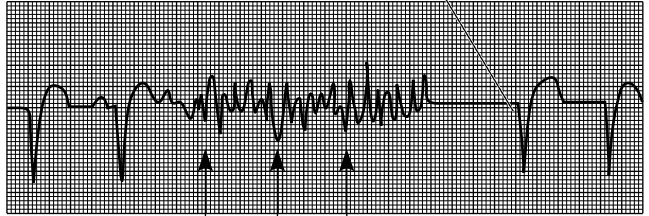
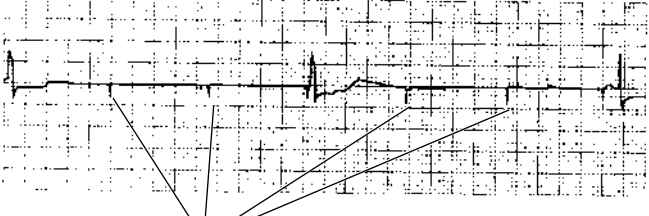
C

**Figure 31–12** ■ Pacing artifacts. *A*, Atrial pacing and ventricular sensing. Note the pacer spike preceding the P wave. *B*, Ventricular demand pacing. Note the absence of pacer spikes when the client's natural rhythm predominates. *C*, Atrioventricular pacing. Note the pacer spikes preceding both P waves and QRS complexes.

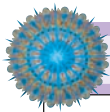
### BOX 31–5 Safety for Clients with a Temporary Pacemaker

- Ensure that all electrical equipment in use has a grounded plug; do not use adapters or extension cords.
- Encourage the use of battery-powered equipment (e.g., electric razor).
- Remove any damaged electrical equipment from the unit, including equipment that
  - a. Has been abused (e.g., has been dropped or in which liquid has been spilled).
  - b. Has given anyone a shock.
  - c. Has frayed, worn, or otherwise damaged electrical cords or plugs.
  - d. Has other evidence of impaired function, such as a hot smell during use or control knobs that are loose or do not consistently produce the expected response.
- Wear gloves when handling the pacemaker electrodes or wires.
- Insulate pacemaker terminals and pacing wires with nonconductive, moistureproof material (e.g., a rubber glove).
- Test the pacemaker battery prior to use.
- Keep a spare pacemaker, cable, batteries, and battery tester available at all times.
- Immediately report any apparent deviation from expected pacemaker function.

TABLE 31–9 Potential Pacemaker Problems and Corrective Strategies

PROBLEM	POSSIBLE CAUSES	CORRECTIVE MEASURES
<p><b>Undersensing</b> Device fails to detect existing cardiac depolarizations, therefore competes with the native rhythms.</p> <p>Undetected R waves</p>  <p>Competing pacer spikes</p>	<p>Lead disconnected from pacer or from viable myocardium. Sensitivity set too low. Lead fracture. Low battery.</p>	<p>Check connection of lead to pacer. Increase sensitivity. Reposition or change lead. Change battery.</p>
<p><b>Oversensing</b> Device detects noncardiac electrical events and interprets them as cardiac depolarizations, therefore is wrongly inhibited from pacing.</p> <p>When artifact ceases, pacing resumes</p>  <p>Pacer interprets artifact as cardiac activity and fails to fire</p>	<p>Sensitivity set too high.  Interference from electrical sources (ungrounded equipment, short circuits) is detected and misinterpreted by the device. Lead disconnected from pacer or from viable myocardium.</p>	<p>Decrease sensitivity (turn sensing control to a LARGER number). Remove all ungrounded electrical equipment or have it evaluated by hospital engineers.  Check connection of lead to pacer.</p>
<p><b>Noncapture</b> Device emits stimuli that fail to depolarize the myocardium.</p>  <p>Pacer stimuli that fail to initiate myocardial depolarization</p>	<p>Output set too low in the noncaptured chamber. Lead fracture. High pacing threshold due to medication or metabolic changes. Low battery.</p>	<p>Increase output in the noncaptured chamber. Reposition or change lead. Alter medication regimen, correct metabolic changes. Change battery.</p>

Source: From "Cardiac Rhythm Control Devices" (p. 92) by C. L. Witherell, 1994, *Critical Care Nursing Clinics of North America*, 6(1).



## NURSING CARE OF THE CLIENT HAVING A Permanent Pacemaker Implant

### Preoperative Care

- Provide routine preoperative care and teaching as outlined in Chapter 4 ∞.
- Assess knowledge and understanding of the procedure, clarifying and expanding on existing knowledge as needed. *Clarifying knowledge, providing information, and conveying emotional support reduces anxiety and fear and allows the client to develop a realistic outlook regarding pacer therapy.*
- Place ECG monitor electrodes away from potential incision sites. *This helps preserve skin integrity.*
- Teach range-of-motion (ROM) exercises for the affected side. *ROM exercises of the affected arm and shoulder prevent stiffness and impaired function following pacemaker insertion.*

### Postoperative Care

- Provide postoperative monitoring, analgesia, and care as outlined in Chapter 4 ∞.
- Obtain a chest x-ray as ordered. *A postoperative chest x-ray is used to identify lead location and detect possible complications, such as pneumothorax or pleural effusion.*
- Position for comfort. Minimize movement of the affected arm and shoulder during the initial postoperative period. *Restricting movement minimizes discomfort on the operative side and allows the leads to become anchored, reducing the risk of dislodging.*
- Assist with gentle ROM exercises at least three times daily, beginning 24 hours after pacemaker implantation. *ROM exercises help restore normal shoulder movement and prevent contractures on the affected side.*
- Monitor pacemaker function with cardiac monitoring or intermittent ECGs. Report pacemaker problems to the physician:
  - Failure to pace. *This may indicate battery depletion, damage or dislodgement of pacer wires, or inappropriate sensing.*
  - Failure to capture (the pacemaker stimulus is not followed by ventricular depolarization). *The electrical output of the pacemaker may not be adequate, or the lead may be dislodged.*
  - Improper sensing (the pacemaker is firing or not firing, regardless of the intrinsic rate). *This increases the risk for decreased cardiac output and dysrhythmias.*
  - Runaway pacemaker (a pacemaker firing at a rapid rate). *This may be due to generator malfunction or problems with sensing.*
  - Hiccups. *A lead positioned near the diaphragm can stimulate it, causing hiccups. Hiccups may occur in extremely thin clients or may indicate a medical emergency with perforation of the right ventricle by the pacing electrode tip.*
- Assess for dysrhythmias and treat as indicated. *Until the catheter is “seated” or adheres to the myocardium, its move-*

*ment may cause myocardial irritability and dysrhythmias. Fibrotic tissue develops within 2 to 3 days.*

