Coronary Artery Disease and Dysrhythmias

OBJECTIVES

After studying this chapter, the learner should be able to:

1. Discuss the role of risk factors in the pathogenesis of coronary artery disease.
2. Recognize the signs and symptoms of coronary artery disease.
3. Explain the collaborative management of stable angina pectoris and acute coronary syndromes.
4. Discuss the nursing role in the care of the patient with coronary artery disease.
5. Recognize common dysrhythmias associated with the cardiac conduction system.
6. Discuss the collaborative care management of patients with cardiac dysrhythmias.
7. Describe the basic components of cardiopulmonary resuscitation.

KEY TERMS

angina, p. 746
antithrombotic, p. 759
antiplauelet agent, p. 757
atrioventricular block, p. 787
automaticity, p. 773
biomarkers, p. 755
bradycardia, p. 776
cardioversion, p. 789
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dyslipidemia, p. 748
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reentry, p. 775
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telemetry, p. 789
tachycardia, p. 776
thrombolytics, p. 757

Patients with coronary artery disease (CAD) often seek health care after experiencing angina or myocardial infarction (MI). CAD is directly implicated in other cardiovascular diagnoses such as dysrhythmias, heart failure, and cardiomyopathy. All nurses need to be familiar with the collaborative care management of CAD because of its high prevalence in the industrialized world. This chapter discusses the origins and management of CAD. It also discusses the recognition and management of common dysrhythmias.

Coronary Artery Disease

Etiology and Epidemiology. Coronary heart disease (CHD), which encompasses acute MI, angina pectoris, atherosclerotic cardiovascular disease, and all other forms of acute and chronic ischemic heart disease, is the leading cause of death in the industrialized Western world, accounting for one of every five deaths in 2001. It is estimated that 13,200,000 Americans have CHD; 7,800,000 have experienced MI; and 6,800,000 have angina. Approximately 865,000 Americans experienced a new or recurrent MI in 2001, with 184,757 deaths. CHD remains the number one health problem in the United States and the leading cause of premature, permanent disability.6

CAD is a generic designation for many different conditions that involve obstructed blood flow through the coronary arteries. The most prevalent etiologies of CAD are atherosclerosis, coronary vasospasm, and microvascular angina. Microvascular angina results from poor function of the smaller blood vessels that supply the heart. Atherosclerosis is by far the most common cause of CAD and is the focus of this chapter.

Both individual risk factors and the presence of concurrent disease states influence the incidence of CAD. Some populations have an increased occurrence of CAD because of definable characteristics or risks. Risk factors are classically categorized as nonmodifiable and modifiable. Nonmodifiable risk factors include age, gender, race, and family history and genetics. Modifiable risk factors include diabetes, hypertension, tobacco use, sedentary lifestyle, obesity, and stress (see Risk Factors box). The American Heart Association (AHA) has not firmly established hyperhomocysteinemia as an independent risk factor; however, it has established guidelines for monitoring homocysteine levels in high-risk individuals.15 C-reactive protein, another measure of inflammation, is also considered a marker for an increased risk of cardiovascular disease and of adverse outcomes in patients with acute coronary syndrome (ACS). People in a high-risk group have about a
twofold increase in relative risk for cardiovascular disease compared with those in a low-risk group. Current guidelines suggest that highly sensitive C-reactive protein can be used as an independent marker of risk, but should not yet be used for mass screening or to guide therapy.20 Table 29-1 links the major risk factors for CAD with their specific physiologic effects.

**AGE AND GENDER.** Clinical evidence of CAD is rarely apparent before the second and third decades of life, but CAD is already a leading cause of mortality in men 35 to 45 years old. Overall, CHD makes up more than half of all cardiovascular events for persons less than 75 years of age. The average age of a person having a first heart attack is 65.8 for men and 70.4 for women. Eighty-four percent of individuals who die of CHD are 65 years of age or older. However, about 80% of CHD deaths in people under age 65 occur during the first attack.7 The incidence of CAD in women significantly increases after menopause, and one in three women over age 61 has some form of CAD. The theoretical cardioprotective benefits of estrogen stimulated a wide variety of research regarding the effects of hormone replacement therapy (HRT). Unfortunately, researchers through large-scale studies instead found an increase in cardiovascular events in women taking HRT. Therefore HRT is no longer recommended for preventing heart disease in women. With increasing longevity in the Western world, the incidence of CAD among both male and female octogenarians and nonagenarians also will increase.

**RACE.** CAD is nondiscriminatory, affecting all races, but the independent role of race in the development of CAD is unclear. Other risk factors such as hypertension, obesity, lifestyle (including cultural practices), ethnic traditions, access to health care, and individual choices may play a more significant role in the development of CAD than race alone.

**FAMILY HISTORY AND GENETICS.** The likelihood that an offspring will have CAD increases if the biologic parent manifests CAD before the age of 55, but it is difficult to determine the

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Physiologic Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age and gender</td>
<td>Decrease in elasticity of arteries with age</td>
</tr>
<tr>
<td>Heredity: family history of coronary artery disease</td>
<td>Undetermined—genetic research pending</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Damage to intima</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Decreased elasticity of blood vessels</td>
</tr>
<tr>
<td>Tobacco use (nicotine)</td>
<td>Decreased high-density lipoproteins</td>
</tr>
<tr>
<td>Sedentary lifestyle</td>
<td>Altered lipid metabolism</td>
</tr>
<tr>
<td>Hypercholesterolemia, familial hyperlipidemia</td>
<td>More substrate provided for lesion formation</td>
</tr>
</tbody>
</table>

**TABLE 29-1 Role of Risk Factors in Coronary Artery Disease**
Hg diastolic as prehypertension. Hypertension affects the abilities of blood pressures of 120 to 139 mm Hg systolic and 80 to 89 mm Hg diastolic, a consequence of peripheral insulin resistance, can occur up to a decade before hyperglycemia is even diagnosed. Elevated levels of circulating insulin may begin the process of atheroma formation by initiating damage to the arterial intima. Impaired insulin regulation is associated with a variety of atherosomatic processes, including elevated triglycerides, decreased high-density lipoprotein (HDL) levels, elevated very-low-density lipoprotein (VLDL) levels, coagulation disorders, increased vascular resistance, obesity, and hypertension.

Hypertension, defined as a measured elevation in blood pressure above 140/90 mm Hg on at least three occasions, increases the incidence of CAD twofold to threefold. National Heart, Lung, and Blood Institute guidelines define blood pressures of 120 to 139 mm Hg systolic and 80 to 89 mm Hg diastolic as prehypertension. Hypertension affects the ability of the blood vessel to constrict and dilate. Shearing forces on the intimal lining caused by hypertension predispose the artery to atherosclerosis. In addition, the heart must work harder to pump against an increased resistance to blood flow. Adequate control of hypertension with medication and lifestyle modifications may decrease the incidence of CAD in the hypertensive population.

Tobacco. The risk of death from CAD is significantly higher in smokers than in nonsmokers, and the risk is proportional to the amount of tobacco used. In addition, approximately 35,000 nonsmokers die from CHD yearly secondary to environmental smoke. Cigarette smokers have the highest incidence of CAD, however, pipe and cigar smokers, as well as tobacco chewers, also have an increased risk of developing CAD compared with nonsmokers.

Sedentary Lifestyle. In 1996 the surgeon general released a seminal report on physical activity and health. This report noted that the incidence of CAD is higher in individuals who do not participate in regular physical activity compared with those who exercise. Exercise is associated with a decrease in total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides. Up to 60% of U.S. adults reported no pattern of regular exercise during the study period in the early 1990s. The Centers for Disease Control and Prevention (CDC) data collected during 2000 and 2001 showed that these numbers had not yet improved, with 54.6% of Americans ages 18 and older considered not active enough to meet physical activity recommendations.

Dyslipidemia. Research findings consistently report an association between abnormal blood cholesterol levels (dyslipidemia) and CAD. In 2001, 50.7% of the U.S. population had total cholesterol levels greater than 200 mg/dl; 45.8% had LDL cholesterol levels greater than 130 mg/dl, and 26.4% had HDL cholesterol levels less than 40 mg/dl. LDLs are the most atherogenic of the lipid compounds, transporting 60% to 70% of the body's cholesterol. An increased triglyceride level, in combination with a high LDL level, is also a strong predictor of heart disease and MI. Research also indicates that elevated plasma lipoprotein (a) levels are predictive of premature CAD in men. Hyperlipidemia may be either primary (familial) or secondary to some other process, such as concomitant disease states (e.g., diabetes) or lifestyle factors, such as diet, sedentary activity levels, and smoking. Excess lipids in the circulation result in endothelial injury and increase the available substrate for foam cell production, an early step in the development of atherosclerotic lesions.

Obesity. Obesity is also associated with an increased risk of cardiovascular disease. Sixty-four percent of the U.S. adult population was defined as overweight in 2001, and 15.3% of children ages 6 to 11 were defined as overweight. Although obesity is commonly cited as a significant coronary risk factor, the extent to which it has an independent effect in predisposing a person to CAD is controversial. But obese persons are more prone to glucose intolerance, hypertension, elevated triglycerides, and low levels of HDL. In addition, obese individuals often demonstrate other behaviors, such as sedentary lifestyles, that are known risk factors for CAD.

Stress. Much discussion has taken place over the years about the relationship between stress and CAD. Catecholamines, released during the stress response, increase platelet aggregation and may also precipitate vasoospasm. A complete understanding of the effects of stress on circulation, lipid metabolism, and coagulability still requires additional research.

Homocysteine. Homocysteine is an amino acid synthesized during protein catabolism by the conversion of methionine to cysteine. Homocysteine is believed to contribute to vascular disease by altering coagulation, activating the inflammatory response, and contributing to endothelial dysfunction. The metabolism of homocysteine depends on vitamin B12, folate, and vitamin B6. Levels of homocysteine greater than 15 μmol/L are predictive of increased mortality and morbidity. The AHA recommends screening for total homocysteine in patients with a personal or family history of premature cardiovascular disease.

Pathophysiology. CAD refers to the development and progression of plaque accumulation in the coronary arteries. Figure 29-1 illustrates the dynamic nature of CAD. Stages along the continuum are stable angina and ACS; the most severe presentation is MI. A patient with CAD may seek treatment at any point along this continuum and may move back and forth along the continuum over time.

Stable Angina. The coronary arteries are small arteries that provide oxygen to the beating heart, a surface that is constantly moving (see Chapter 28). The arteries lie on the epicardial surface...
endothelial injury. The body’s response to the injury involves a complex interplay of chemical mediators designed to protect the area.

Platelets adhere to the collagen and release adenosine diphosphate (ADP). Circulating platelets with ADP-specific surface receptors become activated and bind to the released ADP. Endothelial injury also triggers the release of thromboxane A2, which causes local vasoconstriction to minimize the extent of injury and further stimulates platelet aggregation (Figure 29-2). Endogenous nitric oxide acts to protect the artery through vascular relaxation.

The intima also releases prostacyclin in response to the effects of thromboxane A2. Prostacyclin works to restore equilibrium by stimulating platelet aggregation. Endogenous nitric oxide acts to protect the artery through vascular relaxation.

Continuum of coronary artery disease. Arrow depicts increased severity of continuum to the right.

**Acute Coronary Syndrome.** Atherosclerosis may remain stable if the blockage in the coronary artery does not progress beyond 70%; if collateral (alternate) vessels develop to supply the myocardium; and, most important, if the fibrous cap remains intact. Inflammation plays a critical role in plaque destabilization. Lipoprotein-associated phospholipase A2 (Lp-PLA2) hydrolyzes oxidized LDL, generating proinflammatory mediators that increase adhesion molecules, cytokine production, and the migration of monocytes into the intima. Monocytes differentiate into macrophages that engulf oxidized LDLs to become foam cells. Pressure within the lesion (plaque) can increase adhesion molecules, connective tissue enzymes that break down collagen, weakening the fibrous cap. Activated macrophages also cause the secretion of connective tissue enzymes that break down collagen, weakening the fibrous cap.20,21 Smaller, soft, lipid-rich lesions appear to be the most likely to rupture. Rupture of the fibrous cap exposes the inner plaque to the circulating blood, activating clotting factors and causing both collagen accumulation and smooth muscle cell proliferation (Figure 29-3). The process of platelet activation is once again initiated to seal the rupture.

The presence of certain risk factors also contributes to this destructive pathophysiologic process. Nicotine from tobacco use increases platelet adhesion and increases the potential for clotting at the site of disruption. Catecholamines released during the stress response also increase platelet aggregation.

Plateau rupture has several possible outcomes (Figure 29-4, A and B). The area can heal over with the platelet plug absorbed into the plaque under a new cap, in which case the larger plaque further narrows the vessel lumen and may precipitate symptoms. The second outcome leaves a residual fibrous clot extending into the lumen, partially obstructing the artery. Endothelial injury also causes the release of leukocyte-soluble adhesion molecules and chemotactic factors. These factors mediate the attachment of monocytes to endothelial cells and encourage monocyte migration into the subintima. Smooth muscle cells and fibrous tissue then form a fibrous cap over the fatty streak. The fatty streak continues to grow, accumulating macrophages, mast cells, and activated T cells and invading both the intima and media. Involvement of the media affects the vessel wall’s ability to vasodilate and vasoconstrict. The artery continues to supply oxygen and nutrients to the myocardium as long as the blockage is less than 70% of the arterial lumen. Stable plaques may even occlude the coronary artery by more than 70% and still not cause symptoms.
death from dysrhythmias. Figure 29-5 illustrates the spiraling series of events that occurs in the cardiovascular system from myocardial ischemia.

The body activates the process of fibrinolysis to lyse the clot and restore blood flow. However, if clot lysis does not immediately restore blood flow, ischemia continues in the area of myocardium distal to the obstruction. Time is a critical factor in this scenario. Ongoing myocardial ischemia for 20 minutes or longer can result in tissue death. This is termed *acute myocardial infarction* (AMI). A zone of ischemia, made up of potentially viable tissue, surrounds the infarcted area of myocardium. The final size of an infarct depends on whether this marginal area in the ischemic zone succumbs to the effects of prolonged ischemia (Figure 29-6). The entire thickness of the myocardium may not become

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**Figure 29-5** Illustration of the cardiovascular system. **A**, Diseased coronary arteries. **B**, Normal coronary arteries. **C**, Atheromatous plaques. **D**, Atheromatous plaques with calcification.

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**Figure 29-2** Progression of atherosclerosis. **A**, Thromboxane stimulates platelet aggregation. Inflammatory response initiates monocyte activity. **B**, Medial smooth muscle migrates into intima, increasing permeability of the wall to cholesterol. **C**, Fibrous cap seals plaque. **D**, Macrophages secrete enzymes that weaken fibrous cap. Rupture of plaque stimulates thrombus formation and acute coronary syndrome.
Coronary Artery Disease and Dysrhythmias

CHAPTER 29

Platelet adhesion

Platelet aggregation

Thromboplastin

Prothrombin

Thrombin

Fibrin (insoluble)

Fibrinogen (soluble)

Thrombus

Figure 29-3 Process of thrombogenesis.

Atherosclerotic plaque

"rupture"

Thrombogenesis

Figure 29-4 Possible pathophysiologic scenarios after plaque fissure. A, Clot is resorbed into plaque, healing over area of fissure, but with smaller lumen resulting. B, Clot remains at site of fissure, decreasing lumen diameter. C, Clot extends into lumen, completely obstructing lumen (myocardial infarction).

Myocardial ischemia

↑ Myocardial oxygen demand

↓ Myocardial oxygen supply

↑ Myocardial contractility

↓ Myocardial contractility

↓ Coronary perfusion

↑ Diastolic filling

↓ Afterload

↑ Heart rate

Peripheral vasoconstriction

Sympathetic response

Stimulation of baroreceptors

↑ Myocardial oxygen demand

↓ Myocardial oxygen supply

↑ Myocardial contractility

↓ Myocardial contractility

↓ Coronary perfusion

↑ Diastolic filling

↓ Afterload

↑ Heart rate

Peripheral vasoconstriction

Sympathetic response

Stimulation of baroreceptors

Figure 29-5 Effects of prolonged myocardial ischemia.
ischemic or infarcted if some blood is able to reach the area. However, the potential for further damage remains as long as the coronary artery lumen is atherosclerotic.

Infarctions are classified according to their anatomic location (Table 29-2). The left anterior descending (LAD) artery supplies the anterior surface of the left ventricle and the bundle branches of the conduction system. This area of the heart is responsible for most of the contractility necessary to eject blood into the aorta. This portion of the heart requires a substantial source of oxygen to generate the force needed to pump against the aorta’s high-pressure system. Lesions in the LAD artery that lead to anterior infarctions are often associated with a decrease in contractility and cardiac output that results in heart failure. Sudden death secondary to ventricular dysrythmias may also occur.

The right coronary artery supplies the inferior surface of the left ventricle, the entire right ventricle, and both the sinoatrial (SA) and atrioventricular (AV) nodes in most individuals. Inferior infarctions or right ventricular infarctions may be complicated by transient or permanent heart blocks or right-sided heart failure.

The circumflex artery most often supplies the lateral surface of the left ventricle. Obstruction affecting only this area is often well tolerated. Infarctions that extend through the full thickness of the ventricular wall and exhibit pathologic Q waves on the electrocardiogram (ECG) are termed Q wave infarctions.

The patient with CAD usually seeks health care during an episode of ischemia or after an ischemic event. Many patients experience the classic midsternal chest pain; however, a number of patients instead complain of indigestion, “heartburn,” left arm pain, or pain radiating from the chest to the scapula, neck, jaw, or the left or right arm. Women often experience “atypical” symptoms such as chest heaviness, heartburn, fatigue, or shortness of breath (see Clinical Manifestations box). The occurrence of angina is often perceived as sudden; however, some individuals may perceive it as gradual, especially if the initial intensity was mild.

The classic location of ischemic pain is retrosternal. The pain may radiate down the left arm or both arms, upward to the neck or jaw, or backward to the scapular region. Some patients do not experience pain at all, a condition called silent ischemia. This is especially true for elderly patients or patients with diabetes because of alterations in sensory perception. Therefore the quality and intensity of pain may be an unreliable indicator of the severity of ischemia. For example, some patients with MI describe the pain as “mild indigestion” or “tightness,” whereas others describe the pain as excruciating and viselike.

Symptoms of stable angina are often of short duration, ending when the demand for oxygen is decreased. Symptoms of unstable angina are of longer duration and usually require intervention. Symptoms of MI continue until blood flow is restored or the myocardium dies.

In addition to chest pain, patients may complain of dizziness, dyspnea, nausea, vomiting, or anxiety. Patients experiencing an AMI often report a feeling of doom or as though they are “going to die.” Changes in vital signs may include tachycardia or bradycardia, increased or decreased blood pressure, and shortness of breath. Dysrythmias may develop from myocardial ischemia, and decreased cardiac output can result in classic shock symptoms such as pale, cool, diaphoretic skin.

Precipitating factors for stable angina symptoms include any circumstance that increases myocardial oxygen demand, such as exercise, stress, sexual intercourse, and smoking. ACS may have

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**Table 29-2 CORRELATION OF CORONARY ARTERY WITH AFFECTED MYOCARDIUM**

<table>
<thead>
<tr>
<th>Coronary Artery</th>
<th>Structure Supplied</th>
<th>Potential Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left anterior descending</td>
<td>Anterior surface of left ventricle</td>
<td>Bundle branch blocks</td>
</tr>
<tr>
<td></td>
<td>Ventricular septum</td>
<td>Left-sided heart failure</td>
</tr>
<tr>
<td></td>
<td>Bundle branches of conduction system</td>
<td>Rupture of septum</td>
</tr>
<tr>
<td>Circumflex</td>
<td>Lateral and posterior surfaces of left ventricle</td>
<td>When circumflex artery supplies SA node, bradydysrhythmias a possibility</td>
</tr>
<tr>
<td></td>
<td>Sinotral (SA) node (45% of people)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atrioventricular (AV) node and bundle of His (10% of people)</td>
<td></td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>Right atrium</td>
<td>Bradydysrhythmias and heart blocks</td>
</tr>
<tr>
<td></td>
<td>Right ventricle</td>
<td>Right-sided heart failure</td>
</tr>
<tr>
<td></td>
<td>Posterior surface of left ventricle</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SA node (55% of people)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>AV node (90% of people)</td>
<td></td>
</tr>
</tbody>
</table>
Dysrhythmias. Dysrhythmias often occur secondary to the ischemic processes of CAD. Ischemia alters the stability of the myocardial cell membrane, and ischemia of the specialized conduction pathways (SA node, AV node, and bundle branches) can result in heart blocks. Individuals with right coronary artery blockages and inferior MIs may experience heart block and bradycardia, since the right coronary artery most often supplies the SA and AV nodes. Patients with LAD artery blockages may have complete or incomplete bundle branch blocks and ventricular dysrhythmias, since the LAD artery supplies the bundle branch system and a disproportionately large surface of the anterior myocardium. Direct damage to the myocardial cell creates electrolyte imbalances that alter the cells’ action potential. Dysrhythmias are not usually treated unless they are considered hemodynamically significant. Management of common dysrhythmias is presented later in the chapter.
**Pericarditis.** After an AMI the heart’s pericardial lining can become inflamed, and fluid may accumulate between the pari-
etal and visceral layers. The patient complains of severe precor-
dial chest pain that closely resembles that of the original infar-
tion. The presence of a characteristic pericardial friction rub is
helpful in making the differential diagnosis. Pericarditis is usual-
lly treated with nonsteroidal antiinflammatory drugs or occa-
sionally corticosteroids. Pericarditis is presented in greater detail
in Chapter 30.

**Collaborative Care Management**

**Diagnostic Tests.** When a patient has signs or symptoms of
CAD, a variety of diagnostic tests are used to confirm the diag-
osis of MI and to guide therapeutic options (Box 29-1).

**Electrocardiography.** The ECG remains a critical tool in
diagnosing CAD and is most useful while the patient is sympto-
matic. Because the ECG represents only one point in time, serial
12-lead ECGs, continuous monitoring, or both are the standard of
care for the evaluation of chest pain. ST segment elevation is the
hallmark of acute myocardial ischemia that is progressing toward
infarction.25 (Figure 29-7). ST elevation resolves when blood flow is
restored or the MI is complete. If the full thickness of the myocardi-
um becomes necrotic, significant Q waves evolve over the next
week. Future ECGs continue to show the Q wave, indicating that
the patient suffered an MI in the past. When only the subendocar-
dial surface infarcts (non–Q wave MI), Q waves do not develop.
T wave abnormalities such as T wave inversion may also occur at
the time of acute infarction. Non–ST segment elevation MI
(NSTEMI) refers to an ACS that does not cause ST elevation but
does produce elevated serum troponin levels. Normal or nonspecif-
ic findings on ECG do not always exclude the possibility of MI.

Gender and ethnicity affect ECG interpretation in subtle
ways. Women with an MI may not exhibit the dramatic ST ele-
vation of acute injury, perhaps because of less cardiac muscle
mass, estrogens, and dampening of the ECG signal by breast tis-
sue. Early repolarization patterns and ST segment elevation at
the J point (the point where the QRS complex ends and the ST
segment begins) are more prevalent in African-Americans. Com-
parison with the patient’s prior ECG, when possible, helps with
the differential diagnosis. Acute pericarditis, digitalis effects, elec-
trolyte imbalances, hypothermia, subarachnoid hemorrhages, and
ventricular hypertrophy may all affect the ST segment and
should be considered with the patient’s presentation. The pres-
ence of a left bundle branch block creates additional challenges
to ECG interpretation.

The 12-lead ECG represents 12 different anatomic views of
the myocardium (see Chapter 28). ST changes occur in leads that
are specific to the area of myocardium involved. Table 29-3 shows
the relationship of specific leads to the affected area of myocardi-
um. Additional leads are necessary to reveal damage to the right
ventricle or posterior wall of the left ventricle. These 15- and 18-
lead ECGs use the right chest wall and posterior thorax sites for
localizing damage to the myocardium.

ST segment monitoring is an important part of patient moni-
toring to detect ischemia in patients who are seen with ACS and

**Box 29-1 Diagnostic Tests for Coronary Artery Disease**

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>12-Lead Electrocardiogram</strong></td>
<td>Serial tests or continuous monitoring. ST segment elevation is a critical marker for myocardial ischemia progressing to infarction. Elevation of greater than 1 mm in 2 contiguous leads plus the presence of new Q waves indicate a high probability of myocardial infarction (MI). ST depression reflects ischemia that may resolve with improved perfusion. Normal or nonspecific findings do not rule out the possibility of MI.</td>
</tr>
<tr>
<td><strong>Serum Biomarkers</strong></td>
<td><strong>Serum Troponin</strong> Composed of three proteins: troponin C, troponin I, and troponin T. Levels are normally undetectable. Myocardial damage causes levels of troponin I to rise within 3 hours. Levels remain elevated for up to 7 days (see Table 29-4). <strong>Creatine Kinase</strong> Conforms the presence of myocardial damage. Levels rise within 3 to 9 hours and return to normal in 1 to 3 days. Levels decrease at 12 hours and help determine the endpoint of myocardial damage and the presence of reinfarction. <strong>Myoglobin</strong> Levels increase within 1 hour and return to normal within 24 to 36 hours. <strong>Erythrocyte sedimentation rate</strong> May remain elevated for several weeks. <strong>C-Reactive Protein</strong> Levels are normally undetectable. Elevation may last 3 to 7 days.</td>
</tr>
</tbody>
</table>
those who receive thrombolytic therapy or coronary interventions. ST segment monitoring is also useful in detecting silent ischemia. Research supports the value of ST segment monitoring in ACS (see Research box).

**BLOOD TESTS.** **Biomarkers** provide definitive information about the presence and severity of myocardial damage and are drawn immediately in patients experiencing unrelenting chest pain. Biomarkers are especially valuable in evaluating patients who are seen for possible thrombolytic therapy. The most specific biomarker for MI is serum troponin, which is composed of troponin C, troponin I, and troponin T. Any elevation of serum troponin indicates myocardial cell damage. Cardiac troponin I that is already elevated on admission is associated with an increase in both complications and mortality (Table 29-4). However, elevated troponin levels may also reflect minor myocardial injury from causes other than ACS.

Injured myocardial cells release another biomarker, the enzyme creatine kinase (CK), during AMI. CK elevation confirms the presence of myocardial damage. Brain tissue and skeletal muscle also release CK with injury, but the isoenzyme CKMB is specific to the myocardium. Myoglobin, an oxygen-binding protein found in cardiac and skeletal muscle, is another early biomarker for MI.

Blood chemistry tests and a complete blood count (CBC) are performed to determine concurrent disease states and help with differential diagnosis. C-reactive protein, another measure of inflammation, is also considered a marker for an increased risk of cardiovascular disease. The AHA and CDC have established risk guidelines for C-reactive protein as follows: concentrations of less than 1.0 mg/L are considered low risk, 1.0 to 3.0 mg/L are average risk, and higher than 3.0 mg/L are high risk. People in the high-risk group have about a twofold increase in relative risk for cardiovascular disease compared with those in the low-risk group.

**Stress Testing and Echocardiography.** Stress testing is a noninvasive test that highlights areas of the myocardium that do not receive adequate perfusion at peak exercise and relates the significance of coronary artery blockages to the patient’s functional status. The ECG tracings recorded during the exercise component of the test can also indicate which coronary arteries might be involved. Echocardiography is another noninvasive test that may be used (see Chapter 28).
The AHA recommends diagnostic catheterization in the presence, severe, or prolonged (longer than 20 minutes) ischemic medical management, and for patients with one or more recurred for patients who have recurrent symptoms despite intensive cardiac catheterization.

**ADDITIONAL NONINVASIVE STUDIES.** Radionuclide myocardial perfusion imaging has been helpful in diagnosing AMI in the emergency room. Normal resting perfusion imaging studies have been used to exclude MI and avoid unnecessary hospitalizations. Options for scanning are listed in Box 29-1.

**CARDIAC CATHETERIZATION.** Cardiac catheterization is indicated for patients who have recurrent symptoms despite intensive medical management, and for patients with one or more recurrent, severe, or prolonged (longer than 20 minutes) ischemic episodes. The AHA recommends diagnostic catheterization in the following emergent or urgent situations: candidates for primary or rescue percutaneous coronary interventions (PCI), patients with cardiogenic shock who are candidates for revascularization, and patients with spontaneous episodes of myocardial ischemia or episodes of myocardial ischemia provoked by minimal exertion during recovery from STEMI.

Right-sided heart catheterization provides information on the heart’s hemodynamic status. Left-sided heart catheterization includes coronary angiography and left ventriculography, and visualizes the coronary arteries, as shown in Figure 29-8 (see Chapter 28). Fluoroscopic imaging allows direct visualization of the contractility of the left ventricle. Ventriculography can identify areas of poor contractility (hypokinesis), overcompensation (hyperkinesis), nonmovement (akinesis), and asynergy (dyskinesis). An infarcted area is usually akinetic.

**MEDICATIONS.** Box 29-2 lists the basic principles of CAD management in an A-E format, and drug therapy plays a major role. Figures 29-9 and 29-10 outline treatment algorithms for the management of stable angina and ACS that incorporate both drug therapy and risk factor modification. An overview of medications commonly used to treat CAD is found in Table 29-5.

<table>
<thead>
<tr>
<th>Cardiac Enzyme</th>
<th>Elevation (hr)</th>
<th>Elevation (hr)</th>
<th>Duration (hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatine kinase MB</td>
<td>3-5</td>
<td>12-18</td>
<td>1-3</td>
</tr>
<tr>
<td>Troponin T</td>
<td>3-6</td>
<td>10-24</td>
<td>7</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3-5</td>
<td>10-24</td>
<td>10-14</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1</td>
<td>4-6</td>
<td>1-2</td>
</tr>
</tbody>
</table>

**TABLE 29-4 CARDIAC BIOMARKER LEVELS IN ACUTE MYOCARDIAL INFARCTION**

Figure 29-8 Coronary arteriogram showing coronary artery thrombus (arrow) in patient with unstable angina.

**Box 29-2 Guidelines for Management of Stable Angina**

- Aspirin and antianginals
- Beta-blocker and blood pressure
- Cholesterol and cigarettes
- Diet and diabetes
- Education and exercise

ANTIPLATELET AGENTS. Aspirin is the primary antiplatelet agent used in the prevention and treatment of CAD. Aspirin is given in the emergency department (or in the prehospital setting) to any patient suspected of having an MI. Aspirin blocks the formation of thromboxane A2, inhibiting platelet aggregation; research has demonstrated that a single daily dose of 81 mg (one baby aspirin) can effectively sustain the desired antiplatelet effect. Enteric-coated forms can be prescribed for individuals who cannot tolerate pure aspirin.

Thienopyridines have an irreversible effect on the platelets that is sustained for the life of the platelet but takes several days to become manifest. Clopidogrel (Plavix) prevents platelet activation by blocking ADP-induced platelet binding. It is used for individuals who cannot tolerate aspirin and for patients undergoing PCIs. Ticlopidine (Ticlid) is rarely used because of the associated risk of neutropenia.

THROMBOLYTICS. Patients seen with a STEMI at a facility without the capability of performing primary PCIs within 90 minutes receive thrombolytic therapy unless contraindicated. Thrombolytics activate thrombolytic processes to lyse the clot that is occluding the lumen of the coronary artery. Therapy should be initiated within 30 minutes of arrival at the facility. Symptom onset should be within the prior 12 hours, and ST elevation should be greater than 0.1 mV in at least two contiguous leads. Prehospital administration of thrombolytics may be initiated by rescue personnel trained and supported by expert practitioners; however, prehospital thrombolysis is not generally accepted practice at this time, although protocols continue to be established and evaluated.

Thrombolytics are administered intravenously when the ECG confirms the diagnosis of AMI. Commonly used thrombolytics include tissue plasminogen activators such as reteplase (Retavase), alteplase (Activase), tenecteplase (TNK-tPA), and streptokinase (Streptase). Streptokinase activates the conversion of plasminogen to plasmin, which degrades fibrin and fibrinogen into fragments. Tissue plasminogen activators also activate plasmin, but preferentially at the site of occlusion. Depending on the chosen agent, heparin may or may not be given concurrently. The risk of bleeding associated with the use of thrombolytic agents necessitates thorough screening of all patients for bleeding risks (see Guidelines for Safe Practice box). When contraindications to thrombolytic therapy exist, primary PCI is initiated without delay. The reperfusion of previously ischemic myocardium results in numerous biochemical and cellular events, which can include myocardial ischemia, dysrhythmias, and depressed myocardial contractility.

GLYCOPROTEIN IIB/IIIa RECEPTOR INHIBITORS. GP IIb/IIIa antagonists have been used successfully to affect the final pathway in platelet-thrombus formation in both ACSs and in conjunction with other therapeutic strategies. They inhibit platelet aggregation and fibrinogen binding, and thus may reduce the risk of restenosis, ischemic complications, and mortality in patients with unstable angina.

Figure 29-9 Algorithm for management of chronic stable angina. ACE, Angiotensin-converting enzyme; CAD, coronary artery disease; MI, myocardial infarction.
with PCI. By binding to the GP Ib/IIa receptor site, these drugs block the binding of fibrinogen to the platelet, thereby preventing platelet aggregation and clot formation. Approved agents currently include tirofiban (Aggrastat), eptifibatide (Integrilin), and abciximab (ReoPro). Inhibition of platelet aggregation persists for up to 48 hours after abciximab is discontinued; effects of eptifibatide and tirofiban are reversed when the infusion is discontinued.

**Anticoagulants.** Anticoagulants are often prescribed for the patient with ACS. Intravenous unfractionated heparin binds to antithrombin III, inactivating coagulation factors Xa, IXa, and thrombin, thereby blocking the conversion of fibrinogen to fibrin. Weight-adjusted doses are administered to achieve activated partial thromboplastin (aPTT) levels of 50 to 70 seconds. Low-molecular-weight heparins (LMWH) have a more predictable dose-response curve and an increased plasma half-life compared with unfractionated heparin. Enoxaparin (Lovenox) is the only LMWH approved for use in ACS.

**Direct Thrombin Inhibitors.** Bivalirudin (Angiomax) is used for anticoagulation in ACS patients undergoing coronary interventions. It is a synthetic analog of recombinant hirudin that binds to thrombin to inhibit the final step in the coagulation pathway. It is more predictable than conventional heparin and is active against clot-bound thrombin with continued efficacy after plasma clearance.

**Nitrates.** Nitrates are effective in the treatment of both stable angina and ACS. Nitrates cause vasodilation, reducing the amount of blood returning to the heart from the venous system, thus decreasing preload. This decreases both the workload of the heart and the myocardial oxygen demand. Nitrates also dilate the peripheral arteries, decreasing the resistance against which the left ventricle must pump, decreasing afterload, and reducing myocardial oxygen demand. In addition, nitrates act specifically to dilate coronary arteries that are not atherosclerotic, increasing collateral flow to the ischemic parts of the myocardium.

Many nitrate preparations are available for use. Sublingual nitroglycerin is used most commonly for acute episodes of angina. The tablets, absorbed within minutes from beneath the tongue, are highly effective in relieving the acute symptoms of angina. Intravenous nitroglycerin may be used for patients experiencing prolonged chest pain. Nitrates are also available as topical preparations, ointments, and patches that provide a sustained therapeutic effect. Shorter-acting ointment preparations are used during the hospitalization as medications are initiated and adjusted, since...
they can be quickly removed from the skin surface if hypotension occurs.

**Beta-Blockers.** Most beta-blockers used to treat stable angina and ACS are cardioselective, blocking predominantly the beta-1 receptor and causing a decrease in the force of contraction, a slowing of heart rate, and a slowing of impulse conduction. These three mechanisms of action combine to decrease myocardial oxygen demand. In addition, by slowing the heart rate, beta-blockers indirectly increase the blood supply to the myocardium by increasing diastole, thus increasing the time available for coronary artery perfusion. Beta-blockers also decrease blood pressure through their effect on the renin-angiotensin system.

The use of beta-blockers is associated with a decreased incidence of morbidity and mortality when they are administered within 48 hours of MI and continued for 5 to 2 years after AMI. Beta-blockers may be administered intravenously in the emergency department and then orally once the patient is stabilized.

**Calcium Channel Blockers.** The role of calcium channel blockers in the management of CAD is limited. Nondihydropyridine calcium channel blockers (diltiazem [Cardizem] or verapamil [Calan]) may be used when beta-blockers are contraindicated. These agents inhibit the influx of calcium through the slow calcium channels. They slow the heart rate and decrease myocardial oxygen supply by increasing the time for coronary perfusion during diastole. These agents also block the calcium used for myocardial contractility, decreasing the force of contraction (and hence oxygen demand).