- Document the date of pacemaker insertion, the model and type, and settings. *This information is important for future reference.*
- Immediately report signs of potential complications, including myocardial perforation, cardiac tamponade, pneumothorax or hemothorax, emboli, skin breakdown, bleeding, infection, endocarditis, or poor wound healing (see Chapter 32 ∞ for more information about cardiac tamponade and endocarditis, and Chapter 38 ∞ for pneumothorax and hemothorax). *Early identification of complications allows for aggressive intervention.*
- Provide a pacemaker identification card including the manufacturer’s name, model number, mode of operation, rate parameters, and expected battery life. *This card provides a reference for the client and future healthcare providers.*

### Home Care

Provide appropriate teaching for the client and family about:

- Placement of the pacemaker generator and leads in relation to the heart.
- How the pacemaker works and the rate at which it is set.
- Battery replacement. Most pacemaker batteries last 6 to 12 years. Replacement requires an outpatient surgery to open the subcutaneous pocket and replace the battery.
- How to take and record the pulse rate. Instruct to assess pulse daily before arising and notify the physician if 5 or more bpm slower than the preset pacemaker rate.
- Incision care and signs of infection. Bruising may be present following surgery.
- Signs of pacemaker malfunction to report, including dizziness, fainting, fatigue, weakness, chest pain, or palpitations.
- Activity restrictions as ordered. This usually is limited to contact sports (which may damage the generator) and avoiding heavy lifting for 2 months after surgery.
- Resume sexual activity as recommended by the physician. Avoid positions that cause pressure on the site.
- Avoid tight-fitting clothing over the pacemaker site to reduce irritation and avoid skin breakdown.
- Carry the pacemaker identification card at all times, and wear a Medic-Alert bracelet or tag.
- Notify all care providers of the pacemaker.
- Do not hold or use certain electrical devices over the pacemaker site, including household appliances or tools, garage door openers, antitheft devices, or burglar alarms. Pacemaker will set off airport security detectors; notify security officials of its presence.
- Maintain follow-up care with the physician as recommended.

discharge (like a “blow to the chest”). A person in direct contact with the client when the device discharges may experience a tingling sensation.

### Cardiac Mapping and Catheter Ablation

Cardiac mapping and catheter ablation are used to locate and destroy an ectopic focus. These diagnostic and therapeutic measures use electrophysiology techniques, and can be per-

formed in the cardiac catheterization laboratory. *Cardiac mapping* is used to identify the site of earliest impulse formation in the atria or the ventricles. Intracardiac and extracardiac catheter electrodes and computer technology are used to pinpoint the ectopic site on a map of the heart. These same catheters can be used to deliver the ablative intervention.

*Ablation* destroys, removes, or isolates an ectopic focus. In most instances, radio-frequency energy produced by high-





frequency alternating current is used to create heat as it passes through tissue. Catheter ablation is used to treat supraventricular tachycardias, atrial fibrillation and flutter, and, in some cases, paroxysmal ventricular tachycardia (Woods et al., 2004).

Anticoagulant therapy may be started after catheter ablation to reduce the risk of clot formation at the ablation site.

### Other Therapies

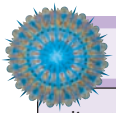
In addition to medications and interventional techniques, other measures may be used to treat selected dysrhythmias. Vagal maneuvers that stimulate the parasympathetic nervous system may be used to slow the heart rate in supraventricular tachycardias. These maneuvers include *carotid sinus massage* and the *Valsalva maneuver*. Carotid sinus massage is performed only by a physi-

cian during continuous cardiac monitoring. Excessive slowing of the heart rate may result. The Valsalva maneuver, forced exhalation against a closed glottis (e.g., bearing down), increases intrathoracic pressure and vagal tone, slowing the pulse rate.



## NURSING CARE

Caring for the client with cardiac dysrhythmias requires the ability to recognize, identify, and, in some cases, promptly treat the dysrhythmia. The urgency of intervention is determined by the effects of the dysrhythmia on the client. Nursing care focuses on maintaining cardiac output, monitoring the response to therapy, and teaching. Also see the Nursing Care Plan for a client with a dysrhythmia.



### NURSING CARE PLAN A Client with Supraventricular Tachycardia

Elisa Vasquez, 53 years old, is admitted to the cardiac unit with complaints of palpitations, light-headedness, and shortness of breath. Her history reveals rheumatic fever at age 12 with subsequent rheumatic heart disease and mitral stenosis. An intravenous line is in place and she is receiving oxygen. Marcia Lewin, RN, is assigned to Ms. Vasquez.

#### ASSESSMENT

Ms. Lewin's assessment reveals that Ms. Vasquez is moderately anxious. Her ECG shows supraventricular tachycardia (SVT) with a rate of 154. Vital signs: T 98.8°F (37.1°C), R 26, BP 95/60. Peripheral pulses weak but equal, mucous membranes pale pink, skin cool and dry. Fine crackles noted in both lung bases. A loud S<sub>3</sub> gallop and a diastolic murmur are noted. Ms. Vasquez is still complaining of palpitations and tells Ms. Lewin, "I feel so nervous and weak and dizzy." Ms. Vasquez's cardiologist orders 2.5 mg of verapamil to be given slowly via intravenous push and tells Ms. Lewin to prepare to assist with synchronized cardioversion if drug therapy does not control the ventricular rate.

#### DIAGNOSES

- *Decreased Cardiac Output* related to inadequate ventricular filling associated with rapid tachycardia
- *Ineffective Tissue Perfusion: Cerebral/Cardiopulmonary/Peripheral* related to decreased cardiac output
- *Anxiety* related to unknown outcome of altered health state

#### EXPECTED OUTCOMES

- Maintain adequate cardiac output and tissue perfusion.
- Demonstrate a ventricular rate within normal limits and stable vital signs.
- Verbalize reduced anxiety.
- Verbalize an understanding of the rationale for the treatment measures to control the heart rate.

#### PLANNING AND IMPLEMENTATION

- Provide oxygen per nasal cannula at 4 L/min.
- Continuously monitor ECG for rate, rhythm, and conduction. Assess vital signs and associated symptoms with changes in ECG. Report findings to physician.
- Explain the importance of rapidly reducing the heart rate. Explain the cardioversion procedure and encourage questions.

- Encourage verbalization of fears and concerns. Answer questions honestly, correcting misconceptions about the disease process, treatment, or prognosis.
- Administer intravenous diazepam as ordered before cardioversion.
- Document pretreatment vital signs, level of consciousness, and peripheral pulses.
- Place emergency cart with drugs and airway management supplies in client unit.
- Assist with cardioversion as indicated.
- Assess LOC, level of sedation, cardiovascular and respiratory status, and skin condition following cardioversion.
- Document procedure and postcardioversion rhythm, and response to intervention.