**Angiotensin-Converting Enzyme Inhibitors.** ACE inhibitors may be used in the management of CAD to decrease preload and afterload and the overall workload of the heart. ACE inhibitors are recommended for patients with chronic stable angina who have significant CAD documented by angiography, who have diabetes, or who have left ventricular systolic dysfunction. Decreasing preload works to remodel the left ventricle, which involves the development of hypertrophy in the unaffected left ventricle that attempts to compensate for the loss of function in the infarcted area. The long-term consequence of remodeling is a steady increase in myocardial oxygen demand for the enlarged muscle and the onset of heart failure.

**Analgesics.** Despite the use of thrombolytics, acetylsalicylic acid, and heparin to open the coronary arteries and decrease chest pain, severe chest pain often persists. Pain activates the sympathetic nervous system, increasing heart rate and producing vasoconstriction. These changes decrease myocardial oxygen supply and increase myocardial oxygen demand. The immediate administration of intravenous opioid analgesics interrupts these deleterious effects of pain. The drug of choice is morphine sulfate, which not only blunts the sensation of pain, but also promotes vasodilation, thereby decreasing preload.

**Anxiolytics.** Alprazolam (Xanax) and other anxiolytics may be administered to patients who experience significant anxiety.

**Oxygen.** Oxygen is administered to the patient with ACS to maintain arterial oxygen saturation levels above 90%. This simple but effective intervention is key to increasing myocardial oxygen supply. Oxygen may be administered by nasal cannula or mask.

**Cholesterol-Lowering Agents.** Because considerable evidence links hypercholesterolemia to atherosclerosis, drugs that can reduce plasma lipids and lipoproteins are often prescribed in the treatment of patients with CAD. Drug classes include hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, niacin, absorption inhibitors ( bile acid resins, ezetimibe), and fibrates. The statin group of drugs (HMG-CoA reductase inhibitors) increases receptor activity that removes LDL from the blood, and blocks the production of LDLs. These lipid-lowering agents are especially useful as adjuncts to dietary management for patients with familial hypercholesterolemia (Table 29-6). In high-risk persons the recommended LDL cholesterol goal is 100 mg/dl. Latest clinical trial evidence recommends an LDL cholesterol goal of 70 mg/dl, especially for patients at very high risk. Combination therapy is instituted to obtain desirable LDL, HDL, and triglyceride levels.

**Treatments.** Patients with AMI may experience alterations in tissue perfusion to the skin, brain, kidneys, and other organs in addition to alterations in myocardial perfusion. Meticulous monitoring is an essential aspect of care. These alterations occur from a decrease in cardiac output that results from impaired myocardial contractility. Nurses, because of their ongoing presence at the bedside, assume most of the responsibility for ongoing monitoring for altered perfusion. Frequent measurement of vital signs is essential. The nurse performs head-to-toe assessments that include level of consciousness and orientation, breath sounds, heart sounds, pulse amplitude, rhythm strips, bowel sounds, urinary output, and skin turgor and hydration. Abnormal findings require immediate collaboration with the physician to prevent further complications.

**Intraaortic Balloon Pump.** Patients who experience hemodynamic instability after an MI may benefit from placement of an intraaortic balloon pump (IABP). The IABP inserted into the descending thoracic aorta, inflates during diastole, augmenting early diastolic pressure and coronary artery perfusion. The balloon deflates rapidly at the end of diastole, decreasing afterload and increasing cardiac output. A more complete description of the IABP is found in Chapter 30.

**Percutaneous Coronary Interventions.** An estimated 1,051,000 coronary interventional procedures were performed in the United States in 2001. These procedures can be performed in conjunction with diagnostic cardiac catheterization or as a separate procedure.

**Percutaneous Transluminal Coronary Angioplasty.** With balloon angioplasty the physician first inserts a guidewire across and beyond the lesion in the blocked artery, then advances a catheter with a cylindrical balloon over the guidewire, and positions the balloon centrally in the blockage. The balloon, filled with radiopaque dye and saline, is inflated at pressures great enough to
# Antiplatelet Agents

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Inhibits thromboxane-induced platelet aggregation</td>
<td>Antiplatelet agents should be prescribed unless true hypersensitivity reaction is present or patient has severe risk of bleeding.</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>Prevents platelet activation by blocking ADP-induced platelet binding</td>
<td></td>
</tr>
</tbody>
</table>

## Glycoprotein IIb/IIIa Receptor Inhibitors

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tirofiban (Aggrastat)</td>
<td>Inhibits final pathway in platelet thrombus formation by binding to GP IIb/IIIa receptor site</td>
<td>Observe patients for bleeding complications. Ensure correct weight-based dose. Monitor platelet counts.</td>
</tr>
<tr>
<td>Eptifibatide (Integrilin)</td>
<td>Inhibits ADP-induced platelet binding</td>
<td></td>
</tr>
<tr>
<td>Abciximab (ReoPro)</td>
<td>Inhibits ADP-induced platelet binding</td>
<td>Monitor platelet counts.</td>
</tr>
</tbody>
</table>

## Thrombolytics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alteplase (Activase)</td>
<td>Activates plasminogen to lysine clot associated with plaque rupture and vessel occlusion of MI</td>
<td>Carefully screen patients before administration of thrombolytic agents. Monitor for reperfusion, reoclusion, and bleeding complications with thrombolytic administration.</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>Activates conversion of plasminogen to plasmin, which degrades fibrin and fibrinogen into fragments</td>
<td>Monitor for reperfusion, reoclusion, and bleeding complications with thrombolytic administration.</td>
</tr>
<tr>
<td>Reteplase</td>
<td>Activates plasminogen to lysine clot associated with plaque rupture and vessel occlusion of MI</td>
<td>Monitor for reperfusion, reoclusion, and bleeding complications with thrombolytic administration.</td>
</tr>
<tr>
<td>Tenecteplase (TNK-tPA)</td>
<td>Activates plasminogen to lysine clot associated with plaque rupture and vessel occlusion of MI</td>
<td>Monitor for reperfusion, reoclusion, and bleeding complications with thrombolytic administration.</td>
</tr>
</tbody>
</table>

## Anticoagulants

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin</td>
<td>Prevents growth of established thrombus by rapidly inhibiting thrombin</td>
<td>With unfractionated heparin, measure heparin partial thromboplastin times (PTTs) 6 hr after any change in dose. Dose is weight based. Maintain therapeutic levels between 50 and 70 sec. LMWH does not require heparin PTTs. Monitor heparin levels and plasminogen for downward trends. Observe platelets for heparin-induced thrombocytopenia. Recurrent ischemia, active bleeding, and hypotension may signify subtherapeutic or supratherapeutic dosages and should be evaluated immediately.</td>
</tr>
<tr>
<td>Low-molecular-weight heparin (LMWH) (Enoxaparin [Lovenox])</td>
<td>Prevents growth of established thrombus by rapidly inhibiting thrombin</td>
<td>With unfractionated heparin, measure heparin partial thromboplastin times (PTTs) 6 hr after any change in dose. Dose is weight based. Maintain therapeutic levels between 50 and 70 sec. LMWH does not require heparin PTTs. Monitor heparin levels and plasminogen for downward trends. Observe platelets for heparin-induced thrombocytopenia. Recurrent ischemia, active bleeding, and hypotension may signify subtherapeutic or supratherapeutic dosages and should be evaluated immediately.</td>
</tr>
</tbody>
</table>

## Direct Thrombin Inhibitors

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bivalirudin (Angiomax)</td>
<td>Binds to thrombin, preventing further platelet aggregation and clot formation</td>
<td>Used with preservative coronary interventions. Monitor for bleeding, back pain, pain, nausea, headache, hypotension.</td>
</tr>
</tbody>
</table>

## Nitrates

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isosorbide dinitrate</td>
<td>Decreases myocardial oxygen demand</td>
<td>Administer sublingual nitrates with patients lying or sitting. Titrare intravenous nitrates to relieve symptoms or limit side effects such as headache or systolic BP &lt; 90 mm Hg. Replace intravenous preparations with oral or topical preparation when patient has been symptom free for 24 hr. Use caution in patients with known aortic stenosis. Anticipate headache, and administer analgesics as appropriate. Tolerance to nitrates can develop within 24 hr. A nitrate-free interval of 6-8 hr may improve responsiveness to therapy. Clean topical nitrates from skin surface before applying new dose. Appropriate areas of application include any hair-free area, preferably in noticeable areas when initial dose is being determined. Rotate application areas. Wear gloves when applying topical preparations.</td>
</tr>
</tbody>
</table>

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**TABLE 29-5 COMMON MEDICATIONS for Coronary Artery Disease**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isosorbide mononitrate (Imdur)</td>
<td>Decreases myocardial oxygen demand</td>
<td>Administer sublingual nitrates with patients lying or sitting. Titrare intravenous nitrates to relieve symptoms or limit side effects such as headache or systolic BP &lt; 90 mm Hg. Replace intravenous preparations with oral or topical preparation when patient has been symptom free for 24 hr. Use caution in patients with known aortic stenosis. Anticipate headache, and administer analgesics as appropriate. Tolerance to nitrates can develop within 24 hr. A nitrate-free interval of 6-8 hr may improve responsiveness to therapy. Clean topical nitrates from skin surface before applying new dose. Appropriate areas of application include any hair-free area, preferably in noticeable areas when initial dose is being determined. Rotate application areas. Wear gloves when applying topical preparations.</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Increase myocardial oxygen supply</td>
<td>Administer sublingual nitrates with patients lying or sitting. Titrare intravenous nitrates to relieve symptoms or limit side effects such as headache or systolic BP &lt; 90 mm Hg. Replace intravenous preparations with oral or topical preparation when patient has been symptom free for 24 hr. Use caution in patients with known aortic stenosis. Anticipate headache, and administer analgesics as appropriate. Tolerance to nitrates can develop within 24 hr. A nitrate-free interval of 6-8 hr may improve responsiveness to therapy. Clean topical nitrates from skin surface before applying new dose. Appropriate areas of application include any hair-free area, preferably in noticeable areas when initial dose is being determined. Rotate application areas. Wear gloves when applying topical preparations.</td>
</tr>
</tbody>
</table>
**Beta-Blockers**

- **Atenolol** (Tenormin): Decrease myocardial oxygen demand.
- **Metoprolol** (Lopressor): Decrease contractility.
- **Esmolol** (Brevibloc): Slow heart rate, thereby increasing diastolic filling time and coronary perfusion.

Give intravenous metoprolol in 5 mg increments over 1-2 min. Atenolol may be prescribed intravenously instead of metoprolol. Intravenous preparations are followed by oral preparations after patient is stabilized.

Decrease BP (through renin interaction) Monitor ECG and BP.

Slow heart rate, thereby increasing diastolic filling. Monitor for atrioventricular block (including measuring time and coronary perfusion P-R interval), symptomatic bradycardia, hypotension, left ventricular failure (rales, decreased cardiac output), and bronchospasm.

Decrease morbidity and mortality after acute MI, left ventricular failure (rales, decreased cardiac output), and bronchospasm. Target heart rate for beta-blockade at discharge is 50-60 beats/min.

**Calcium Channel Blockers**

- **Diltiazem** (Cardizem): Decrease afterload and preload, thereby decreasing workload of heart and decreasing remodeling of left ventricle.
- **Verapamil** (Calan): Long-term consequences of remodeling: increased oxygen demand and heart failure.

These are prescribed when vasospasm is considered part of pathologic condition or significant hypertension exists.

Monitor for symptomatic bradycardia, prolonged P-R intervals, advanced heart blocks, hypotension, heart failure.

**Angiotensin-Converting Enzyme Inhibitors**

- **Captopril** (Capoten): Decrease myocardial oxygen demand.
- **Enalapril** (Vasotec): Venodilate (decrease preload).
- **Benazepril** (Lotensin): With first doses, take BP before and 30 min after administration.
- **Lisinopril** (Prinivil): Fosinopril (Monopril):

Increase arterial oxygen saturation.

Monitor for adverse effects: angioneurotic edema, cough, hypotension, hyperkalemia, pruritic rash, renal failure.

Doses are usually given in increments of 2-5 mg.

**Analgesics**

- **Morphine sulfate**: Blunts deleterious consequences of sympathetic stimulation with pain. Vasodilates, decreasing preload

Monitor for hypotension, respiratory depression, changes in level of consciousness.

Doses are usually given in increments of 2-5 mg.

**Anxiolytics**

- **Alprazolam** (Xanax): Binds receptors at several sites within CNS, including limbic system and reticular formation.

Monitor for lessening anxiety, which may allow for reduction of doses of analgesics.

**Oxygen**

Increased arterial oxygen saturation.

Monitor for adequate arterial oxygenation with finger pulse oximetry. Maintain saturation levels above 90%.

**Cholesterol-Lowering Agents**

- **Atorvastatin** (Lipitor)
- **Lovastatin** (Mevacor)
- **Pravastatin** (Pravachol)
- **Rosuvastatin** (Crestor)
- **Simvastatin** (Zocor)
- **Ezetimibe** (Zetia)
- **Gemfibrozil** (Lopid)
- **Niacin** (nicotinic acid)

**TABLE 29-5 COMMON MEDICATIONS for Coronary Artery Disease—cont’d**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Beta-Blockers</strong></td>
<td>Atenolol</td>
<td>Decrease myocardial oxygen demand</td>
<td>Give intravenous metoprolol in 5 mg increments over 1-2 min. Atenolol may be prescribed intravenously instead of metoprolol. Intravenous preparations are followed by oral preparations after patient is stabilized.</td>
</tr>
<tr>
<td></td>
<td>Metoprolol</td>
<td>Decrease contractility</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Esmolol</td>
<td>Slow heart rate, thereby increasing diastolic filling time and coronary perfusion</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Calcium Channel Blockers</strong></td>
<td>Diltiazem</td>
<td>Decrease afterload and preload, thereby decreasing workload of heart and decreasing remodeling of left ventricle</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>Long-term consequences of remodeling: increased oxygen demand and heart failure</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Angiotensin-Converting Enzyme Inhibitors</strong></td>
<td>Captopril</td>
<td>Decrease myocardial oxygen demand</td>
</tr>
<tr>
<td></td>
<td>Enalapril</td>
<td>Venodilate (decrease preload)</td>
<td>Monitor for adverse effects: angioneurotic edema, cough, hypotension, hyperkalemia, pruritic rash, renal failure. With first doses, take BP before and 30 min after administration.</td>
</tr>
<tr>
<td></td>
<td>Benazepril</td>
<td>With first doses, take BP before and 30 min after administration.</td>
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<tr>
<td></td>
<td>Lisinopril</td>
<td>Fosinopril</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Analgesics</strong></td>
<td>Morphine sulfate</td>
<td>Blunts deleterious consequences of sympathetic stimulation with pain. Vasodilates, decreasing preload</td>
</tr>
<tr>
<td></td>
<td><strong>Anxiolytics</strong></td>
<td>Alprazolam</td>
<td>Binds receptors at several sites within CNS, including limbic system and reticular formation</td>
</tr>
<tr>
<td></td>
<td><strong>Oxygen</strong></td>
<td></td>
<td>Increase arterial oxygen saturation. Monitor for adequate arterial oxygenation with finger pulse oximetry. Maintain saturation levels above 90%.</td>
</tr>
<tr>
<td></td>
<td><strong>Cholesterol-Lowering Agents</strong></td>
<td>Atorvastatin</td>
<td>Reduce substrate for lipid deposition in coronary artery</td>
</tr>
<tr>
<td></td>
<td>Lovastatin</td>
<td></td>
<td>Side effects vary with drug class. Tolerance to side effects may limit usefulness of certain medications. Obtain lipid levels at regular intervals to monitor for success in effecting changes. Teach patients that cholesterol-lowering agents do not substitute for dietary modifications (see Table 29-7).</td>
</tr>
<tr>
<td></td>
<td>Pravastatin</td>
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<tr>
<td></td>
<td>Rosuvastatin</td>
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<td></td>
<td>Simvastatin</td>
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<tr>
<td></td>
<td>Ezetimibe</td>
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<tr>
<td></td>
<td>Gemfibrozil</td>
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<tr>
<td></td>
<td>Niacin</td>
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</tbody>
</table>

ADP, Adenosine diphosphate; BP, blood pressure; CNS, central nervous system; ECG, electrocardiogram; GP, glycoprotein; MI, myocardial infarction.
UNIT 7 Cardiovascular Problems

Guidelines for Safe Practice: The Patient Receiving Thrombolytic Therapy

**Patient Eligibility**
Within 6 to 12 hours of symptom onset
Symptom duration of at least 30 minutes
Electrocardiogram (ECG) pattern strongly suggestive of acute myocardial infarction (ST elevation or new left bundle branch block)

**Patient Screening**
Screen for bleeding risks: history of cerebral hemorrhage at any time, ischemic stroke within 3 months (except acute ischemic stroke within 3 hours), intracranial neoplasm, active bleeding, suspected aortic dissection, severe hypertension, recent anticoagulation therapy, traumatic or prolonged cardiopulmonary resuscitation (over 10 minutes), major surgery within 3 weeks, significant closed head or facial trauma within 3 months, arteriovenous malformation.

Establish baseline vital signs and physical examination for overt or covert bleeding, such as unexplained hypotension or tachycardia, rigid abdomen, subtle neurologic changes.

**Monitor for Successful Reperfusion**
Resolution of chest pain
Resolution of ECG ST changes
Presence of reperfusion dysrhythmias, such as accelerated idioventricular rhythm
Early peak of cardiac biomarkers

**Minimize Risk of Bleeding**
Continue assessment for bleeding, including intracranial, internal, retroperitoneal, and puncture sites.
Monitor for frank and occult blood (heme, guaiac).
Monitor for any change in neurologic status in first 24 hours.
Monitor laboratory values for therapeutic ranges.
Use caution with patient transfers.
Limit and coordinate venipunctures; avoid establishing noncompressible intravenous access sites.
Apply pressure to all venous and arterial access sites.
Avoid arterial punctures after fibrinolysis.
Maintain a safe, clean environment.

**Monitor for Reclosure**
Recurrence of chest pain
Return of ST abnormalities
Evidence of hemodynamic compromise

**Support Patient and Family During Crisis**
Approach in a calm, quiet manner.
Provide simple explanations of procedures and care.
Offer realistic reassurance.
Encourage family presence when interventions permit.