#### EVALUATION

Intravenous verapamil lowers Ms. Vasquez's heart rate to 138 for a short time, after which it increases to 164 with BP of 82/64. Her cardiologist, Dr. Mullins, performs carotid sinus massage. The ventricular rate slows to 126 for 2 minutes, revealing atrial flutter waves, and then returns to a rate of 150. Dr. Mullins explains the treatment options, including synchronized cardioversion. Ms. Vasquez agrees to the procedure.

Ms. Vasquez is lightly sedated and synchronized cardioversion is performed. One countershock converts Ms. Vasquez to regular sinus rhythm at 96 beats/min with BP 112/60.

Ms. Vasquez is sleepy from the sedation but recovers without incident. She states that she feels "much better," and her vital signs return to her normal levels. She remains in NSR with a rate of 86 to 92 for the remainder of her hospital stay. Dr. Mullins places Ms. Vasquez on furosemide to treat manifestations of mild heart failure.

#### CRITICAL THINKING IN THE NURSING PROCESS


1. What is the scientific basis for using carotid massage to treat supraventricular tachycardias? Was this an appropriate maneuver in the case of Ms. Vasquez?
2. What other treatment options might the physician have used to treat Ms. Vasquez's supraventricular tachycardia if she had been asymptomatic with stable vital signs?
3. Develop a teaching plan for Ms. Vasquez related to her prescription for furosemide.

*See Evaluating Your Response in Appendix C.*

## Health Promotion

Health promotion measures to prevent coronary heart disease also reduce the risk for dysrhythmias. In most cases, dysrhythmias develop as a result of ischemic or structural changes in the heart, rather than in isolation. Advise clients who are at risk or who complain of occasional palpitations or “flutters” in their chest to reduce their intake of caffeine and other sympathetic nervous system stimulants, such as excess chocolate.

## Assessment

Assessment is vital before treating any suspected dysrhythmia. What appears to be ventricular tachycardia on the monitor may be the client scratching or brushing the teeth. Apparent asystole on the monitor may be due to a loose electrode patch. Similarly, a heart rate of 52 beats/min may not affect the overall cardiac output in some clients. Review Chapter 30  for complete assessment of the client with a cardiac problem.

- **Health history:** Complaints of palpitations (ask for further definition of palpitations), “fluttering” sensations, or a sensation of the heart racing; episodes of dizziness, lightheadedness, or syncope (fainting); timing (duration, time of day); correlation with food or beverage intake, activity; presence of chest pain, shortness of breath, or other associated symptoms; history of heart or endocrine disease (such as hyperthyroidism); current medications.
- **Physical examination:** LOC; vital signs, including apical pulse for a full minute; regularity and amplitude of peripheral pulses; color; presence of dyspnea, adventitious lung sounds; ECG rhythm analysis; oxygen saturation levels.

## Nursing Diagnoses and Interventions

The effect of the dysrhythmia on cardiac output is the priority of nursing care. Other potential nursing diagnoses related to dysrhythmias may include *Ineffective Tissue Perfusion*, *Activity Intolerance*, and *Fear or Anxiety*.

### Decreased Cardiac Output

Dysrhythmias can affect cardiac output. Bradycardias decrease cardiac output if the stroke volume does not increase to compensate for the slow heart rate. Tachycardia reduces diastolic filling time, affecting stroke volume and coronary artery perfusion. Loss of the atrial kick in junctional rhythms, atrial fibrillation, and AV blocks also decreases ventricular filling and cardiac output. In ventricular fibrillation, loss of ventricular contractions causes cardiac arrest and no cardiac output.

#### PRACTICE ALERT

Before treating any dysrhythmia, assess the client, not just the monitor! Loose electrode pads, disconnected leads or cables, and muscle movement can simulate critical dysrhythmias. The client’s condition is the best indicator of the need for treatment.

- Assess for decreased cardiac output: decreased LOC; tachycardia; tachypnea; hypotension; low oxygen saturation; diaphoresis; low urine output; cool, clammy, mottled skin; pallor or cyanosis; diminished peripheral pulses. *Initial signs of decreased cardiac output may be subtle, such as decreased*

*LOC. Early recognition of the dysrhythmia’s effect on cardiac output facilitates appropriate treatment and may prevent further adverse effects.*

- Monitor ECG; post ECG strip every shift and when rhythm changes occur. *Documenting cardiac rhythm provides a record of disease progression and treatment effectiveness.*

#### PRACTICE ALERT

Assess vital signs, ECG, and oxygen saturation every 5 to 15 minutes during acute dysrhythmic episodes and during antidysrhythmic drug infusions. These data provide a record of cardiac output during the dysrhythmia. Antidysrhythmic drugs can adversely affect heart rate, rhythm, and blood pressure, further decreasing cardiac output.

- Assess for underlying causes of dysrhythmias, such as hypovolemia, hypoxia, anemia, vagal stimulation, or medications. *Sinus tachycardia often develops in response to tissue hypoxia. Vagal stimulation (such as the Valsalva maneuver) can precipitate bradycardia.*
- Assess serum electrolytes (especially potassium, calcium, and magnesium) and digitalis and antidysrhythmic drug levels as indicated. Report abnormal values. *Electrolyte imbalances affect cardiac depolarization and repolarization and may cause dysrhythmias. Toxic levels of digitalis and antidysrhythmic drugs can precipitate further dysrhythmias. Impaired renal or hepatic function increases the risk for toxicity, as does aging.*
- Be prepared to administer antidysrhythmic medications as indicated. Implement advanced cardiac life support (ACLS) protocols as needed. *Emergency drugs should be readily available, especially on units with high-risk clients. See Table 31–7 and the Medication Administration box on page 1006 for drugs used to treat common dysrhythmias that may affect cardiac output.*
- If appropriate, instruct to perform the Valsalva maneuver (bear down as if straining or coughing) for supraventricular tachycardia or ventricular tachycardia without angina. *Vagal maneuvers stimulate the parasympathetic system and may terminate some dysrhythmias. The Valsalva maneuver is contraindicated if chest pain occurs with the dysrhythmia.*
- Prepare to assist with cardioversion. Prepare the client per orders or hospital protocol (see Procedure 31–2). Explain the procedure to reduce anxiety. Have emergency equipment readily available. *Elective or emergency cardioversion is a treatment of choice for certain dysrhythmias.*

#### PRACTICE ALERT

On recognizing ventricular fibrillation and cardiac arrest, begin emergency procedures. Call for help. Obtain defibrillator and immediately defibrillate. If the defibrillator will be brought by another healthcare provider, begin CPR. Initiate ACLS protocols and assist with resuscitation measures as directed. Cardiac output ceases with ventricular fibrillation. Immediate or early defibrillation has been shown to have the greatest impact on survival following cardiac arrest.

- After cardiac arrest, transfer to critical care. Perform and document head-to-toe assessment; obtain laboratory tests,

12-lead ECG, and chest X-ray as ordered; monitor and maintain oxygenation and intravenous infusions; and monitor vital signs and cardiac rhythm. *The period following resuscitation is critical, necessitating careful monitoring. Postarrest assessment allows comparison of the client's condition with prearrest status and may identify CPR-related injuries. Correcting electrolyte disturbances, hypoxia, and acid–base imbalances is important to prevent further dysrhythmias and potential adverse effects on cardiac output. Intravenous access is crucial to maintain drug infusions. Hemodynamic monitoring may be instituted. The 12-lead ECG documents myocardial status, and the chest X-ray provides information about pulmonary status and possible thoracic injury due to CPR.*

- Notify the family of significant changes in the client's condition or cardiac arrest, providing up-to-date information. Prepare family members prior to visits by explaining interventions (such as invasive tubes, a ventilator, or additional equipment) implemented since the last visit. *Concern for the family and significant others is part of holistic nursing. Researchers studying the needs of families have found that one of the most important needs was information about their loved one's condition. Clients and families need and appreciate honest communication and compassionate care. Preparing the family for critical changes in the client's condition and plan of care helps them to cope with a situational crisis.*

## Community-Based Care

Dysrhythmias have a significant physical and psychologic impact on the client and all family members. Many of these clients and their families are under a great deal of stress from frequent hospitalizations, experimentation with therapies, frustration, and the fear of sudden cardiac death. A major teaching effort focuses on coping strategies and lifestyle changes, as well as specific management of prescribed therapies. Include the following topics as appropriate when teaching the client and family for home care:

- Function, maintenance, precautions, and signs of malfunction or complications of any implanted device such as a pacemaker or ICD
- Monitoring pulse rate and rhythm
- Activity or dietary restrictions, and any potential effects of the dysrhythmia or its treatment on lifestyle
- Medication management to reduce the risk of dysrhythmias, including the desired and potential adverse effects of anti-dysrhythmic drugs
- Specific instructions related to planned diagnostic tests or procedures
- The importance of follow-up visits with the cardiologist
- The importance of and where to obtain CPR training for the client and family members.

In addition, discuss fears related to treatment or implanted devices, such as that of shocking a significant other during close contact or sexual activity. Explain that if a shock occurs, the partner may feel a slight buzz or tingling but should not be harmed. Refer to and encourage the client and family to attend a peer support group for the specific condition.

## THE CLIENT WITH SUDDEN CARDIAC DEATH

**Sudden cardiac death (SCD)** is defined as unexpected death occurring within 1 hour of the onset of cardiovascular symptoms. It usually is caused by ventricular fibrillation and cardiac arrest. *Cardiac arrest* is the sudden collapse, loss of consciousness, and cessation of effective circulation that precedes biologic death. Worldwide, less than 6% of out-of-hospital cardiac arrest victims survive. In communities of North America that have organized lay rescuer and automated external defibrillator (AED) programs, the survival rate is significantly better, ranging from 49% to 74% when a witnessed arrest due to ventricular fibrillation occurs (AHA, 2005a).

Almost 50% of all deaths due to coronary heart disease are attributed to SCD. Coronary heart disease causes up to 80% of all sudden cardiac deaths in the United States. Other cardiac pathologies such as cardiomyopathy and valvular disorders also may lead to SCD. Noncardiac causes of sudden death include electrocution, pulmonary embolism, and rapid blood loss from a ruptured aortic aneurysm.

Ventricular fibrillation is the most common dysrhythmia associated with sudden cardiac death, accounting for 65% to 80% of cardiac arrests. Sustained severe bradydysrhythmias, *asystole* or cardiac standstill, and pulseless electrical activity (organized cardiac electrical activity without a mechanical response) are responsible for most remaining SCDs (Kasper et al., 2005). Selected cardiac and noncardiac causes of sudden cardiac death are listed in Box 31–6.

Risk factors for SCD are those associated with coronary heart disease (see the first section of this chapter). Advancing age and male gender are powerful risk factors. After age 65, the gap between male and female incidence of SCD narrows (Kasper et al., 2005). Clients with dysrhythmias such as recurrent VT may have a higher risk of SCD. Women with acute myocardial infarction, however, are more likely to present with

### BOX 31–6 Selected Causes of Sudden Cardiac Death

#### Cardiac Causes

- Coronary heart disease
- Reperfusion following ischemia
- Myocardial hypertrophy
- Cardiomyopathy
- Inflammatory myocardial disorders
- Valve disorders
- Primary electrical disorders
- Dissecting or ruptured aortic or ventricular aneurysm
- Cardiac drug toxicity

#### Noncardiac Causes

- Pulmonary embolism
- Cerebral hemorrhage
- Autonomic dysfunction
- Choking
- Electrical shock
- Electrolyte and acid–base imbalances

cardiac arrest and cardiogenic shock than with ventricular tachycardia (Kasper et al., 2005).

## Pathophysiology

Evidence of coronary heart disease with significant atherosclerosis and narrowing of two or more major coronary arteries is found in 75% of SCD victims. Although most have had prior myocardial infarction, only 20% to 30% have recent acute myocardial infarction. An acute change in cardiovascular status precedes cardiac arrest by up to 1 hour; however, often the onset is instantaneous or abrupt. Tachycardia develops, and the number of PVCs increases. This is followed by a run of ventricular tachycardia that deteriorates into ventricular fibrillation (Kasper et al., 2005).

Abnormalities of myocardial structure or function also contribute. Structural abnormalities include infarction, hypertrophy, myopathy, and electrical anomalies. Functional deviations are caused by such factors as ischemia followed by reperfusion, altered homeostasis, autonomic nervous system and hormone interactions, and toxic effects. The interactions of the two cause myocardial instability and may precipitate fatal dysrhythmias.

## Manifestations

SCD may be preceded by typical manifestations of acute coronary syndrome or myocardial infarction, including severe chest pain, dyspnea or orthopnea, and palpitations or light-headedness. The event itself is abrupt, with complete loss of consciousness and death within minutes. If ventricular tachycardia precedes cardiac arrest, consciousness and mentation may be impaired prior to collapse and loss of consciousness.

## INTERDISCIPLINARY CARE

The goal of care is to restore cardiac output and tissue perfusion. Treatment measures are initiated as soon as clinical cardiac arrest is verified by the absence of respirations and carotid or femoral

pulses. Basic and ACLS measures must be instituted within 2 to 4 minutes of cardiac arrest to prevent permanent neurologic damage and ischemic injury to other organs.

## Basic Life Support

Basic life support (BLS) begins with identification of the cardiac arrest and initiation of an emergency response.