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Lipid and Lipoprotein Effects</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydroxymethylglutaryl–coenzyme A reductase inhibitors:</td>
<td>↓ LDL 18%-60%</td>
<td>Myopathy</td>
</tr>
<tr>
<td>Atorvastatin, fluvastatin, lovastatin, pravastatin, simvastatin, rosvastatin</td>
<td>↑ HDL 5%-15%</td>
<td>Increased liver enzymes</td>
</tr>
<tr>
<td>↑ TG 7%-30%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ HDL 3%-5%</td>
<td>Bloating</td>
<td></td>
</tr>
<tr>
<td>TG: no change or ↓</td>
<td>Constipation</td>
<td></td>
</tr>
<tr>
<td>↓ LDL 18%-25% when used alone or added to a statin</td>
<td>Decreased absorption of other drugs</td>
<td></td>
</tr>
<tr>
<td>Bile acid sequestrants:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colestipol, colestevam, colestyramine</td>
<td>↓ LDL 15%-30%</td>
<td></td>
</tr>
<tr>
<td>↓ HDL 3%-5%</td>
<td>Constipation</td>
<td></td>
</tr>
<tr>
<td>↑ TG 7%-30%</td>
<td>Angioedema</td>
<td></td>
</tr>
<tr>
<td>Fibrates:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gemfibrozil, fenofibrate, clofibrate</td>
<td>↓ LDL 5%-20% (may increase if high TG)</td>
<td>Dyspepsia</td>
</tr>
<tr>
<td>↓ HDL 10%-20%</td>
<td>Gallstones</td>
<td></td>
</tr>
<tr>
<td>↑ TG 20%-50%</td>
<td>Myopathy</td>
<td></td>
</tr>
</tbody>
</table>


LDL, low-density lipoprotein; HDL, high-density lipoprotein; TG, triglyceride.
reconfigure the blockage. This reconfiguration includes both controlled dissection (splitting) of the intima and to a lesser extent vessel dilatation (Figure 29-11). The controlled dissection creates a wider passage for arterial blood flow. At times, the dissection may create enough turbulence to stimulate clot formation and obstruct the coronary lumen. In these situations, GP IIb/IIIa receptor inhibitors or additional interventional measures (such as intra-coronary stenting) may be necessary.

The major limitation of percutaneous transluminal coronary angioplasty (PTCA) is the strong chance of lesion recurrence or restenosis, usually within 6 months. Restenosis occurs in response to the controlled injury caused by balloon inflation. In approximately 50% of procedures, the arterial wall continues to heal with smooth muscle proliferation into the arterial lumen. Although this is not the same lipid accumulation that caused the original blockage, it nevertheless compromises myocardial blood flow and results in myocardial ischemia. Arterial constriction can also occur with intimal hyperplasia.

Stents. Intracoronary stents help maintain the patency of the treated coronary arteries and decrease the incidence of restenosis (see Figure 29-11). Bare-metal stents remain in the coronary artery as a scaffold and endothelialize over 3 weeks, gradually decreasing the risk of thrombus formation on the foreign material. Stent thrombosis most frequently occurs in the first days to weeks after stent implantation. Patients usually are seen with severe chest pain and often exhibit ST segment elevation. The incidence of stent thrombosis is decreased with the administration of aspirin and loading doses of platelet ADP-receptor inhibitor therapy before the procedure, and treatment with aspirin and other platelet inhibitors after the procedure. Drug-eluting stents have been studied as one avenue of decreasing stent thrombosis. The use of sirolimus, an immunosuppressive that blocks growth factors or cytokines that stimulate smooth muscle cell proliferation, was approved in 2002. The stent is coated in sirolimus, which is slowly released over 30 days. Paclitaxel (Taxol), a drug approved for the treatment of various cancers, is being evaluated as another stent-coating agent. Because of concern that endothelialization may simply be delayed and not prevented, and late stent thrombosis may still develop in patients who are treated with drug-eluting stents, clopidogrel and aspirin therapy are administered for 6 months after the PCI. Most coronary stents in current use are stainless steel; some are weakly ferromagnetic. Therefore magnetic resonance imaging (MRI) procedures are considered safe when clinically indicated.

Other PCIs. Less commonly used PCI procedures include directional coronary atherectomy, laser therapy, transluminal extraction catheterization, and rotablation. These procedures are especially beneficial for specific types of lesions. The effect of hypothermia during PCI for MI is being evaluated in a prospective, international study23 (see Future Watch box).

Procedural complications associated with PCI include allergic dye reactions, contrast nephropathy, and access site complications. Patients with known allergic reactions to contrast dye should be pretreated with steroids and a histamine1 blocker. Patients at greatest risk for contrast nephropathy include those
Future Watch

**Hypothermia, Myocardial Infarction, and Percutaneous Coronary Intervention**

An international, multicenter, prospective, randomized trial is currently in progress to investigate the effects of cooling on patients with myocardial infarction (COOL MI). The patient’s body temperature is cooled as blood contacts a catheter filled with circulating cool saline that is placed into the inferior vena cava via the femoral vein during percutaneous coronary intervention. The patient’s temperature is maintained during the procedure at 31° C (87.8° F) to 34° C (93.2° F) for 1 hour. The patient’s core temperature is rapidly warmed to 36° C (96.8° F) after the procedure. The Guidelines for Safe Practice box summarizes the Guidelines for Safe Practice box.

**Postprocedural Considerations.** Postprocedural protocols are carefully implemented to prevent or promptly identify complications. The patient undergoing interventional procedures requires close monitoring for vessel occlusion, bleeding and hematoma formation, thromboembolism, pseudoaneurysms, and contrast dye reactions. Hemostasis at the access site is achieved through manual compression, suture-mediated devices (Perclose), vascular plugs (VasoSeal, AngioSeal), and procoagulants. Care of the patient undergoing PCI is summarized in the Guidelines for Safe Practice box.

**Transmyocardial Laser Revascularization.** Transmyocardial laser revascularization uses laser energy to create channels through the left ventricular free wall into the ischemic myocardium. The procedure is used to treat refractory angina and is believed to increase blood flow to the myocardium through the channels and stimulate angiogenesis to increase collateral blood flow. It can be performed percutaneously, similar to other PCI, or surgically, through a median sternotomy or left thoracotomy approach. Depending on the type of laser used, myocardial channels are created from thermal ablation or by breaking of molecular bonds within the myocardial cells. Complications may include cardiac tamponade and heart failure.

**Surgical Management.** Coronary artery bypass graft (CABG) surgery bypasses the obstruction in a coronary artery by grafting an artery or vein to the coronary artery beyond the blockage, reestablishing blood flow (Figure 29-12). The decision to operate depends on the location of the coronary lesion and the surgical risks and benefits. CABG is indicated for patients with significant left main CAD, for patients with three-vessel disease and a left ventricular ejection fraction (LVEF) of less than 0.50; and for patients with two-vessel disease with significant proximal LAD CAD and either an LVEF of less than 0.50 or evidence of significant ischemia with stress testing. The increased use of antiplatelet therapy for ACS increases the risk of bleeding complications with CABG. The use of epifibatide, if discontinued at least 2 hours before surgery, appears to cause less bleeding than other GP IIb/IIIa inhibitors. It is recommended that, when possible, dipidergolide be held for 5 days before surgery.27

Although CABG surgery is not curative because the grafts can occlude, it improves the quality of life for many patients. Heart transplants may be used in selected patient whose hearts are so badly damaged that conventional therapy is of no benefit. Chapter 30 presents more information on CABG and transplant surgery.

**Diet.** The patient being evaluated for acute chest pain is given nothing by mouth (NPO) until the diagnosis of MI can be ruled out. Keeping the patient NPO prevents vomiting, which commonly accompanies chest pain from vagal nerve effects. Patients may also be NPO before cardiac procedures.

**The National Cholesterol Education Program III (NCEP III) guidelines recommend the Therapeutic Lifestyle Changes diet for patients with cardiac disease (Table 29-7).** Diet teaching includes reducing fat content, substituting polyunsaturated fat for saturated fat, and maintaining body weight at normal levels. An update to the original NCEP III guidelines recommends more stringent lipid values for those at high risk for CAD.66 An LDL of less than 100 mg/dl is still an overall goal for high-risk patients, but for very high-risk patients it may be preferable to lower LDL levels to less than 70 mg/dl. The 2004 update also recommends that patients with LDL levels from 100 to 129 mg/dl receive cholesterol-lowering drug therapy.37 Research has clearly indicated that when polyunsaturated fats replace saturated fats in the diet, blood cholesterol levels tend to fall. Dietary sources of polyunsaturated fats include corn, cottonseed, soy, and safflower oils; and margarines incorporating these oils in liquid form. Hydrogenated oils contain more saturated fat, as do tropical oils, butterfat, and animal fats. Transfatty acids are created when oil is hydrogenated, a process that makes an oil more solid at room temperature, extending the product’s shelf life. When an unsaturated fat converts to a transfatty acid, it then acts in the body in much the same way as a saturated fat. Transfatty acids increase LDLs and total cholesterol and may even decrease
For patients with hyperhomocysteinemia, interventions focus on ways to lower total homocysteine levels. Effective measures include increasing the consumption of vitamin-enriched or fortified foods such as fish, fortified grains and cereals, fruits, legumes, meats, and vegetables. Additional sources of folate, vitamin B6, and vitamin B12 include fish, fortified grains and cereals, fruits, legumes, meats, and vegetables. Supplemental vitamins may also be given.

### Health Promotion and Prevention

Every patient with CAD needs a comprehensive educational plan aimed at promoting health that is based on the individual’s unique risk factors. The Patient/Family Teaching box presents an overview of health promotion guidelines for patients with CAD.

### General Concepts to Reinforce

Indications for the procedure

PCI not a surgical intervention: no incisions, no general anesthesia

Risks factors associated with procedure, including a less than 1% chance of emergency surgery in uncomplicated PCIs

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### Health Promotion and Prevention

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Saphenous vein grafts

Figure 29-12 Coronary artery bypass graft surgery. Common grafts: saphenous vein and internal mammary artery.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Recommended Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fat</td>
<td>Less than 7% of total calories</td>
</tr>
<tr>
<td>Polyunsaturated fat</td>
<td>Up to 10% of total calories</td>
</tr>
<tr>
<td>Monoinsaturated fat</td>
<td>Up to 20% of total calories</td>
</tr>
<tr>
<td>Total fat</td>
<td>25%-35% of total calories</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>50%-60% of total calories</td>
</tr>
<tr>
<td>Fiber</td>
<td>20-30 g/day</td>
</tr>
<tr>
<td>Protein</td>
<td>Approximately 15% of total calories</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Less than 200 mg/day</td>
</tr>
<tr>
<td>Total calories (energy)</td>
<td>Balance energy intake and expenditure to maintain desirable body weight, prevent weight gain</td>
</tr>
</tbody>
</table>


Complementary & Alternative Therapies

Drinking hot tea has been advocated throughout the years as a remedy for numerous ailments. This complementary therapy reduced the risk of myocardial infarction (MI) in 4807 men and women monitored for more than 5 years. Individuals who drank more than three cups of black tea reduced their risk of MI by 43% compared with those who did not. The threat of a fatal coronary event was reduced by 70%. Women had more favorable results than men. The researchers postulate that flavonoids within the tea mediate an estrogenic effect, creating the cardioprotective effects.


TABLE 29-7 THE THERAPEUTIC LIFESTYLE CHANGES DIET

<table>
<thead>
<tr>
<th>Risk Factor Modification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Provide specific verbal and written instructions on smoking cessation, stress management, and diet modification.</td>
</tr>
<tr>
<td>Consider referral to a smoking cessation program or outpatient cardiac rehabilitation program.</td>
</tr>
<tr>
<td>Encourage adherence to a diet low in calories, saturated fats, and cholesterol.</td>
</tr>
<tr>
<td>Discuss the benefits of stress management techniques in decreasing negative effect on oxygen demand. Refer to individual or group counseling as needed.</td>
</tr>
</tbody>
</table>

Resumption of Activity

Discuss the benefits of exercise and encourage a regular exercise program.
Provide specific instructions on activities that are permissible and those to avoid.
Discuss resumption of driving and return to work.
Discuss guidelines for resuming sexual relations.

Medications

Ensure understanding of the role of aspirin.
Instruct patient that recurrent symptoms lasting more than 1 to 2 minutes should prompt the patient to stop all activities, sit down, and take a sublingual nitroglycerin (NTG) tablet. This may be repeated at 5-minute intervals for two additional tablets if needed. If symptoms persist, patient should call emergency medical services (911). (High-risk patients may be taught to call after the first NTG.)
Teach correct use and storage of nitroglycerin (see Patient/Family Teaching box, p. 000).
Instruct patient on the purpose, dose, and major side effects of each medication prescribed.
Before the patient’s hospital discharge, the nurse thoroughly reviews with the patient and family all medications, their purpose, dose, and possible side effects and establishes a medication schedule suited to the patient’s lifestyle. This collaborative effort promotes adherence to the medical regimen. The nurse reminds the patient to discuss drug side effects with his or her health care provider and not to discontinue any medications without consultation.

The importance of exercise in preventing disease progression cannot be overstated. A regular exercise regimen can decrease LDLs, increase collateral circulation, decrease resting heart rate, and decrease blood pressure. Despite these benefits, patients with known cardiac disease must take precautions to prevent overtaxing the already compromised balance of myocardial oxygen supply and demand. Activity guidelines promote conditioning and simultaneously prevent overexertion that could further increase myocardial oxygen demand. The family is included, if possible, in all discussions of activity progression after MI. Disagreements over acceptable activity are a major source of conflict between spouses and patients, adding to the stress of this crisis situation. The nurse also facilitates discussion regarding the stress of this illness on children of all ages, who commonly exhibit behavior changes, sleep disturbances, and somatic complaints in response to the stress of an MI involving a parent.

Many of the Healthy People 2010 goals target the primary prevention of CAD (see Healthy People 2010 box). The ability to meet these goals depends on the development of new and creative approaches that minimize accessibility barriers related to culture, ethnicity, race, and socioeconomic status.11

**Healthy People 2010**

### Objectives Related to Heart Disease

- Reduce coronary heart disease deaths to no more than 166 per 100,000 people.
- Increase the proportion of adults ages 20 years and older who are aware of the early warning symptoms and signs of a heart attack and the importance of accessing rapid emergency care by calling 911.
- Increase the proportion of eligible patients with heart attacks who receive artery-opening therapy within an hour of symptom onset.
- Increase the proportion of adults ages 20 years and older who call 911 and administer cardopulmonary resuscitation when they witness an out-of-hospital cardiac arrest.
- Increase the proportion of eligible persons who had an out-of-hospital cardiac arrest with witnesses who receive their first therapeutic electrical shock within 6 minutes after collapse recognition.
- Reduce the mean total blood cholesterol levels among adults to 199 mg/dl.
- Reduce the proportion of adults with high total blood cholesterol levels to no more than 17%.
- Increase to at least 80% the proportion of adults who have had their blood cholesterol checked within the preceding 5 years.
- Increase the proportion of persons with coronary heart disease who have their low-density lipoprotein-cholesterol level treated to reach a goal of less than or equal to 100 mg/dl.
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**From US Department of Health and Human Services, Healthy people 2010: understanding and improving health, Washington, DC, 2000, The Department.**

**ARE YOU READY?**

In nurse recognizes which of the following as the most specific biomarker for myocardial infarction?

1. CPK-MB
2. Myoglobin
3. Serum troponin
4. Creatine protein

**Nursing Management of the Patient with Coronary Artery Disease**

### ASSESSMENT

**Health History.** Assess for:

- Chest pain: location, severity, intensity, quality, duration, time of onset (Patient may be asymptomatic; classic pattern is retrosternal pain that may radiate down the left arm or both arms, upward to neck or jaw, or backward to scapular region; MI pain may be described as crushing or worst pain ever experienced.)
- Precipitating factors (e.g., exercise, stress, smoking)
- Measures attempted to control pain (e.g., nitroglycerin, lying down, eating or drinking, using antacids); effectiveness
- Risk factors for CAD (e.g., positive family history, lipid profile, tobacco use, history, stress levels, exercise pattern)
- Other symptoms (e.g., indigestion, heartburn, nausea, abdominal pain, malaise, diziness, dyspnea, anxiety or feeling of doom)
- Other illnesses (e.g., diabetes, hypertension, bleeding disorders, recent trauma or surgery); current management regimen and allergies
- Medications in use—prescription, over the counter, herbal products, nutritional supplements
- Support systems, insurance coverage, financial resources for rehabilitation
- Current employment, activity level
- Altered level of consciousness, syncope
- Vomiting
- Declining urinary output
- Pale, cool, diaphoretic skin

**Physical Examination.** Assess for:

- Posture indicating presence of chest pain (e.g., clutching or rubbing chest, leaning forward)
- Changes in vital signs: tachycardia or bradycardia, hypertension or hypotension
- Dyspnea or shortness of breath, rales (crackles)
- Presence of S3 or S4
- Dysrhythmias
- Posture indicating presence of chest pain (e.g., clutching or rubbing chest, leaning forward)
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NURSING DIAGNOSES, OUTCOMES, AND INTERVENTIONS

Nursing Diagnosis: Acute Pain

OUTCOMES. Common examples of expected outcomes for the patient with a diagnosis of acute pain are:

Patient will:

• Be free from chest pain or anginal equivalent.
• Be able to effectively control angina through the use of interventions.

NURSING INTERVENTIONS. Because ischemic cardiac pain results from an imbalance between myocardial oxygen supply and demand, treatment of pain attempts to increase myocardial oxygen supply while reducing myocardial oxygen demand (see Guidelines for Safe Practice box). Immediate nursing interventions include administering prescribed oxygen, opioids, and nitrates; and assisting with measures to open an occluded artery (reperfusion therapy). Before medication administration, the nurse validates the absence of allergies and bleeding risks and establishes baseline vital signs, level of consciousness, and orientation. The nurse observes the patient for any deviations from this baseline after the administration of nitrates, thrombolytics, and opioids. It is helpful to have the patient rate the chest pain on a scale of 0 to 10, where 0 is no pain and 10 is the worst pain ever. The patient's pain ratings over time provide a baseline from which to evaluate the effectiveness of the immediate interventions. Morphine and fentanyl are the preferred analgesics for cardiac pain.

Because of the vasodilatory effects of nitrates, the nurse instructs the patient to lie down before administration. An ECG may also be obtained before the first dose of nitroglycerin is given. When the patient has documented CAD and the treatment strategy has already been determined, nitroglycerin administration can be initiated before a diagnostic ECG. This prevents additional delays in treatment. Because of the vasodilator effects of nitroglycerin on cerebral arteries, many patients receiving nitroglycerin complain of headache that may be severe enough to require analgesic administration. The Patient/Family Teaching box provides detailed information about the safe and effective use of sublingual nitroglycerin.

Topical nitrates, supplied as ointments, creams, and pastes, may also be used. The nurse administering the medication must handle these preparations carefully and use clean gloves when applying the medication. The nurse places the topical nitrate on the chest or upper arm, avoiding areas with excess hair, and rotates the site of application with each dose. Topical nitrates can be easily removed if untoward effects develop, and this advantage proves useful during dose adjustments in the early phases of treatment.

GUIDELINES FOR SAFE PRACTICE

The Patient Experiencing Angina

• Stay with patient. Ask for assistance in obtaining needed equipment (e.g., 12-lead electrocardiogram [ECG] and oxygen setup).
• Assess for presence of chest pain (or anginal equivalent). Document baseline intensity.
• Obtain baseline vital signs. Continue to monitor vital signs every 5 minutes during interventions.
• Apply oxygen when available. Monitor changes in oxygen saturation.
• Ensure intravenous access (two sites).
• Obtain an ECG as soon as possible. For diagnostic purposes an ECG should be performed before administration of nitroglycerin. If patient has known coronary artery disease, nitroglycerin may be administered before ECG. Set up continuous ST segment monitoring. Obtain serial ECGs as indicated.
• Ensure that patient has received aspirin.
• Administer nitroglycerin and morphine per orders until pain resolves. If pain is not responsive to sublingual nitroglycerin and morphine, anticipate additional interventions such as intravenous nitroglycerin.
• Treat alterations in vital signs with appropriate medications. If ECG indicates acute myocardial infarction, anticipate and prepare for thrombolytic therapy or primary percutaneous coronary intervention.
• Obtain laboratory specimens as indicated. Specimens may include complete blood count, chemistry, coagulation studies, and cardiac biomarkers (troponin and creatine kinase MB).
• Assess patient’s level of anxiety and offer realistic reassurance. Explain all interventions. Approach patient and family in a calm, confident manner. Minimize environmental stimulation.

Use of Sublingual Nitroglycerin

Sit or lie down at onset of angina or chest pain. Place tablet under the tongue and allow tablet to dissolve; do not chew.

If pain is not relieved within 5 minutes, take a second tablet. A third tablet can be used after an additional 5 minutes if pain persists. Continuing pain after three tablets and 15 minutes indicates a need to receive immediate medical evaluation. (High-risk patients may be taught to call 911 after the first nitroglycerin.)

Tablets will cause a tingling sensation under the tongue; rest for 15 to 20 minutes after taking nitroglycerin to avoid faintness.

A tablet may, with the physician’s permission, be taken 10 minutes before an activity known to trigger an anginal attack. Anticipate the occurrence of hypotension, tachycardia, and headache in response to the medication. Headache may persist for 15 to 20 minutes after administration.

Keep a record of the number of anginal attacks experienced, the precipitating factors if known.

NOTE: Sublingual spray is administered following the same guidelines as above.

Storage of Nitroglycerin

Carry tablets for immediate use if necessary. Do not pack in luggage when traveling.

Keep tablets in tightly closed, original container. Protect tablets from exposure to light and moisture.

Store tablets in a cool, dry place.

Check expiration date on prescription. Discard tablets after 6 months once the bottle has been opened. Plan for replacement of supply.
Oral nitrates typically replace topical nitrates for long-term therapy. Nicotine tolerance develops rapidly with ongoing use, and it is important to provide a nicotine-free interval, usually at night, to minimize its development. Intravenous nitroglycerin may be used in the treatment of ACS. During the administration of intravenous nitroglycerin, the nurse frequently monitors the patient’s blood pressure. Intravenous nitroglycerin is typically titrated to keep the patient pain free while maintaining a systolic blood pressure above 90 mm Hg. Thrombolysis is used emergently to open the blocked coronary artery, increase the blood supply to the myocardium, and relieve pain. Before thrombolytic administration, all members of the health care team participate in screening the patient for bleeding risk (see Guidelines for Safe Practice box, p. 762). No question can be asked too often in this situation. Thrombolysis must be administered without delay, preferably within 4 to 6 hours of symptom onset, although benefits are still possible up to 12 hours later. The nurse assisting in the administration of thrombolysis must be knowledgeable about all current treatment protocols to minimize the preparation time. The nurse obtains baseline vital signs and completes a physical examination for signs and symptoms of overt or covert bleeding. During the administration of thrombolysis, the nurse monitors the patient’s heart rate and blood pressure. If the ECG is within the expected range of ST-segment elevation, the patient must also be observed for any signs of cardiac ischemia. Some thrombolysis treatment efforts also address the need to decrease the patient’s myocardial oxygen demand. MI patients are often started on intravenous beta-blockers in the emergency room, and the nurse further decreases myocardial oxygen demand by modifying the patient’s environment in subtle ways such as adjusting the room temperature and restricting visitors who increase the patient’s anxiety or prevent the patient from resting. In addition, the nurse attempts to decrease the patient’s anxiety level by approaching the patient in a calm, quiet manner (often the opposite occurs in the hectic setting of the emergency department or coronary care unit) and carefully explaining all care and procedures. Anxiety may also be administered to decrease the sympathetic effects of the stress response.

**Related NIC Interventions.** Analytical Auditing, Anxiety Reduction, Cardiac Precautions, Medication Management, Pain Management

**Nursing Diagnosis:** Anxiety

**Outcomes.** A common example of an expected outcome for the patient with a diagnosis of anxiety is:
- Patient will:
  - Experience only manageable levels of anxiety, permitting patient to seek and process information.

**Nursing Interventions.** Nursing interventions to relieve anxiety are best directed at its cause. For the MI patient, the threat of death is real and is a common source of severe anxiety. Psychologic support, realistic reassurance, brief explanations about care (to the extent desired by the patient), and family visiting should be priorities for a patient with an MI. All interventions aimed at reducing anxiety should also include the family, who are also likely to experience high levels of anxiety and may even make the patient’s level of anxiety worse. Anxiolytics may be prescribed to decrease patient anxiety, especially during the acute phase of MI. Severe anxiety is common and increases the patient’s myocardial oxygen demand at a time of decreased oxygen supply. Persistent anxiety may be managed with stress reduction techniques alone or in combination with anxiolytics. Stress reduction techniques include relaxation therapy, guided imagery, music therapy, and exercise. Supportive listening is a simple but effective intervention, especially when combined with realistic reassurance and appropriate sharing of information. All these interventions are beneficial, but research indicates that a structured exercise program eventually offers the best overall outcomes for the patient with CAD.

**Related NIC Interventions.** Anxiety Reduction, Presence, Simple Relaxation Therapy

**Nursing Diagnosis:** Activity Intolerance

**Outcomes.** Common examples of expected outcomes for the patient with a diagnosis of activity intolerance are:
- Patient will:
  - Tolerate gradually increasing levels of activity.
  - Verbalize the guidelines for resuming sexual activity.

**Nursing Interventions.** Initially the patient experiencing chest pain is restricted to bed rest. This activity restriction decreases myocardial oxygen demand until biomarkers peak and a definitive diagnosis of MI can be made or ruled out. After the patient is hemodynamically stable and free of chest pain, activity can be increased gradually (see Guidelines for Safe Practice box). Assessment for activity tolerance includes monitoring for changes in blood pressure in response to position changes, dysrhythmias, appropriate changes in blood pressure and heart rate in response to activity, and symptoms such as dyspnea or chest pain. The presence of symptoms or hemodynamic changes necessitates cessation of activity until the patient stabilizes and the potential for cardiac ischemia decreases.

Before discharge or soon thereafter, postinfarction patients may undergo stress testing to determine a safe individual exercise level. Patients ideally enroll in outpatient cardiac rehabilitation programs. These programs supervise the progression of activity and offer variety in modes of exercise (bicycle, steps, weights), although outpatient programs vary in their effectiveness in creating and sustaining lifestyle changes (see Research box). Unfortunately, not all insurance companies recognize the benefit of structured rehabilitation programs, and financial constraints may prevent patients from enrolling. In these situations standardized home exercise programs are recommended (see Patient/Family Teaching box). Activity prescriptions consider the location and extent of myocardial damage, results of stress testing when available, and specific patient needs. The activity pyramid (Figure 29-13) may help patients and families appreciate the progressive nature of building activity into their daily lives. The base of the pyramid emphasizes the importance of at
Guidelines for Safe Practice

Activity Progression—Admission
0 to 12 hours: bed rest with bedside commode
12 to 24 hours: orthostatic check; out of bed to chair with meals, ad lib in room

Activity Progression—Day 2 to Discharge
Duration: first session, walk 1 to 2 minutes; increase duration 1 to 2 minutes per session if patient tolerates (see Criteria)
Frequency: initially one to four times per day for less than 10 minutes
Intensity: maintain heart rate no greater than 20 beats/min over baseline; patient should be able to converse on ambulation without shortness of breath

Criteria for Progressing Activity
Heart rate* within 20 beats/min of standing baseline heart rate
Systolic blood pressure* within 20 mm Hg of standing baseline blood pressure
Absence of chest pain, pressure, or anginal equivalent; shortness of breath; dysrhythmia; fatigue; lightheadedness; diaphoresis

*Blood pressure and heart rate checks must be taken with patient in a standing position and immediately postambulation. Postambulation heart rate should be measured by taking pulse for 10 seconds and multiplying by 6.

least 30 minutes of moderately intense exercise on most, if not all, days of the week. More structured and varied activity options are built on this base. The ultimate goal is to decrease amount of time spent in the sedentary activities found at the peak of the pyramid.

The nurse includes information about returning to work and sexual activity as part of the overall activity guidelines. Return to work is individualized to the patient's occupation. A patient with a desk job and low stress levels receives different guidelines than the patient with high occupational stress or heavy labor demands. Medications often improve a patient's tolerance of activity. Nitroglycerin taken before an activity that is known to cause angina may allow the patient to complete the activity without experiencing chest pain. Beta-blockers decrease the sympathetic response to exercise, allowing patients to exercise at an increased intensity but with a safer heart rate. Both myocardial oxygen demand and efficiency improve with the use of beta-blockers. Fatigue commonly limits the patient's exercise tolerance and can be related to medications, particularly beta-blockers. The nurse informs the patient about potential fatigue and what to do if it occurs. The patient taking beta-blockers is cautioned not to discontinue the medication abruptly, since this can result in rebound angina and hypotension. The nurse encourages the patient to discuss concerns with a primary care provider. Interventions for medication-induced fatigue include altering the dose; prescribing another type or class of medication; and offering counseling or referral, particularly if the fatigue is associated with depression.

The patient with ACS requires additional guidance about resuming sexual activity safely. For the patient with unstable angina, nitroglycerin may be taken before intercourse if intercourse causes angina. For the post-MI patient, guidelines for sexual activity are based on successful progression through a home walking or structured outpatient exercise program. Traditional parameters for resuming intercourse include being able to climb two flights of stairs or walk at a pace of 3 to 4 miles/hr without dyspnea or chest pain. The patient's spouse or partner, who may also have fears about the effects of sexual activity on the patient's heart, should be included in all counseling and educational sessions (see the Patient/Family Teaching box).

Beta-blockers cause impotence in some men. The nurse is honest in communicating the side effects of these drugs, since the patient who is aware of the possibility of impotence may be better able to cope with the problem should it occur. Herbal supplements, marijuana, and cocaine are additional drugs that may alter sexual function and place the myocardium at risk. Patients should consult with their primary care provider before using sildenafil (Viagra) or any other drug for erectile dysfunction because of their vasodilatory effects.

Research


Researchers compared the impact of three cardiac rehabilitation approaches on cardiovascular risk factors in 84 patients who had undergone bypass surgery or percutaneous coronary intervention. The traditional program studied included the first three of a standard four-phase approach: phase 1, in-hospital walking and bed exercises; phase 2, supervised outpatient aerobic exercise for 1 to 3 months; phase 3, supervised exercise in a community setting for 6 to 12 months; and phase 4 (not studied), lifelong fitness and exercise programs. The Ornish approach includes a low-fat vegetarian diet; stress management techniques; moderate aerobic exercise, and group support meetings. The third group was a control group who chose not to participate in these two options, and returned home without additional structured rehabilitation.

Data were collected at baseline, 3 months, and 6 months and included blood lipids, glucose concentrations, diet (3-day diet diary), weight, body mass index (BMI), waist-to-hip ratio, blood pressure, exercise participation, anginal pain frequency, and adherence. Ornish program participants had significantly greater reductions in anginal frequency, body weight, BMI, systolic blood pressure, total cholesterol, low-density lipoprotein cholesterol, glucose, and dietary fat and increases in complex carbohydrates. The traditional rehabilitation group had significant reductions in anginal pain severity and waist-to-hip ratio and increased high-density lipoprotein cholesterol, but they also demonstrated significantly increased body weight, BMI, and systolic blood pressure. The control group experienced the greatest reduction in anginal pain severity, but also had significantly higher systolic blood pressure, total cholesterol, and low-density lipoprotein cholesterol.

A major limitation of this study was self-selection into the three groups. The groups were matched for income and demographic measures; however, motivation and education may have confounded the findings. Nevertheless, the study reinforced the value of cardiac rehabilitation, since both the Ornish program and the traditional rehabilitation program (to a less extent) helped participants deal positively with cardiovascular risk factors.
**Nursing Diagnosis: Risk for Injury**

**RELATED NIC INTERVENTIONS**

In addition to monitoring for bleeding complications, the nurse acts to prevent patient injury. The nurse assesses in all transfers to ensure minimum abrasion to skin surfaces. The nurse limits the number of venipunctures and applies direct manual pressure to the puncture site until complete hemostasis is obtained. Arterial punctures are avoided once thrombolytic therapy is begun, especially at sites that cannot easily be compressed to control bleeding.

Anticoagulation therapy is often used in the treatment of ACS. Anticoagulation prevents future clot formation but does not lyse existing clots. Nursing interventions for the patient receiving anticoagulants (e.g., heparin) are the same as those for the patient receiving thrombolytics. During the administration of intravenous unfractionated heparin, the nurse monitors the patient’s partial thromboplastin time (PTT) to evaluate the therapy’s effectiveness. The nurse follows established algorithms and adjusts the dosage of heparin to keep the PTT in the therapeutic range of 50 to 70 seconds. If patients are receiving warfarin (Coumadin) for other health problems, it is important to ensure that their INR (international normalized ratio) is less than 1.6 before they undergo any invasive procedure.

Antithrombotic therapy, both aspirin and other drugs, is often used for ACS and PCH. The purpose of antithrombotic therapy is to minimize clot formation, especially in the area of unstable plaque or at the site of coronary intervention. Nursing interventions include physical assessment for bleeding, prevention of physical injury, and maintenance of hemostasis of puncture sites. The nurse reminds patients to read over-the-counter product labels carefully to avoid using any other aspirin-containing product.

**Coronary Artery Disease and Dysrhythmias**

**Chapter 29**

**Patient/Family Teaching**

**Home Walking Program**

- Count pulse: Take your pulse before, during, and immediately after your walk. Stop and rest if your heart rate is higher than 20 beats/min over resting heart rate, and then continue at a slower pace.
- Safety: Carry your nitroglycerin with you and use as directed if symptoms occur.
- Warm-up: Start with 1 minute of arm and chest exercises followed by 4 or 5 minutes of stationary walking. This gradually increases blood flow to the muscles, preventing injury.
- Duration: Walk at moderate intensity for 5 to 10 minutes. Increase your time by 1 to 2 minutes each time you walk with a goal of a 30- to 45-minute walk.
- Intensity: Stay within a heart rate not higher than 20 beats/min above your resting heart rate and less than 120 beats/min initially. If you are taking beta-blockers, stay within 20 beats/min of your baseline.
- Cool down: Cool down with 5 to 10 minutes of low-intensity walking followed by stretching. The purpose is to gradually decrease effort and prevent a drop in blood pressure, causing dizziness.
- General tips: Preferably walk on a level surface. If you must walk uphill, go more slowly. Walk at least three times per week. In the summer do not walk if the temperature is higher than 85°F or if the humidity is higher than 75%. Wear loose clothing. Drink plenty of water to prevent dehydration. In the winter do not walk outside if the temperature is lower than 40°F. Wear a hat and a face scarf. Avoid exercise for 1 to 2 hours after eating. Patients with diabetes should have a light snack before walking. Do not use tobacco for 1 hour before exercise.

Patients with CAD frequently have numerous concerns related to sexuality. They may be concerned about the occurrence of chest pain during sexual intercourse or their ability to perform sexually. If the patient and the nurse have established a therapeutic relationship, the nurse is usually able to address these concerns with the patient. The nurse reassures the patient that concerns about sexuality after MI or with the diagnosis of CAD are normal and that it is particularly important to discuss them openly with his or her partner.

**RELATED NIC INTERVENTIONS**

Cardiac Care: Rehabilitative, Energy Management, Exercise Promotion, Teaching. Prescribed Activity/Exercise

**Nursing Diagnosis: Risk for Injury**

**OUTCOMES**

A common example of an expected outcome for the patient with a diagnosis of risk for injury (bleeding) is:

- Not experience bleeding, or bleeding will be effectively controlled and treated if it occurs.

**NURSING INTERVENTIONS**

The patient who receives thrombolytic therapy has an increased risk of bleeding, and the nurse has primary responsibility for frequently assessing the patient for any indications of bleeding. Relevant findings include the onset of bleeding from the nose or gums, excessive bruising, frank blood in the urine or guaiac-positive stool, unexplained hypotension or tachycardia, and a rigid abdomen. Subtle symptoms such as headache and visual disturbances may be indicative of cerebral hemorrhage and require evaluation with cerebral imaging. CBC and blood coagulation studies are performed at prescribed intervals and monitored for trends indicative of bleeding.

If the patient and the nurse have established a therapeutic relationship, the nurse is usually able to address these concerns with the patient. The nurse reassures the patient that concerns about sexuality after MI or with the diagnosis of CAD are normal and that it is particularly important to discuss them openly with his or her partner.

**Related NIC Interventions**

Bleeding Precautions, Environmental Management, Medication Management, Risk Identification

**Patient/Family Teaching**

Patient and family teaching is one of the most important aspects of the nursing care provided to patients who are experiencing CAD. Teaching is a priority during the diagnostic, emergency room, coronary care unit, hospital, and rehabilitative phases of care. The nurse needs to be extremely knowledgeable about the disease and its pharmacologic and ventilatory management to help patients and families become full partners in disease management. Relevant aspects of teaching are discussed under each nursing diagnosis and highlighted in the Patient/Family Teaching boxes.