Providers trained in use of the AED should immediately defibrillate the client in VF. Self-adhesive conductive pads attached to connecting cables are positioned on the chest (Figure 31–13 ■). The AED analyzes the rhythm, and advises the provider to charge the device if VF is detected. After warning all personnel to stand clear, the shock button is depressed to deliver a shock. Following the shock, CPR is immediately initiated. After approximately 2 minutes or five cycles of CPR, the rhythm is evaluated and circulation checked. The sequence of analysis, shock, CPR is continued and ACLS protocols are initiated (AHA, 2005a).

*Cardiopulmonary resuscitation* is a mechanical attempt to maintain tissue perfusion and oxygenation using oral resuscitation and external cardiac compressions. All healthcare providers need to be proficient in CPR. The technique should be performed according to AHA guidelines and hospital protocol. (See Box 31–7.) Research demonstrates clear benefit from sustained, effective chest compressions, yet compressions often are interrupted for ventilation, assessment of pulses, and other measures. Many clients are excessively ventilated and underperfused during CPR (Sanders & Ewy, 2005). The AHA 2005 guidelines for CPR reflect this research (AHA, 2005a).

CPR carries a high risk for both cardiac and noncardiac trauma. CPR-related complications include injuries to the skin, thorax, upper airway, abdomen, lungs, heart, and great vessels. These complications can be minimized by adhering to accepted CPR techniques.

## Advanced Life Support

Advanced life support (ALS), provided by specially trained healthcare personnel, includes advanced airway support (inser-

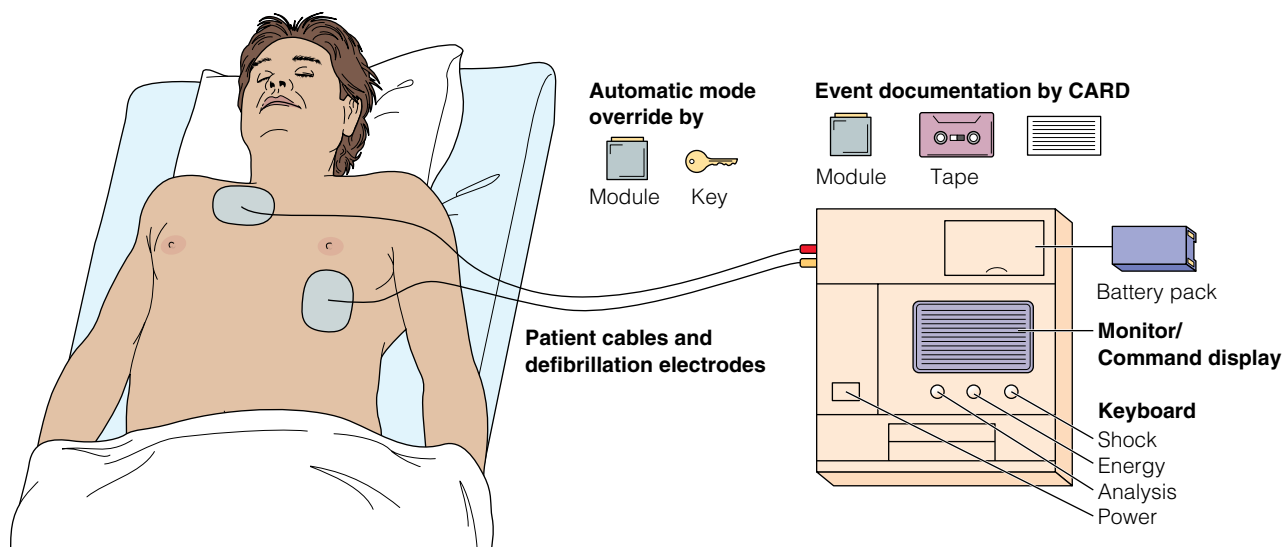


Figure 31–13 ■ Schematic of an automated external defibrillator (AED) attached to a client.

**BOX 31–7 Cardiopulmonary Resuscitation**

1. Assess for responsiveness; shake the client and shout.
2. Call for help. Dial 911 (if outside the healthcare facility) or initiate the institutional code or cardiac arrest procedure.
3. Open the airway using the head-tilt/chin-lift maneuver. Simultaneously press down on the forehead with one hand while lifting the chin upward with the other (part A of the accompanying figure).
4. Check for breathing; look and listen. Inspect the chest for rise and fall with respirations; listen and feel for air movement through the nose or mouth. This step should take no more than 10 seconds.
5. If not breathing, begin rescue breathing using a pocket mask, mouth shield, or bag-valve mask (see part A of the figure). Administer two breaths (1 second per breath), observing for rise of the chest with each breath.
6. Check the carotid or femoral artery for a pulse ( $\leq 10$  second).
7. If a pulse is present, continue rescue breathing, administering 8 to 10 breaths per minute, until help arrives or spontaneous respirations resume. Recheck the carotid pulse every 2 minutes.
8. If no pulse is present, analyze rhythm and defibrillate or, if unwitnessed arrest or AED unavailable, initiate external cardiac compressions. Place on a firm surface. Position the heel of one hand in the center of the chest between the nipples (child and adult), with the other hand on top and the fingers either interlocked or extended (see part B of the figure).
9. Initiate hard and fast cardiac compressions, pressing straight down to depress the sternum 1.5 to 2 inches, keeping the elbows locked and positioning the shoulders directly over the hands (part C of the figure). Release pressure completely between compressions but do not lift the hands from the chest.
10. Compress the chest at a rate of approximately 100 times per minute. With one- or two-rescuer CPR, provide two breaths after each 30 compressions. Assess the pulse after five complete cycles of 30 compressions and two breaths; continue CPR until help arrives.



A



B



C

A, Head-tilt/chin-lift maneuver and using a bag-valve mask.  
 B, Placement of hands on the sternum between the nipples.  
 C, Arm, hand, and shoulder position for cardiac massage.

tion of a laryngeal mask airway, esophageal-tracheal Combitube, or endotracheal intubation) to maintain the airway and oxygenation, use of intravenous drugs following specific protocols, and additional interventions such as repeated defibrillation procedures and cardiac pacing. Epinephrine, vasopressin, sodium bicarbonate, and antidysrhythmic drugs such as amiodarone, bretylium, lidocaine, procainamide, magnesium sulfate, and atropine are used to attempt to restore and maintain an effective cardiac rhythm.

**Postresuscitation Care**

Clients who experience sudden cardiac death associated with ventricular fibrillation and acute MI have the best prognosis (Kasper et al., 2005). The client is transferred to a coronary care unit and MI treatment measures are instituted. Antidysrhythmic drugs may be continued for 24 to 48 hours to reduce the risk of subsequent episodes of VF.

Because the risk for recurrent SCD is significant in survivors, extensive diagnostic testing and interventions such as

angioplasty or surgical revascularization of the myocardium, ablation, or an implantable cardioverter-defibrillator may be indicated.