After the patient’s condition stabilizes, the nurse makes appropriate referrals for inpatient cardiac rehabilitation and initiates discharge planning. The experienced staff nurse recognizes when she or he is able to meet the patient’s needs and when it is more appropriate to refer the patient to someone with greater expertise, ability, or time for either immediate crisis intervention or long-term follow-up care.
Cardiovascular Problems

SIT SPARINGLY
• watch TV
• play computer games

ENJOY LEISURE
ACTIVITIES
• golf
• bowling
• yardwork

STRETCH/
STRENGTHEN
• push-ups
• weight lifting

STRETCH/
STRENGTHEN
• curl-ups
• push-ups
• weight lifting

DO AEROBIC ACTIVITIES
• long walks
• biking
• swimming

ENJOY RECREATIONAL
SPORTS
• tennis
• racquetball
• basketball

EVERYDAY
• take extra steps in your day
• walk the dog
• take the stairs instead of the elevator
• park your car farther away and walk

Start your weekly activity plan with the daily activities at the base of the pyramid. Enhance your fitness by choosing other activities on the pyramid. Move more, sit less.

Figure 29-13 Activity pyramid.

EVALUATION
To evaluate the effectiveness of nursing interventions, compare patient behaviors with those stated in the expected patient outcomes.

RELATED NOC OUTCOMES. Activity Tolerance, Anxiety Level, Blood Coagulation, Cardiac Disease Self-Management, Comfort Level, Knowledge: Medication, Pain Control, Risk Control, Stress Level

GERONTOLOGIC CONSIDERATIONS
The prevalence of CAD increases with age. In assessing chest pain in the older adult, the nurse is aware that older adults may experience atypical signs and symptoms and often delay seeking care. Older patients often experience “silent MIs” and come to the emergency department with shortness of breath, heart failure, or pulmonary edema, but without chest pain. Absence of chest pain as a classic symptom often impedes recognition of the older person’s heart attack. Older adults may therefore delay seeking medical care for the evaluation of their “heart condition,” especially when they have a long history of angina. Older adults also may delay seeking care because they are reluctant to go to the hospital, do not want to bother anyone, or are lonely and depressed. Diminished cardiac reserve and altered response to inotropic medications place the older patient at risk for heart failure or cardiogenic shock.

Older adults may also be especially sensitive to certain medications. The nurse carefully observes for side effects and drug interactions and anticipates that the older patient may require higher doses of vasoactive agents to achieve desired effects. However, secondary prevention interventions appear to be just as effective in older adults as in younger patients, and nurses should encourage older adults to get involved in secondary prevention programs to fully realize their rehabilitation potential. (1

Cardiac Dysrhythmias

Etiology and Epidemiology. Normal sinus rhythm (NSR) begins with the spontaneous depolarization of the SA node. The impulse passes through the atria to the AV node and then through the bundle of His and bundle branches to the Purkinje fibers (see Figure 28-4). A rhythm is classified as “normal” when it meets the following criteria: presence of one upright and consistent-appearing P wave before each QRS complex, all P-R intervals between 0.12 and 0.20 second, a consistent-appearing QRS complex of less than 0.12 second, a consistent R-R interval, and a heart rate between 60 and 100 beats/min (Figure 29-14). All rhythm strips displayed are from lead II.

Cardiac dysrhythmias occur as the result of alterations in impulse formation or propagation. The anatomic site of the dys-
PATIENT/FAMILY TEACHING Guidelines for Sexual Activity After Myocardial Infarction

Stages of Sexual Response

Arousal:flushed;breathing and heart rate increase; blood pressure goes up slightly
Plateau: increase in respirations, blood pressure, and heart rate
Orgasm (15 to 20 seconds): further increases in pulse and blood pressure
Resolution: return to resting state within seconds; angina or palpitations more likely to occur during resolution

General Guidelines

Sexual foreplay at a relaxed pace allows your heart rate and blood pressure to increase more slowly.
Hugging, stroking, and touching are safe ways to get back in touch with your partner.
Talk with your partner. Express your feelings.
External sexual affairs or sex with new partners may produce more stress.
Avoid positions for sex that you find uncomfortable.
Have sex in a pleasant, comfortable environment.
Do not take very hot or cold baths or showers before or after sex. Be rested before sex.
Do not have sex after a heavy meal or drinking alcohol.
If you have any questions about side effects of any drug, do not stop taking the drug, but talk to your health care provider.
Masturbation and manual or oral stimulation are not harmful to your heart. Anal intercourse may lead to an irregular heart-rate (Figure 29-16).

The result may be premature beats or tachycardias. Some causes of enhanced automaticity are hypoxia, catecholamines, atropine, hypokalemia, hypocalcemia, heat, trauma, and digitalis toxicity.

Erectile dysfunction is more prevalent in patients with cardiac disease. In patients with CAD a benign rhythm may have negative consequences because the myocardium is already compromised. Common dysrhythmias and their management are presented in the Collaborative Care Management section.

Pathophysiology. An understanding of normal cardiac electrophysiology, presented in Chapter 28, is necessary to grasp the pathophysiology of dysrhythmias. Alterations in impulse formation and propagation arise from one of three main pathophysiologic processes: altered automaticity, altered conduction resulting in delays or blocks, and reentry mechanisms.

Alterations in Automaticity. Automaticity, the ability to depolarize spontaneously without external stimulation, is a property normally confined to the cells of the SA node. Depolarization, however, is not unique to the SA node and occurs all along the electrical impulse pathway (Figure 29-15). These nonpacemaker cells may be responsible for dysrhythmias. The SA node usually depolarizes at a faster rate than other potential pacemaker cells because of the steep slope of phase 4, allowing sinus cells to reach threshold at a faster rate (Figure 29-16). A variety of conditions can alter the automaticity of the SA node and produce faster or slower than usual heart rates. Vagal stimulation decreases this slope, resulting in a slower heart rate (Figure 29-17). Sympathetic stimulation and hypoxia steepen phase 4, resulting in a faster heart rate (Figure 29-18).

If the rate of phase 4 depolarization found in the AV node or ventricular conduction system increases, enhanced automaticity is said to exist. The result may be premature beats or tachycardias. Some causes of enhanced automaticity are hypoxia, catecholamines, atropine, hypokalemia, hypocalcemia, heat, trauma, and digitalis toxicity.

Even cells that do not normally have automaticity may develop abnormal automaticity if the resting membrane potential or threshold potential is altered. Making the threshold potential less negative slows the heart rate, since more time is needed to reach threshold (Figure 29-18). Abnormal automaticity is not easily suppressed by the activity of the usual pacemakers.

One variation of automaticity often associated with ventricular dysrhythmias is afterdepolarization. Afterdepolarizations arise from fluctuations in the cellular membrane potential occurring after phase 0 has been initiated. If the fluctuation reaches threshold amplitude, an early action potential, an “afterdepolarization,” occurs. Afterdepolarizations can occur soon after phase 0 is initiated or later, after repolarization is complete. Delayed afterdepolarizations are often associated with increased intracellular calcium, catecholamines, and digitalis toxicity.

Alterations in Conduction. When the rate or amplitude of depolarization decreases, conduction also decreases. Any condition that decreases the amplitude of the action potential, such as ischemia, hypercalcemia, or calcification of the conducting fibers,
Figure 29.15 Phases of action potential of a cardiac cell. In resting phase (4), cell membrane is polarized. Cell’s interior has net negative charge, and membrane is more permeable to potassium ions ($K$) than to sodium ($Na$). When cell is stimulated and begins to depolarize (0), sodium ions enter cell, potassium leaves cell, calcium (Ca) channels open, and sodium channels close. In its depolarized phase (1), cell’s interior has net positive charge. In plateau phase (2), calcium and other positive ions enter cell and potassium permeability declines, lengthening action potential. Then (3), calcium channels close and sodium is pulled from cell by sodium-potassium pump. Cell’s interior then returns to its polarized, negatively charged state (4).

Figure 29.16 Action potential recorded from pacemaker cell.

Figure 29.17 Decreased automaticity. Left curve: Normal action potential recorded from pacemaker cell. Right curve: Vagal stimulation decreases rate of phase 4 depolarization, decreasing heart rate.
can cause cardiac conduction disturbances. Abnormalities in conduction can occur anywhere in the conduction system, including the SA node, AV node, and bundle branches. The severity of the impaired conduction ranges from a slight delay to complete cessation or block of impulse transmission.

**Reentry.** Reentry involves impulse transmission around a unidirectional block. Reentry occurs when an impulse is delayed within a pathway of slow conduction long enough that the impulse is still viable when the remaining myocardium repolarizes. The impulse then reenters surrounding tissue and produces another impulse. This typically occurs when two different pathways share an initial and final segment. The first impulse travels down the faster pathway, leaving behind its refractory tail. Should a second, early impulse follow, it is blocked because that path is refractory. The second impulse then enters the slow pathway and can return retrogradely through the fast path, initiating a circumferential pattern (Figure 29-21).

**Clinical Manifestations.** Many patients with dysrhythmias are asymptomatic as long as cardiac output meets the body's metabolic demands. The clinical manifestations associated with most dysrhythmias relate to decreases in cardiac output from slow or fast heart rates (see Clinical Manifestations box). Significant changes in heart rate may not allow adequate time for the ventricles to fill and empty. In addition, patients may complain of palpitations (e.g., a “racing heart” or “skipping beats”) related to changes in heart rate and stroke volume. These symptoms often create acute anxiety.

![Figure 29-18](image1.png)  
**Figure 29-18** Increased automaticity. Left curve: Sympathetic stimulation and hypoxia steepen phase 4 depolarization, increasing heart rate. Right curve: Normal action potential recorded from pacemaker cell.

![Figure 29-19](image2.png)  
**Figure 29-19** Decreased automaticity. A, Normal action potential recorded from nonpacemaker cell. B, Making threshold potential (TP) less negative increases time needed to reach threshold, decreasing heart rate. RMP, Resting membrane potential.

![Figure 29-20](image3.png)  
**Figure 29-20** Increased automaticity. A, Normal action potential recorded from nonpacemaker cell. B, Making resting membrane potential (RMP) less negative makes it easier to reach threshold, increasing heart rate. TP, Threshold potential.

![Figure 29-21](image4.png)  
**Figure 29-21** Reentry. A, Shaded area shows refractory area after first impulse passes down path 1. Premature impulse is then blocked from entering path 2 but can travel down path 2. B, Path 1 is no longer refractory to stimulation; therefore premature impulse can travel backward up path 1. C, Reentry down path 2 establishes circumferential pathway.
Collaborative Care Management. The diagnosis of dysrhythmias begins with the 12-lead ECG. Each dysrhythmia exhibits characteristic changes in the ECG tracing. A systematic approach to analyzing the ECG rhythm helps distinguish the different dysrhythmias (Box 29-3). Table 29-8 outlines the rhythm criteria that define each common dysrhythmia and their common associated causes. Some rhythms, especially fast rhythms, seem to defy interpretation using the ECG alone. Additional diagnostic tests (Box 29-4) are often needed to determine the dysrhythmia itself and, most important, its cause. These tests are further discussed in Chapter 28. Electrophysiology studies are used to determine the electrophysiologic properties of the various dysrhythmias. Management is then determined based on an understanding of the mechanism responsible for the dysrhythmia.

The collaborative management of dysrhythmias focuses on alleviating symptoms produced by altered cardiac output and eliminating or reversing the cause. Common interventions specific to each dysrhythmia are included in the discussion that follows.

SINUS BRADYCARDIA. Sinus bradycardia is characterized by atrial and ventricular rates of less than 60 beats/min (Figure 29-22), but in all other respects is a NSR. It may develop gradually or occur suddenly for a brief period. Bradycardia generally results from increased vagal tone or decreased sympathetic tone. It is commonly seen in athletes and may also be associated with sleep, vomiting, and MI. Carotid sinus stimulation and drugs such as digoxin, morphine sulfate beta blockers, and sedatives induce sinus bradycardia in many patients.

Generally sinus bradycardia is a benign rhythm. In association with MI it may even be a beneficial rhythm because it reduces myocardial oxygen demand. If the heart rate is too slow to maintain adequate cardiac output, however, the patient may be predisposed to syncope and heart failure. Administration of atropine is usually effective in increasing the heart rate. Secondary interventions include transcutaneous pacing, dopamine, epinephrine, and isoproterenol. Postcardiac transplant patients with unstable bradycardia will not respond to atropine secondary to denervation of nervous control.

SINUS TACHYCARDIA. Sinus tachycardia is characterized by an atrial and ventricular rate of 100 beats/min or more (Figure 29-23). Generally the upper limit of sinus tachycardia is 160 beats/min. The P waves are sinus in origin, but they may be buried in the T wave with very high heart rates. Intervals and
complexes are within normal limits. The onset of sinus tachycardia usually is gradual, as the sinus node rate increases in response to higher metabolic needs.

Sinus tachycardia is associated with the ingestion of alcohol, caffeine, and tobacco and is a normal physiologic response to exertion, fever, fear, excitement, acute pain, or any condition that requires a higher basal metabolism. Clinically, sinus tachycardia can be a short-term compensatory response to heart failure, anemia, hypovolemia, and hypotension. Sinus tachycardia is also seen with hyperthyroidism and may be produced by drugs such as atropine and amphetamines.

Generally, sinus tachycardia is a benign rhythm that slows with resolution of the cause. The patient may complain of palpitations or have no symptoms. In the patient with a compromised myocardium the tachycardia increases myocardial oxygen demand and may cause a decrease in cardiac output with resultant lightheadedness, chest pain, and heart failure. Sinus tachycardia can usually be slowed with digoxin, beta-blockers, or diltiazem if necessary.

**Sinus Dysrhythmia.** Sinus dysrhythmia is typically found in young adults and older persons. Sinus dysrhythmia is an irregular rhythm in which P-P intervals vary by more than 0.16 second. The P waves have a consistent shape, and the P-R interval and QRS duration are within normal limits. Changes in P-P intervals are accompanied by changes in R-R intervals (Figure 29-24). The cyclic pattern of changing P-P or R-R intervals often correlates with the patterns of inspiration and expiration. During inspiration the intervals shorten as the heart rate increases. Conversely, the intervals lengthen during expiration.

Sinus dysrhythmia is not treated unless the bradycardic phase is severe, causing symptoms. With slower heart rates, some patients may experience palpitations or dizziness if the P-P intervals are unusually long. Atropine may be effective in treating symptomatic bradycardia.

**Sick Sinus Syndrome.** Tachycardia-bradycardia syndromes are characterized by the presence of bradycardia with intermittent episodes of tachydysrhythmias. The episode of tachydysrhythmia often is followed by a long pause before returning to bradycardia. Sick sinus syndrome (SSS) is one type of tachycardia-bradycardia syndrome. In SSS the bradycardia and tachycardia are both sinus in origin. Complications of this inefficient rhythm include heart failure and stroke resulting from thromboembolism. In addition, cerebral blood flow may be decreased, producing confusion in the elderly. SSS is associated with ischemia or degeneration of the SA node.

Some patients may remain free of symptoms or complain only of palpitations. For the patient with severe symptoms, the heart rhythm is stabilized with a permanent implantable pacemaker for the slow phase and the administration of digoxin or beta-blockers to control the ventricular rate of the tachycardic phase.

**Sinus Exit Block and Sinus Arrest.** Sinus exit block occurs when an impulse originates in the SA node but is immediately blocked (Figure 29-25). No P wave or QRS complex is generated, resulting in a long pause. The next impulse occurs in a time interval representing the normal P-P interval. The term sinus arrest implies that the SA node never fired; therefore there is no P or QRS complex. The next impulse is asynchronous to the normal P-P interval.

Sinus exit block and sinus arrest may occur as a result of medications such as digoxin, hypoxia, myocardial ischemia, and injury to the SA node. The patient becomes symptomatic from a decrease in cardiac output when the pauses are long or frequent.
### Dysrhythmias of Sinus Node

<table>
<thead>
<tr>
<th>Dysrhythmia</th>
<th>ECG Diagnostic Criteria</th>
<th>Etiologic Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus bradycardia</td>
<td>P waves present followed by QRS Rhythm regular Heart rate &lt; 60 beats/min</td>
<td>Athletics Vagal stimulation Digitalis, beta-blockers, sedatives</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>P waves present followed by QRS Rhythm regular Heart rate 100-160 beats/min</td>
<td>Increased metabolic demands Compensatory mechanism for heart failure, shock, hemorrhage, anemia</td>
</tr>
<tr>
<td>Sinus dysrhythmia</td>
<td>Phasic shortening of P-P and R-R intervals with inspiration, lengthening with expiration</td>
<td>Respiratory variation in impulse initiation by SA node</td>
</tr>
<tr>
<td>Sick sinus syndrome</td>
<td>Sinus bradycardia alternating with sinus tachycardia</td>
<td>SA node ischemia, degeneration Hypertension Ischemia Digoxin</td>
</tr>
<tr>
<td>Sinus exit block and sinus arrest</td>
<td>Isoelectric line (pause) without P or QRS; P wave returns in synchrony (exit block) or asynchrony (sinus arrest)</td>
<td>Hypoxia Hypopnea Ischemia SA node ischemia, degeneration Digoxin</td>
</tr>
</tbody>
</table>

### Atrial Dysrhythmias

<table>
<thead>
<tr>
<th>Dysrhythmia</th>
<th>ECG Diagnostic Criteria</th>
<th>Etiologic Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature atrial beat</td>
<td>Early P wave QRS may or may not be normal Pause follows QRS</td>
<td>Stress Ischemia Atrial enlargement Caffeine, nicotine</td>
</tr>
<tr>
<td>Wandering atrial pacemaker</td>
<td>P waves of different appearances or buried in QRS; varying P-R intervals</td>
<td>Cardiac disease Drug toxicity</td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>P wave present (may be hidden in previous T wave), QRS usually normal heart rate usually 150-250 beats/min</td>
<td>Sympathetic stimulation, caffeine, nicotine, drug toxicity Pulmonary disease Heart disease</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Atrial rate 240-480 beats/min; F waves usually in a ratio to QRS complexes such as 2:1, 3:1; QRS complexes normal</td>
<td>Pulmonary disease Valve disease Cardiac surgery</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Rapid, indiscernible P waves (&gt; 350 beats/min) Ventricular rhythm irregularly irregular Ventricular rate varies</td>
<td>Rheumatic heart disease Atrial ischemia Coronary atherosclerotic disease Hypertension Thyrotoxicosis Cardiac surgery Alcohol</td>
</tr>
</tbody>
</table>