## NURSING CARE

Nursing care of the client experiencing sudden cardiac death requires prompt recognition of the event and immediate initiation of BLS and ALS protocols. As noted before, fast and effective cardiac compressions and early defibrillation of unstable VT and VF are the most important keys to survival of cardiac arrest victims. Important concepts of emergency cardiac care follow:

- Treat the client, not the monitor. Recognize signs and symptoms of cardiac compromise early.
- Activate the emergency medical services system (i.e., call a code or call 911).
- Begin and continue basic cardiac life support principles throughout the resuscitation effort.
- Continually assess the effectiveness of emergency interventions.
- Defibrillate pulseless VT or VF as soon as possible.
- Initiate ALS protocols early.

The family is not forgotten during resuscitation. If the family is present, they are usually offered a private consultation room in which to await the outcome. If the family is not present, they are notified that their family member is not doing well and asked to come to the hospital as soon as possible. The situation is presented in a careful manner to prevent the family from racing to the hospital, precipitating an automobile crash. Pastoral care or the family's choice of spiritual support is offered to help during this difficult time. Attendance of family members during resuscitation efforts is controversial, and depends on institutional protocols and family desires.

After successful resuscitation, the nurse provides care specific to the client's underlying disease processes and needs. In-

travenous infusions such as lidocaine, bretylium, or dopamine may be ordered to prevent further dysrhythmias and maintain hemodynamic stability.

If the client does not survive the arrest, the nurse provides post-mortem care and emotional and spiritual support to the family.

Nursing diagnoses to consider for the client experiencing SCD include the following:

- *Ineffective Tissue Perfusion: Cerebral* related to ineffective cardiac output
  - *Impaired Spontaneous Ventilation* related to cardiac arrest
  - *Spiritual Distress* related to unexplained sudden cardiac death
  - *Disturbed Thought Processes* related to compromised cerebral circulation
  - *Fear* related to risk for future episodes of sudden cardiac death.
- The risk for a future episode of sudden cardiac death requires careful and effective teaching for home care prior to discharge. Discuss the following topics with the client and family:
- Risk factor reduction for coronary heart disease
  - Planned diagnostic studies to identify the cause of SCD and possible interventions
  - The risks and benefits of an ICD if appropriate
  - The importance of carrying a card at all times listing all current medications and the healthcare provider
  - Early manifestations or warning signs of cardiac arrest
  - The importance of CPR training and maintaining proficiency in performing CPR (Provide referral to local CPR training providers or scheduled classes through the AHA or American Red Cross.)

Nurses can impact death rates from cardiac arrest through community teaching as well. Survival rates from sudden cardiac death improve in communities in which a significant portion of the population is trained in CPR and early response by EMS agencies is stressed. Work with community groups and individuals can help create a population of people able to perform effective CPR.

## EXPLORE MEDIA LINK

### Prentice Hall Nursing MediaLink DVD-ROM



Audio Glossary  
NCLEX-RN® Review

#### Animation/Video

Automated External Defibrillation  
Coronary Heart Disease  
Dysrhythmias  
Nifedipine  
Propranolol

### COMPANION WEBSITE [www.prenhall.com/lemone](http://www.prenhall.com/lemone)



Audio Glossary  
NCLEX-RN® Review  
Care Plan Activity: Perioperative Pacemaker Care  
Case Study: Myocardial Infarction  
Exercise: Match the Rhythm  
MediaLink Applications  
Links to Resources



## CHAPTER HIGHLIGHTS

- Atherosclerosis is the primary underlying process in coronary heart disease, impaired perfusion of myocardial tissue.
- The risk factors for coronary heart disease are those for atherosclerosis: age, gender, and genetic factors; hypertension, diabetes, abnormal blood lipids; cigarette smoking, obesity, physical inactivity, and diet; and emerging risk factors such as the metabolic syndrome and homocysteine levels.
- Smoking cessation, exercise, diet modification, weight loss, medications to achieve desired blood lipid values, and effective hypertension and diabetes management are the primary treatment measures for coronary heart disease.
- Atherosclerosis of coronary vessels impairs the supply of blood, oxygen, and nutrients to the myocardium. Myocardial ischemia results in the manifestations of coronary heart disease: angina pectoris, acute coronary syndrome, and myocardial infarction.
- Stable angina develops with a predictable amount of activity or stress, and typically follows an activity–pain, rest–relief pattern. Stable angina often can be managed effectively by medications and risk factor modification. The nursing focus is on education.
- Acute coronary syndrome or unstable angina is characterized by increasingly severe chest pain that occurs unpredictably. Acute coronary syndrome often requires aggressive interventions such as percutaneous coronary revascularization or coronary artery bypass surgery.
- Myocardial infarction, necrosis of myocardial tissue, results from complete blockage of a coronary artery, usually due to atherosclerotic plaque rupture and thrombus formation. Prompt restoration of blood flow through a revascularization procedure or administration of a fibrinolytic drug to dissolve the blood clot is necessary to preserve functional muscle tissue.
- The nursing focus for clients with acute coronary syndrome and myocardial infarction is on reducing myocardial work through measures such as pain relief and activity limitation, promoting blood flow and oxygenation through medication and oxygen administration and positioning, and early recognition and treatment of complications.
- Cardiac dysrhythmias may arise anywhere in conductive tissue of the myocardium. Dysrhythmias may be either benign or fatal, depending on their effect on cardiac output.
- Tachycardias increase the workload of the heart and may interfere with cardiac output if ventricular filling is impaired by the rapid rate.
- Bradycardias can affect cardiac output when the rate is too slow to meet the metabolic needs of the body.
- Atrial fibrillation is a common dysrhythmia that can lead to formation of blood clots within the heart and subsequent stroke if these clots lodge in cerebral blood vessels.
- Frequent ventricular dysrhythmias may indicate an increased risk for ventricular fibrillation and cardiac arrest.
- AV conduction blocks interfere with conduction of the sinus or atrial impulse through the AV node and to the ventricles.
- Although many antidysrhythmic medications are available, all increase the risk of dysrhythmia development, so they are used sparingly.
- The nurse's role in caring for clients with cardiac dysrhythmias focuses on prompt identification of the rhythm disruption, assessment of its effect on the client, administration of medications and other treatment measures, and institution of life support procedures as indicated.

## TEST YOURSELF NCLEX-RN® REVIEW

- 1 The nurse evaluates her teaching as effective when a client identifies which of the following modifiable risk factors for coronary heart disease (CHD) as contributing to the greatest extent?
  1. obesity
  2. diet
  3. smoking
  4. stress
- 2 When teaching a client about lovastatin (Mevacor), the nurse instructs the client to:
  1. promptly report muscle pain or tenderness to the physician.
  2. consume a diet that includes no more than 20% of calories from saturated fat.
  3. abstain from alcohol use while taking this drug.
  4. take the drug with meals to minimize gastric distress.
- 3 When assessing a client with stable angina, the nurse would expect to find:
  1. persistent ECG changes.
  2. correlation between activity level and pain.
  3. increasing nocturnal pain.
  4. evidence of impaired cardiac output such as weak peripheral pulses.
- 4 The nurse caring for a client with acute coronary syndrome identifies which of the following nursing diagnoses to be of highest priority?
  1. *Anxiety* related to unknown outcome of disorder
  2. *Ineffective Health Maintenance* related to lack of knowledge about coronary heart disease
  3. *Decreased Cardiac Output* related to myocardial ischemia
  4. *Ineffective Tissue Perfusion: Cardiopulmonary* related to underlying coronary heart disease
- 5 The nurse caring for a client returning from a coronary angioplasty with stent placement plans which of the following interventions?
  1. securing chest tubes to bedding
  2. maintaining leg extension on the affected side
  3. discontinuing intravenous lines when taking oral fluids
  4. treating chest pain with intravenous morphine as needed
- 6 In planning care for the client with acute myocardial infarction (AMI), the nurse identifies the highest priority goal of care as:
  1. stable ECG rhythm.
  2. ability to verbalize causes and effects of CHD.
  3. compliance with prescribed bed rest.
  4. relief of pain.
- 7 Which of the following nursing diagnoses is of highest priority for the client undergoing fibrinolytic therapy?
  1. *Ineffective Protection*
  2. *Ineffective Health Maintenance*
  3. *Risk for Powerlessness*
  4. *Anxiety*

- 8** In reviewing laboratory results for a client admitted with acute chest pain, the nurse is most concerned about which of the following?
1. hematocrit 35%
  2. AST 65 unit/L
  3. CK 320 unit/L
  4. APTT 35 seconds
- 9** The nurse recognizes second-degree AV block, type II (Mobitz II), and intervenes appropriately when he:
1. records the finding in the chart.
  2. prepares for temporary pacemaker insertion.
  3. administers a class IB antidysrhythmic drug.
  4. places the client in Fowler's position.

- 10** On identifying sinus bradycardia at a rate of 45 bpm, the nurse should:
1. assess mental status and blood pressure.
  2. assess peripheral pulses on all four extremities.
  3. determine if an apical-radial pulse deficit is present.
  4. prepare to administer intravenous atropine.

See Test Yourself answers in Appendix C.