### Junctional Dysrhythmias

<table>
<thead>
<tr>
<th>Dysrhythmia</th>
<th>ECG Diagnostic Criteria</th>
<th>Etiologic Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature junctional beat</td>
<td>Early beat P before, during, or after QRS P inverted or retrograde P-R interval &lt; 0.12 sec if P before QRS QRS normal</td>
<td>Increased metabolism Nicotine, caffeine Ischemia Electrolyte imbalance</td>
</tr>
<tr>
<td>Junctional rhythm</td>
<td>P before, during, or after QRS P inverted or retrograde</td>
<td>Accelerated: Heart disease</td>
</tr>
</tbody>
</table>
Dysrhythmia ECG Diagnostic Criteria Etiologic Factors
---
**Junctional rhythm—cont’d**
- P-R interval < 0.12 sec if P before QRS
- QRS normal
- Rate 40-60 beats/min: junctional rhythm
- Rate 60-100 beats/min: accelerated junctional rhythm
- Rate > 100 beats/min: junctional tachycardia
- Caffeine
- Pain
- Digitalis

**Ventricular dysrhythmias**
- Premature ventricular beats
  - Early, wide, bizarre QRS, not associated with P wave
  - Rhythm irregular
- Stress, acidosis, ventricular enlargement
- Electrolyte imbalance
- Myocardial infarction
- Digitalis toxicity
- Hypoxemia, hypercapnia
- Digoxin
- Pain
- Hypertension

- Accelerated idioventricular rhythm (AIVR)
  - P not associated with QRS, QRS wide and bizarre
  - VT: ventricular rate > 100, usually 140-240 beats/min
  - AIVR: rate 40-100 beats/min
- VT: hypoxemia, drug toxicity, electrolyte imbalance, bradycardia, coronary artery disease
- AIVR: reperfusion of ischemic myocardium

- Torsades de pointes
  - No associated P waves
  - Wide, bizarre QRSs twist along isoelectric line
  - Heart rate > 100 beats/min
- Myocardial infarction
- Ventricular fibrillation
  - No recognizable complexes
  - Wavy line of varying amplitude
- VT: hypoxemia, drug toxicity, electrolyte imbalance, bradycardia, coronary artery disease
- AIVR: reperfusion of ischemic myocardium

- Ventricular asystole
  - No complexes
  - "Straight line"
- Myocardial infarction
- Chronic diseases of conducting system

**Impulse conduction deficits**
- First-degree AV block
  - P-R interval prolonged, > 0.20 sec
- Rheumatic fever
- Myocardial infarction
- Cardiac medications

- Second-degree AV blocks
  - Mobitz I
    - P waves usually occurring regularly at rates consistent with SA node initiation
    - P-R interval lengthened before nonconducted P wave
    - QRS may be widened
  - Acute myocardial infarction
  - Increased vagal tone
  - Electrolyte imbalance
  - Infection
  - Digitalis toxicity
  - Coronary artery disease
  - Myocardial infarction
  - Rheumatic heart disease
  - Hypoxia

  - Mobitz II
    - Constant P-R intervals
    - Nonconducted P waves at random or patterned intervals
  - Coronary artery disease
  - Myocardial infarction
  - Rheumatic heart disease
  - Digitalis toxicity
  - Coronary artery disease
  - Myocardial infarction

- Complete third-degree AV block
  - P waves have no relation to QRS
  - Ventricular rate as low as 20-40 beats/min if ventricular; 40-60 beats/min if junctional
  - Hypoxia
  - Acute myocardial infarction
  - Myocardial infarction

- Bundle branch block
  - Same as normal sinus rhythm except QRS duration > 0.12 sec
  - Coronary artery disease
  - Myocardial infarction

---

**Table 29-8 Comparison of selected cardiac dysrhythmias—cont’d**

<table>
<thead>
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<td>Junctional rhythm—cont’d</td>
<td>P-R interval &lt; 0.12 sec if P before QRS</td>
<td>Caffeine</td>
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<td>QRS normal</td>
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<td>Rate 40-60 beats/min: junctional rhythm</td>
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<td>Ventricular dysrhythmias</td>
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<td>Early, wide, bizarre QRS, not associated with P wave</td>
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<td>Accelerated idioventricular rhythm (AIVR)</td>
<td>P not associated with QRS, QRS wide and bizarre</td>
<td>VT: hypoxemia, drug toxicity, electrolyte imbalance, bradycardia, coronary artery disease</td>
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<tr>
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<td>VT: ventricular rate &gt; 100, usually 140-240 beats/min</td>
<td>AIVR: reperfusion of ischemic myocardium</td>
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<td>AIVR: rate 40-100 beats/min</td>
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<td>Torsades de pointes</td>
<td>No associated P waves</td>
<td>Myocardial infarction</td>
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<td></td>
<td>Wide, bizarre QRSs twist along isoelectric line</td>
<td>Electrolyte imbalance</td>
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<td>Heart rate &gt; 100 beats/min</td>
<td>Congenital long Q-T interval</td>
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<td>Ventricular fibrillation</td>
<td>No recognizable complexes</td>
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<td>Wavy line of varying amplitude</td>
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<td>Second-degree AV blocks</td>
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<td>Mobitz I</td>
<td>P waves usually occurring regularly at rates consistent with SA node initiation</td>
<td>Acute myocardial infarction</td>
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<td>P-R interval lengthened before nonconducted P wave</td>
<td>Increased vagal tone</td>
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<td>QRS may be widened</td>
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<td>Infection</td>
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<td>Mobitz II</td>
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<td>Nonconducted P waves at random or patterned intervals</td>
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<td>Rheumatic heart disease</td>
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<td>Coronary artery disease</td>
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<td>Myocardial infarction</td>
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<tr>
<td>Complete third-degree AV block</td>
<td>P waves have no relation to QRS</td>
<td>Digitalis toxicity</td>
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<tr>
<td></td>
<td>Ventricular rate as low as 20-40 beats/min if ventricular; 40-60 beats/min if junctional</td>
<td>Coronary artery disease</td>
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<td>Myocardial infarction</td>
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<tr>
<td>Bundle branch block</td>
<td>Same as normal sinus rhythm except QRS duration &gt; 0.12 sec</td>
<td>Hypoxia</td>
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<td>Acute myocardial infarction</td>
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*ECG, Electrocardiogram; SA, sinoatrial; AV, atrioventricular.*
The patient may feel palpitations from the increased stroke volume that accompanies the next beat after the pause. When the patient is symptomatic, atropine may be administered to increase the heart rate and cardiac output. Definitive therapy includes insertion of a permanent pacemaker.

**Premature Atrial Beat.** A premature atrial beat (PAB) is initiated by an ectopic focus in the atria (Figure 29-26) and is characterized by a premature P wave with a contour different from that of a sinus P wave. The location of the ectopic focus within the atria determines its shape. The QRS complex may or may not be normal. The PAB is often followed by a pause. The atrial impulse may be nonconducted (blocked) because of refractoriness of the AV node at the time the impulse arrives. The nonconducted atrial beat (blocked PAB) is a common cause of irregularity in the heart rhythm.

The patient may feel palpitations from the increased stroke volume that accompanies the next beat after the pause. When the patient is symptomatic, atropine may be administered to increase the heart rate and cardiac output. Definitive therapy includes insertion of a permanent pacemaker.

**PAB**

The PAB may be associated with stress or the use of caffeine or tobacco products. It also is seen in the clinical setting with hypoxia, atrial enlargement, infection, inflammation, and myocardial ischemia. Frequent PABs may warn of impending atrial fibrillation (AF) or tachycardia. In the absence of organic disease, no treatment is required. Often the elimination of caffeine and tobacco will suppress the atrial focus. Premature atrial beats may produce palpitations, but cardiac output is generally not affected unless PABs or blocked beats are frequent.

**Wandering Atrial Pacemaker.** Wandering atrial pacemaker occurs when at least three ectopic sites create impulses for the cardiac rhythm (Figure 29-27). The ECG shows P waves of different shapes and P-R intervals of different lengths. The impulse can originate from the area around the AV node, which creates inverted P waves from retrograde conduction. Impulses from this
lower area may also stimulate the atria at the same time as or after the ventricle. The P waves then appear to be buried in the QRS or even occur inverted after the QRS.

Wandering atrial pacemakers usually signify underlying heart disease or drug toxicity. The patient is usually asymptomatic unless the heart rate increases or decreases enough to affect cardiac output. The nurse monitors for changes in the rhythm and in the patient’s symptoms.

**Atrial Tachycardia.** In atrial tachycardia the atrial rate is approximately 150 to 250 beats/min. P waves are present but may be hidden in the T waves of the preceding beats when the ventricular rate is high. When the P waves vary in appearance, the rhythm is called multifocal atrial tachycardia. The QRS complex generally is normal, and the ventricular rhythm is regular (Figure 29-28). Transient episodes of atrial tachycardia occur in young adults in the absence of heart disease. The dysrhythmia is associated with rheumatic heart disease, pulmonary disorders, stress, hypoxia, caffeine, marijuana, and sympathomimetics.

The patient may complain of palpitations, lightheadedness, and anxiety during a tachycardic episode. Short, infrequent episodes require no treatment. Generally, hemodynamic changes are not severe unless the episode is prolonged, the rate is greater than 200 beats/min, or underlying disease exists. Lengthy episodes may respond to carotid sinus pressure or vagal stimulation. Some patients can be taught to perform Valsalva’s maneuvers to slow the rate. Adenosine may be used in the acute situation. Depending on the electrophysiology associated with the atrial tachycardia, one of the following interventions is generally selected: AV nodal blockade with beta-blockers, calcium channel blockers, or digoxin; cardioversion; or antidysrhythmics (procainamide [Pronestyl], amiodarone [Cordarone], or sotalol [Betapace]). For long-term management, symptomatic atrial tachycardia arising from reentry is treated with beta-blockers or calcium channel blockers. If these agents do not control the dysrhythmia, ablation of the ectopic focus with or without pacemaker insertion may be recommended instead of additional antidysrhythmic drugs.

Atrial tachycardia with block is characterized by the same rapid atrial rate, but some impulses are not conducted into the ventricles (i.e., they are blocked). The AV nodal conduction ratio is usually 2:1, producing a ventricular rate of 75 to 125 beats/min. This dysrhythmia is associated with organic heart disease, and both digitalis toxicity and potassium deficit can cause it. Treatment depends on the clinical picture and often is aimed at correcting the underlying cause. Digitalis antibody may be indicated for hemodynamic compromise secondary to digoxin toxicity.

**Atrial Flutter.** In atrial flutter the atria depolarize at a rate of 240 to 400 beats/min. The atrial depolarizations produce flutter (F) waves that give the baseline a sawtooth appearance (Figure 29-29). The QRS configurations are normal. There is no measurable P-R interval because it is difficult to determine electrocardiographically which atrial impulse actually is conducted to the ventricles. With rapid atrial rates, the AV node physiologically prevents conduction of each atrial impulse. The ventricles often respond to the impulses at a regular rate. The number of flutter waves to QRS complexes is expressed as a ratio (e.g., atrial flutter, 3:1 block).

Reentry is the primary pathophysiologic process. Atrial flutter usually indicates underlying disease. It is associated most commonly
with CAD, pulmonary embolism, mitral valve disease, thoracic surgical procedures, and chronic obstructive pulmonary disease.

The potentially rapid or slow ventricular rate of atrial flutter may result in decreased cardiac output. The major goal of treatment is control of the ventricular rate. Diltiazem, digoxin, or beta-blockers usually succeed in slowing the ventricular rate. If these drugs do not slow the heart rate, amiodarone may be tried. Atropine may be used to augment the heart rate when the ventricular response is slow. Drugs used to terminate the rhythm include procainamide, disopyramide (Norpace), propafenone (Rythmol), sotalol, flecainide (Tambocor), amiodarone, dofetilide (Tikosyn), and ibutilide (Corvert). Azimilide, currently under investigation, is a promising treatment for supraventricular dysrhythmias.

Cardioversion is highly successful in converting atrial flutter to sinus rhythm. It may be the initial treatment if the patient is unstable. Care must be taken to prevent cardioembolic events (see Atrial Fibrillation). Pacing may be used when pharmacologic intervention and external cardioversion have been unsuccessful. For long-term management, radiofrequency ablation is often used to interrupt the reentry circuit. This procedure is successful in the majority of cases.

**Atrial Fibrillation.** AF is the most rapid atrial dysrhythmia (Figures 29-30 and 29-31). The atria depolarize chaotically at rates of 350 to 600 beats/min. AF is generated and perpetuated by one or more rapidly firing ectopic foci, with reentry being the pathophysiologic process in many cases. Paroxysmal AF in young adults has been associated with distinct electrically active foci within the pulmonary veins. The baseline in AF is composed of irregular undulations without definable P waves. The QRS complex usually is normal, but the ventricular rhythm is “irregularly irregular.”

AF affects approximately 2.2 million Americans, most of whom are 65 years of age or older. AF may be paroxysmal and transient, or chronic. The latter generally indicates underlying heart disease. AF is typically associated with pericarditis, thyrotoxicosis, cardiomyopathy, CAD, hypertension, rheumatic mitral valve disease, cardiac surgery, heart failure, pulmonary disease, and excessive alcohol intake (“holiday heart”). The underlying cause should be corrected whenever possible.

AF causes irregularity in the ventricular rhythm and impairs the ventricular filling that normally occurs with synchronous atrial contractions (atrial kick), thus decreasing cardiac output. Symptoms include fatigue, dyspnea, and dizziness. Thrombi may form in the stagnant blood in the atria and cause emboli, which can lodge in the pulmonary or peripheral blood vessels. The goal of therapy is to prevent complications through control of the ventricular rate and the restoration of NSR. The severity of the patient’s symptoms, hemodynamic instability, and risk of embolization guide treatment decisions.

Drugs used to control fast ventricular rates include diltiazem, verapamil, digoxin, and beta-blockers. Digoxin is not as effective in controlling the heart rate variations that occur with exercise. In AF with a slow ventricular response, atropine may be necessary to increase the heart rate and cardiac output. When medications are ineffective in controlling the rate and the patient is symptomatic from an ineffective cardiac output, cardioversion may be necessary to restore NSR and a more normal heart rate.

Several antidysrhythmics may be successful in converting AF to NSR. The same drugs may be used to maintain patients in NSR once successful cardioversion occurs. Suggested AHA guidelines for the indication of these drugs are summarized in Box 29-5.3 These drugs can have a prodysrhythmic effect. Therefore patients require careful monitoring, often within the hospital.

External cardioversion (see p. 000) is the most commonly used nonpharmacologic approach for restoring NSR. Internal atrial defibrillation is another treatment option. The surgical maze procedure may also be used, where sinus impulses are rerouted to the AV node through channels created by multiple atrial incisions. Radiofrequency catheter ablation, which isolates and treats specif-
Ablation of the AV node with subsequent placement of a permanent pacemaker may be used in individuals with permanent symptomatic AF. This procedure does not, however, abolish the fibrillatory activity of the atria. Other treatment options include single and dual site atrial pacing and newer implantable atrial defibrillators.

The risk of systemic emboli is high with persistent AF. Patients ideally are stabilized on warfarin therapy for 4 weeks with an INR goal of 2 to 3 before an elective pharmacologic or electrical cardioversion attempt. If the patient is hemodynamically unstable or has refractory symptoms, however, the need to electrically cardiovert may take priority. Transesophageal echocardiography may be helpful in determining the presence of atrial thrombi. If no thrombi are found, the patient may be electrically cardioverted. After successful conversion to NSR, thrombi may still form until the atria contract effectively and in synchrony. Therefore anticoagulation therapy is continued for at least 4 weeks after conversion to NSR. If conversion to NSR is unsuccessful, the patient is maintained indefinitely on warfarin therapy. If the patient cannot tolerate warfarin, aspirin therapy, with a daily dose of 325 mg, is recommended. Ximelagatran, an oral thrombin inhibitor, is currently under investigation as an alternative to warfarin therapy.

**Premature Junctional Beats.** Premature junctional beats (PJBs) arise from an ectopic focus either (1) at the junction of the atria and the AV node or (2) at the junction of the AV node and the bundle of His. If the PJBs arise from the first junction, the P wave will be inverted and premature and will precede the QRS complex. In the second case, the P wave is either hidden in the QRS or is inverted and follows the QRS (Figure 29-32). The abnormal timing and inversion of the P wave are caused by depolarization of the atria in a retrograde fashion. The QRS is normal, but the P-R interval is less than 0.12 second.

PJBs may occur in the normal heart. They also may result from digitalis toxicity, ischemia, hypoxia, pain, fever, anxiety, nicotine, caffeine, or electrolyte imbalance. Treatment, when needed, is directed toward correcting the underlying cause.

**Junctional Rhythms.** When the SA node fires at a rate less than 40 to 60 beats/min, the automatic cells in the AV junction...
may initiate impulses (escape beat) to stabilize the rhythm. A
succession of beats from the junction is a junctional escape
rhythm. The P waves may occur before, during, or after the QRS.
The QRS is normal, and the ventricular rhythm is regular. A junc-
tional escape rhythm occasionally is found in the well-trained ath-
lete or as a complication of an acute inferior wall MI. Junctional
escape rhythm generally is not treated unless the loss of atrial kick
produces symptoms of low cardiac output. These patients may
require artificial pacing.

When the automaticity of a junctional pacemaker increases to
a rate greater than 60 beats/min, it may usurp the SA node as the
cpacemaker of the heart. A rate of 60 to 100 beats/min is called an
accelerated junctional rhythm (Figure 29-33). An accelerated
junctional rhythm may be due to heart disease, pain, anemia, caf-
fine, or amphetamines.

A junctional tachycardia exists when the rate exceeds 100
beats/min. Junctional tachycardia is associated with digitalis toxi-
city, acute rheumatic fever, and heart disease; treatment is aimed
as the underlying cause. If the rate is interfering with cardiac out-
put, vagal maneuvers may be attempted followed by digoxin,
beta-blockers, or chlorthalidone.

Both junctional tachycardia and AT may be collectively
referred to as supraventricular tachycardia (SVT), indicating that
the rhythm originates above the ventricles. Symptomatic SVT
from reentry may be treated with beta-blockers or calcium chan-
nel blockers. If these agents do not control the dysrhythmia, abla-
tion of the irritable focus with or without pacemaker insertion
may be recommended instead of antidyssrhythmics.

PREMATURE VENTRICULAR BEATS. A premature ventricular beat
(PVB) is an early beat arising from an ectopic focus in the
ventricles. The characteristic wide, bizarre QRS (usually greater
than 0.12 second) makes the PVB readily identifiable on the ECG
tracing. There is no associated P wave, and the T wave records in
the opposite direction from the main QRS deflection. Most PVBs
are followed by a pause until the next normal impulse originates
in the SA node.