## BIBLIOGRAPHY

- Alber, C. M., Chae, C. U., Grodstein, F., Rose, L. M., Rexrode, K. M., Ruskin, J. N., et al. (2003). Prospective study of sudden cardiac death among women in the United States. *Circulation*, 2003(207), 2096–2101. Retrieved from <http://www.circulationaha.org>
- American Heart Association. (2005a). 2005 Guidelines for CPR and ECC. *Circulation*, 112, IV1–IV205. Retrieved from [www.circulationaha.org](http://www.circulationaha.org)
- \_\_\_\_\_. (2005b). *Heart disease and stroke statistics—2005 update*. Dallas, TX: Author.
- Artinian, N. T. (2001). Perceived benefits and barriers of eating heart healthy. *MedSurg Nursing*, 10(3), 129–138.
- Aschcraft, A. S. (2004). Differentiating between pre-arrest and failure-to-rescue. *MedSurg Nursing*, 13(4), 211–215.
- Ayers, D. M. M. (2002). EBCT: Beaming in on coronary artery disease. *Nursing*, 32(4), 81.
- Blumenthal, R. S., & Margolis, S. (2005). *The Johns Hopkins white papers. Heart attack prevention*. Redding, CT: Medletter Associates.
- Braunwald, E., Antman, E. M., Beasley, J. W., Calif, R. M., Chaitlin, M. D., Hochman, J. S., et al. (2002). *ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients with Unstable Angina)*. Retrieved from [www.acc.org/clinical/guidelines/unstable/unstable.pdf](http://www.acc.org/clinical/guidelines/unstable/unstable.pdf)
- Chan, D. S., Chan, J. P., & Chang, A. M. (2005). Acute coronary syndromes: Cardiac rehabilitation programmes and quality of life. *Journal of Advanced Nursing*, 49(6), 591–599.
- Copstead, L. C., & Banasik, J. L. (2005). *Pathophysiology* (3rd ed.). St. Louis, MO: Saunders.
- Crane, P. B., & McSweeney, J. C. (2003). Exploring older women's lifestyle changes after myocardial infarction. *MedSurg Nursing*, 12(3), 170–176.
- De Luca, G., Suryapranata, H., Ottavanger, J. P., & Antman, E. M. (2004). Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: Every minute of delay counts. *Circulation*, March 16, 2004. Retrieved from [www.circulationaha.org](http://www.circulationaha.org)
- DeVon, H. A., & Zenwic, J. J. (2004). Differences in the symptoms associated with unstable angina and myocardial infarction. *Progress in Cardiovascular Nursing*, 19(1) 6–11.
- Dochterman, J., & Bulechek, G. (2004). *Nursing Interventions Classification (NIC)* (4th ed.). St. Louis, MO: Mosby.
- Eagle, K. A., Guyton, R. A., Davidoff, R., Edwards, F. H., Ewy, G. A., Gardner, T. J., et al. (2004). *ACC/AHA 2004 guideline update for coronary artery bypass graft surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines*. Retrieved from [www.acc.org/clinical/guidelines/cabg/cabg.pdf](http://www.acc.org/clinical/guidelines/cabg/cabg.pdf)
- Engler, M. B. (2004). Familial hypercholesterolemia: Genetic predisposition to atherosclerosis. *MedSurg Nursing*, 13(4), 253–257.
- Foley, S. (2005). Update on risk factors for atherosclerosis: The role of inflammation and apolipoprotein E. *MedSurg Nursing*, 14(1), 43–50.
- Glessner, T. M., & Walker, M. K. (2001). Standardized measures: Documenting processes and outcomes of care for patients undergoing coronary artery bypass grafting. *MedSurg Nursing*, 10(1), 23–29.
- Granger, B. B., & Miller, C. M. (2001). Acute coronary syndrome. *Nursing*, 31(11), 36–43.
- Granot, M., Goldstein-Ferber, S., & Azzam, A. S. (2004). Gender differences in the perception of chest pain. *Journal of Pain and Symptom Management*, 27(2), 149–155.
- Hart, P. L. (2005). Women's perceptions of coronary heart disease: An integrative review. *Journal of Cardiovascular Nursing*, 20(3), 170–176.
- Hummard, J. (2004). Management of atrial fibrillation. *Nursing Times*, 100(6), 42–44.
- Kasper, D. L., Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (2005). *Harrison's principles of internal medicine* (16th ed.). New York: McGraw-Hill.
- Kellen, J. C. (2004). Implications for nursing care of patients with atrial fibrillation: Lessons learned from the AFFIRM and RACE studies. *Journal of Cardiovascular Nursing*, 19(2), 128–137.
- McCance, K. L., & Huether, S. E. (2006). *Pathophysiology: The biologic basis for disease in adults and children* (5th ed.). St. Louis, MO: Mosby.
- McSweeney, J. C., & Coon, S. (2004). Women's inhibitors and facilitators associated with making behavioral changes after myocardial infarction. *MedSurg Nursing*, 13(1), 49–56.
- Meeker, M. H., & Rothrock, J. C. (1999). *Alexander's care of the patient in surgery* (11th ed.). St. Louis, MO: Mosby.
- Miller, K. H., & Grindel, C. G. (2004). Comparison of symptoms of younger and older patients undergoing coronary artery bypass surgery. *Clinical Nursing Research*, 13(3), 179–193.
- Milner, K. A., Funk, M., Richards, S., Vaccarino, V., & Krumholz, H. M. (2001). Symptom predictors of acute coronary syndromes in younger and older patients. *Nursing Research*, 50(4), 233–241.
- Moorhead, S., Johnson, M., & Maas, M. (2004). *Nursing outcomes classification (NOC)* (3rd ed.). St. Louis, MO: Mosby.
- NANDA International. (2005). *NANDA's nursing diagnoses: Definitions & classification 2005–2006*. Philadelphia: Author.
- National Cholesterol Education Program. (2002). *Third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Final report*. Bethesda, MD: National Institutes of Health.
- National Heart, Lung, and Blood Institute (NHLBI). (2004). *Morbidity & mortality: 2004 chart book of cardiovascular, lung, and blood diseases*. Bethesda, MD: National Institutes of Health.
- \_\_\_\_\_. (2005). Statement from Elizabeth G. Nabel, M.D., director of the National Heart, Lung, and Blood Institute of the National Institutes of Health on the findings of the Women's Health Study. *NIH News*, March 7. Retrieved from [www.nhlbi.nih.gov](http://www.nhlbi.nih.gov)
- National High Blood Pressure Education Program. (2004). *The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*. Bethesda, MD: National Heart, Lung, and Blood Institute, National Institutes of Health.
- Nicholls, C., & Sani, M. (2004). Antiplatelet therapy. *Nursing of Older People*, 16(8), 35–36.
- Obias-Manno, D., & Wijetunga, M. (2004). Risk stratification and primary prevention of sudden cardiac death: Sudden death prevention. *AACN Clinical Issues*, 15(3), 404–418.
- Palatnik, A. M. (2001). Critical care. Acute coronary syndrome: New advances and nursing strategies. *Nursing*, 31(5), 32cc1–32cc2, 32cc4, 32cc6.
- Paparella, S. (2004). Fibrinolytic therapy: No room for error. *Journal of Emergency Nursing*, 30(4), 348–350.
- Pelter, M. M., & Adams, M. G. (2004). Premature beats. *American Journal of Critical Care*, 13(6), 519–520.
- Porth, C. M. (2005). *Pathophysiology: Concepts of altered health states* (7th ed.). Philadelphia: Lippincott.
- Quigley, M. P. (2004). Promoting cardiac rehabilitation. *Nursing*, 34(8), 24.
- Ridker, P. M., Cook, N. R., Lee, I., Gordon, D., Gaziano, T. A., Manson, J. E., et al. (2005). A randomized trial of low-dose aspirin in the primary prevention of cardiovascular disease in women. *New England Journal of Medicine*, 352. Retrieved from <http://content.nejm.org/cgi/content/full/352/13/1293>
- Rosenfeld, A. G. (2004). Treatment-seeking delay among women with acute myocardial infarction: Decision trajectories and their predictors. *Nursing Research*, 53(4), 225–236.
- Ryan, C. J., DeVon, H. A., & Zenwic, J. J. (2005). Typical and atypical symptoms: Diagnosing acute coronary syndromes accurately. *American Journal of Nursing*, 105(2), 34–36.
- Sanders, A. B., & Ewy, G. A. (2005). Cardiopulmonary resuscitation in the real world: When will the guidelines get the message? *JAMA*, 293(3), 363–365.
- Shaffer, R. S. (2002). ICD therapy: The patient's perspective. *American Journal of Nursing*, 102(2), 46–49.
- Stewart, S. (2004). Epidemiology and economic impact of atrial fibrillation. *Journal of Cardiovascular Nursing*, 19(2), 94–102.
- Tierney, L. M., McPhee, S. J., & Papadakis, M. A. (2005). *Current medical diagnosis & treatment* (44th ed.). New York: McGraw-Hill.
- Urden, L. D., Stacy, K. M., & Lough, M. E. (2006). *Thelan's critical care nursing: Diagnosis and management* (5th ed.). St. Louis, MO: Mosby.
- U.S. Department of Health & Human Services. (2005). *Dietary guidelines for Americans 2005*. Washington, DC: U.S. Department of Agriculture. Retrieved from [www.healthier.us.gov/dietaryguidelines](http://www.healthier.us.gov/dietaryguidelines)
- U.S. Preventive Services Task Force. (2002a). Aspirin for the primary prevention of cardiovascular events: Recommendations and rationale. *American Journal of Nursing*, 102(3), 67, 69–70.
- \_\_\_\_\_. (2002b). Screening for lipid disorders in adults: Recommendations and rationale. *American Journal of Nursing*, 102(6), 91, 93, 95.
- Way, L. W., & Doherty, G. M. (2003). *Current surgical diagnosis and treatment* (11th ed.). New York: McGraw-Hill.
- Wilkinson, J. M. (2005). *Nursing diagnosis handbook with NIC interventions and NOC outcomes* (8th ed.). Upper Saddle River, NJ: Prentice Hall Health.
- Wolf, Z. R., Miller, P. A., & Devine, M. (2003). Relationship between nurse caring and patient satisfaction in patients undergoing invasive cardiac procedures. *MedSurg Nursing*, 12(6), 391–396.
- Woods, S. L., Froelicher, E. S., Motzer, S. A., & Bridges, E. (2004). *Cardiac nursing* (5th ed.). Philadelphia: Lippincott.
- Writing Group for the Women's Health Initiative Investigators. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women. [On-line]. *JAMA*, 288(3). Retrieved from <http://jama.ama-assn.org/issues/v288n3/ffull/joc21036.html>
- Wung, S. (2002). Genetic advances in coronary artery disease. *MedSurg Nursing*, 11(6), 296–300.
- Zenwic, J. J., & Ryan, C. J. (2004). Delays in seeking MI treatment. *American Journal of Nursing*, 104(1), 81–83.