If PVBs are of different configurations on the ECG tracing,
they are said to be multifocal. This indicates the presence of more
than one ectopic focus in the ventricles, or one ectopic focus with
multiple reentry pathways, each producing complexes of differing
forms. Premature ventricular beats also may exhibit varying
degrees of prematurity. The relationship of the PVB to the Q, R,
S, and T waves of the preceding beat is important. An electrical
impulse of any kind that stimulates the heart near the peak of the
T wave (thereby preventing full repolarization of the ventricles)
may precipitate a more dangerous or lethal dysrhythmia. The fre-
quency and morphology of PVBs determine their importance.
When every other beat is a PVB, the term bigeminy is used; every
third beat, trigeminy, and so forth (Figure 29-34). Two PVBs
together are termed a couplet.

PVBs occur in the absence of heart disease and increase in
number with age. However, the incidence and frequency of occur-
rence are higher in the population with heart disease. Clinically,
PVBs are associated with AMI, heart failure, digitalis toxicity,
hypoxia, stimulants, catecholamines, and electrolyte imbalances.
In the latter cases treatment of the underlying cause may abolish
the dysrhythmia.

VENTRICULAR RHYTHMS AND TACHYCARDIA. If the SA node
and AV junction fail to initiate impulses, a ventricular pace-
maker cell automatically begins to initiate impulses at a rate of
20 to 40 beats/min. This is known as an idioventricular rhythm
(Figure 29-35). P waves, when seen, are not associated with the
ventricular rhythm, and the QRS complex is greater than 0.12
second, wide, and bizarre.

If the rate of the ventricular-initiated rhythm increases to 40 to
100 beats/min, it is known as an accelerated idioventricular
rhythm (AIVR). An AIVR may be seen in hypoxia, digitalis
toxicity, as a complication of an AMI, and as a reperfusion dys-
rhythmia after thrombolytic therapy. Suppression of the heart's
dominant and perhaps only rhythm could be hazardous. There-
fore idioventricular rhythms are not treated except to correct
underlying abnormalities.

If the cardiac output is low and symptoms of heart failure, syn-
copes, or hypotension develop, the patient may require a tempo-
rary or permanent pacemaker. Atropine may be helpful in stimu-
lating the return of SA node activity.

By definition, three or more successive PVBs constitute ven-
tricular tachycardia (VT) (Figure 29-36). The ventricular rate is
regular or slightly irregular, and is greater than 100 beats/min,
usually 140 to 240 beats/min. P waves may be present but are not
associated with the QRS complexes. VT may complicate any
form of heart disease and may be a direct result of a PVB striking
during the heart's action potential vulnerable period. Conditions
that favor its occurrence include hypoxemia, drug toxicity, elec-
trolyte imbalance, and bradyarrhythmia. Abnormal automaticity can
occur in the postinfarction period from the loss of fast depolariz-
ing sodium channels, contributing to the development of VT. VT
can also be attributable to ischemia, nonischemic heart disease,
and drugs, and can even be found in the structurally normal heart
(e.g., long QT syndrome). Rhythm is often involved. Treatment is
based on the underlying electrophysiology of the dysrhythmia,
which may be difficult to establish.

Figure 29-33 Accelerated junctional rhythm with hidden P waves; heart rate, 70 beats/min.
VT is classified as sustained (lasting more than 30 seconds) or nonsustained. Nonsustained VT may occur in patients with or without cardiac disease and is associated with palpitations or recurrent syncope. In the presence of severe ventricular dysfunction, nonsustained VT may be a precursor to sustained VT and sudden death. As the heart rate increases, cardiac output decreases, since the ventricles do not have sufficient time to fill and empty. Symptoms vary depending on the length of the VT and the rate.

Intravenous lidocaine administration was standard therapy for VT for many years, but more efficacious antidyrrhythmics are
now available. Lidocaine is still the drug of choice for stable VT in many institutions, although some centers use amiodarone as a first-line agent. If pharmacologic measures are unsuccessful, cardioversion is attempted. With pulseless VT, defibrillation is the standard of care. Intravenous amiodarone and vasopressin (Pitressin) have been added to advanced cardiac life support protocols to treat VT refractory to defibrillation. Additional agents include procainamide and bretylium (Bretylol). Patients with persistent or recurrent VT should also be assessed for electrolyte abnormalities, including hypokalemia, hyperkalemia, and hypomagnesemia. Long-term VT suppression is obtained with oral antiarrhythmic medications such as amiodarone or special procedures such as radiofrequency ablation.

**Torsades de Pointes.** Torsades de pointes, a variation of VT, can also progress to ventricular fibrillation (VF) if not managed appropriately. A long Q-T interval (over half of the corresponding R-R interval) commonly precedes torsades de pointes. P waves, when seen, are dissociated from the QRS complexes. The QRS complexes are longer than 0.12 second and bizarre. The QRS complexes “twist” along the isoelectric baseline, varying in size and direction (Figure 29-37).

The initiating electrophysiologic mechanism may be triggered activity or reentry. The rhythm may result from prolonged repolarization, represented on the ECG as a prolonged Q-T interval. Prolongation may occur secondary to various medications or electrolyte abnormalities (hypokalemia or hypomagnesemia), or it may be congenital. Alterations in ion movement secondary to genetic mutations have been shown to be responsible for slightly more than 50% of researched cases of long QT syndrome. Six genetic variants currently are recognized. Cardiac output decreases from inadequate ventricular filling and emptying that result from the increased heart rate.

Magnesium sulfate is administered to stabilize the electrical membrane. Potassium may also be indicated. When torsades is associated with congenital long QT syndrome, beta-blockers have been efficacious. Isoproterenol (Isuprel) has also been used. Overdrive pacing may be of benefit in selected cases. With recurrent torsades, implantable defibrillators are used as prophylaxis. The patient who is unstable is cardioverted or defibrillated as with pulseless VT. Once the initial crisis is resolved, the cause is determined and corrected when possible. Treatment modalities soon may be based on the genotype of the individual.

**Ventricular Fibrillation and Asystole.** In VF the ventricular activity of the heart is chaotic, and the ECG tracing consists of unidentifiable waves. The fibrillatory waves may be coarse or fine (Figure 29-38). In the absence of depolarization there can be no effective ventricular contraction. The most common cause is CAD with areas of lowered fibrillatory thresholds. It frequently involves conduction disturbances and reentry. It can also occur without warning after reperfusion. Nonischemic causes may include antiarrhythmic medications, long QT syndromes, preexcitation syndromes, and systemic hypoxemia.

Defibrillation is the only treatment for VF, and it must be performed as soon as possible. Automated external defibrillators (AEDs) eliminate the need for rhythm recognition and can be manipulated quickly to allow for rapid defibrillation. While awaiting an AED, bystander cardiopulmonary resuscitation (CPR) may prolong the period in which VF may respond to defibrillation. The shock allows the heart to simultaneously depolarize, stopping all reentry and allowing an organized electrical rhythm to return. The administration of epinephrine or vasopressin may increase the effectiveness of defibrillation. Other drugs that may be used for refractory VF include lidocaine, amiodarone, bretylium, and magnesium. For those who survive VF, the long-term use of beta-
blockers may decrease the recurrence rate. An implantable defibrillator is the treatment of choice for survivors.

In asystole the ECG tracing is a flat line and no electrical activity is noted; all pacemaker cells have failed. The patient has no blood pressure, pulse, or audible heartbeat; respirations quickly cease. CPR must be instituted immediately. Epinephrine, atropine, and external pacing are all used in the effort to restore cardiac excitability.

Pulseless electrical activity is the term used to describe the presence of electrical activity in the absence of a heartbeat. CPR is instituted immediately along with measures to restore contraction. These may include pericardiocentesis, if tamponade is inhibiting contraction, or the administration of calcium to stimulate contractile force. Medications may include epinephrine and atropine.

Atrioventricular Block. A block to impulse conduction can occur at any point along the conduction pathways. One common area is the AV junction. The severity of the atrioventricular block is identified by degrees, that is, first-, second-, or third-degree AV block. First-degree AV block is present when the P-R interval is longer than 0.20 second, indicating a conduction delay in the AV node (Figure 29-39). It usually is found in association with rheumatic fever, digoxin, beta-blockers, acute inferior MI, and increased vagal tone. When a first-degree AV block occurs in isolation, the patient is usually asymptomatic and no treatment is necessary.

Second-degree AV block may be subdivided into two categories. Type I (Wenckebach, or Mobitz type I) is characterized by a P-R interval that progressively lengthens until a P wave is not followed by a QRS complex (Figure 29-40). The nonconducted impulse arrives at the AV node during the refractory period. The ratio of P waves to QRS complexes may be 5:4, 4:3, 3:2, or 2:1 and creates a clustered appearance. The pathologic condition is usually within the AV node and produces QRS complexes of less than 0.12 second. Any drug that slows AV conduction may cause a type I block, but such blocks are most often seen in the patient with an acute inferior wall MI, digitalis toxicity, increased vagal tone, electrolyte imbalance, or acute myocarditis or after cardiac surgery. Type I blocks often are transient and reversible, and treatment is not required unless the patient becomes symptomatic. Atropine may be effective in increasing cardiac output.

Type II (Mobitz type II) second-degree AV block is less common but more serious. A type II block is characterized by nonconducted sinus impulses despite constant P-R intervals for the conducted P waves. The nonconducted P waves may occur at random or in patterned ratios (e.g., 2:1, 3:1) (Figure 29-41). The QRS complexes are widened unless the block is within the bundle of His. Type II blocks may occur in patients with CAD, MI, rheumatic heart disease, cardiomyopathy, and chronic fibrotic disease of the conduction system. If cardiac output is decreased, a temporary pacemaker usually is inserted prophylactically until the conduction stabilizes. If the block is persistent, the patient benefits from a permanent pacemaker. Atropine may be used to reduce vagal tone and improve conduction through the AV node. However, this is effective only if the site of block is the AV node. If the block is below the AV node, atropine is not effective.

In third-degree AV block (complete heart block) all the sinus or atrial impulses are blocked, and the atria and ventricles beat independently. Either a junctional or a ventricular pacemaker cell...
drives the ventricles. The lesion is usually in the bundle of His or the bundle branches but may also be at the AV junction. The rate and dependability of the ventricular rhythm are related to the level of the lesion. If a junctional pacemaker drives the ventricles, the ventricular rate will be at least 40 to 60 beats/min and the QRS complexes are narrow. This block may be a transient complication of inferior posterior MI or digitalis toxicity, or it may result from severe heart disease.

If a ventricular pacemaker drives the ventricles, the rate will be 20 to 40 beats/min, and the patient may experience syncope, heart failure, altered mentation, or angina. The QRS complex is abnormally wide, indicating that the block lies below the AV junction (Figure 29-42). The prognosis is more serious if complete heart block accompanies anterior MI. Generally the patient requires a permanent pacemaker. Epinephrine or isoproterenol administered intravenously may increase the ventricular rate temporarily until artificial pacing can be instituted.

**BUNDLE BRANCH BLOCK.** In bundle branch block (BBB) one or both bundle branch paths of the conduction system are blocked. The impulse must travel a different path to stimulate the ventricles; therefore the QRS is prolonged to greater than 0.12 second. Instead of a synchronous QRS complex, each ventricle independently depolarizes, creating characteristic jagged QRS complexes (Figure 29-43). A BBB occurs as a permanent defect or as a transient block secondary to tachycardia, heart failure, AML, pulmonary embolus, hypoxia, or metabolic derangements.

The right bundle branch is the more delicate of the two bundles and has a longer refractory period in some persons. In the younger patient right BBB often results from right ventricular hypertrophy, whereas CAD usually is the cause in the older patient. One classic ECG pattern is an M-shaped QRS in V1 and V2. In the absence of other conduction defects, no intervention is necessary. The left bundle branch has a main trunk that bifurcates into left anterior and left posterior divisions. A block may occur in the main trunk or in either of the divisions. A block in the main trunk produces a complete left BBB, resulting in a QRS greater than 0.12 second; large R waves in V5 and V6; and deep, wide S waves in V1 through V3. Left BBB is associated with severe CAD, valvular disease, hypertensive disease, cardiomegaly, and acute anterior wall MI. It also may occur as a result of degenerative changes in the conduction system. Whenever sufficient blockage is present to leave the heart dependent on just one fascicle for conduction to the ventricles, the patient is a candidate for a permanent pacemaker.

**TREATMENT OPTIONS FOR DYSRHYTHMIAS.** Collaborative care for the patient with a dysrhythmia includes diagnosing the specific dysrhythmia and its associated cause and treating the disorder with medications or interventional procedures. Table 29-9 presents medications commonly used to manage dysrhythmias. The nurse must be knowledgeable about the mechanism of action of specific drugs and their associated nursing interventions. Careful attention is paid to potential drug interactions and synergistic effects when combination therapy is used. The metabolism and excretion of medications may be impaired in older adults and in patients with decreased perfusion to the kidneys and liver. The
Cardioversion differs from defibrillation in that the electrical discharge is synchronized with the R wave to avoid triggering VF from accidental discharge during the vulnerable period of repolarization. Indications for cardioversion include hemodynamically unstable atrial flutters, AF, and atrial tachycardia. Body size and patient stability are used to guide the amount of energy selected, usually 100 to 360 J. Biphasic defibrillators require less energy and have proven more effective in converting AF to NSR. Appropriate levels of anticoagulation should be established for patients with atrial flutter or fibrillation rhythms before treatment with cardioversion. Patches may be placed in either the right anterior and left posterior or right anterior and left lateral positions. Occasionally the simultaneous use of two sets of patches and two defibrillators is needed for large patients or resistant rhythms. Patients should be NPO before the procedure. The nurse prepares patients psychologically for what to expect during cardioversion and reassures them that they will be sedated with intravenous diazepam (Valium), midazolam (Versed), or fentanyl. An anesthesiologist is nearby. Elective cardioversion should be performed in a special laboratory and not the patient’s room. The defibrillator is synchronized so that the impulse is not initiated until the next R wave occurs. This eliminates the danger of entering the vulnerable period. For most elective procedures, the amount of watt-seconds or joules required for conversion is lower than that required for defibrillation. The nurse monitors the patient after cardioversion until vital signs are stable. Although the procedure itself is often successful, the rate of recurrence is high.

Internal atrial cardioversion may be an alternative when external cardioversion fails. Two small electrode catheters are placed in the right atrium and coronary sinus to accomplish the cardioversion. A bipolar catheter is placed in the right ventricle to precisely time the cardioversion. Energy levels are typically 1 to 100 J. The patient is under conscious sedation during the procedure.

Radiofrequency Catheter Ablation. Radiofrequency catheter ablation (RFCA) involves the insertion of a catheter, usually through the patient’s femoral or jugular vein, which delivers programmed electrical simulation to recreate the patient’s dysrhythmia and localize the area for ablation. The site of origin for the dysrhythmia or the pathway necessary for its propagation is then destroyed using radiofrequency energy, a form of high-frequency electromagnetic waves. The thermal energy causes coagulation necrosis in the area selected for ablation. Cryoablation, the use of freezing temperatures, may also be used to destroy the site of origin. The amount of damage caused by the catheter is relatively small because the energy used can be precisely regulated and focused. The patient remains in the electrophysiology laboratory for a short interval after the procedure for observation.
Attempts are then made to reinitiate the dysrhythmia, using electrical or pharmacologic stimulation. If the dysrhythmia recurs, additional ablation bursts are administered until the site is destroyed.

Indications for catheter ablation include AV node reentry tachycardias, accessory pathways (such as Wolff-Parkinson-White syndrome), focal atrial tachycardia, atrial flutter, and bundle branch reentry. Ectopic areas in and near the pulmonary vein are also target sites for catheter ablation in AF. Ablation is also an alternative in select cases of VT. Complications related to RFCA are rare but may include problems at the access site, catheter-induced thrombi, and myocardial perforation. The most common complication of AV node–associated dysrhythmias is heart block.

Patient teaching is a major focus of nursing intervention for RFCA because preprocedure anxiety is often high (see Patient/Family Teaching box). Electrophysiology procedures, both diagnostic and therapeutic, are increasing in number, and scope and nursing research is evolving in this area, with a focus on developing best practice approaches (see Research box).

Pacemakers. Pacemakers are typically inserted when patients experience symptomatic chronic or recurrent dysrhythmias that are unresponsive to pharmacologic therapy. Pacemakers may be placed internally for permanent pacing or used externally to address a temporary need. Permanent pacemakers use a pulse generator, powered by a sealed lithium battery, as the "control center" for the pacemaker's functions (Figure 29-44). The generator attaches to one or two leads that are positioned in the right ventricle or right atrium (Figure 29-45). These leads

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine</td>
<td>Inhibit sodium influx during phase 0 depolarization</td>
<td>Monitor Q-Tc; P-R, QRS; may cause tinnitus.</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Inhibit sodium influx during phase 0 depolarization</td>
<td>Monitor Q-Tc, P-R, QRS; may cause tinnitus.</td>
</tr>
<tr>
<td>Disopyramide*</td>
<td>Inhibit sodium influx during phase 0 depolarization</td>
<td>Monitor Q-Tc, P-R, QRS; may cause tinnitus.</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Modestly inhibit sodium influx during phase 0 depolarization</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Mexiletine</td>
<td>Decrease calcium influx during phase 0 depolarization</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Tocainide</td>
<td>Depression of the AV node and prolongation of the action potential duration</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Morinex®</td>
<td>Depression of the AV node and prolongation of the action potential duration</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Flecainide</td>
<td>Decrease calcium influx during phase 0 depolarization</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Propafenone†</td>
<td>Decrease calcium influx during phase 0 depolarization</td>
<td>May cause anticholinergic effects.</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Acebutolol</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Sotalol</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Butilide</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Dofetilide</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Dilatazem</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Block outward potassium channels or facilitate slow inward sodium current; lengthen repolarization</td>
<td>May cause heart failure in susceptible patients.</td>
</tr>
</tbody>
</table>

Continued
are flexible, insulated wires with electrodes for sensing the heart’s rhythm and delivering electrical impulses when necessary. The leads are introduced into the myocardium transvenously under fluoroscopic visualization through the subclavian or jugular vein with the aid of a guidewire to facilitate correct placement of the leads against the atrial or ventricular endocardium. A subcutaneous pocket is created surgically to enclose the generator, most often infraclavicularly.

Pacemakers in use today have multiple capabilities that can be identified through a five-letter pacemaker code (Table 29-10). The last two letters of the code describe the pacemaker’s specific features such as antitachycardic pacing and rate-responsive pacing. When an antitachycardic pacemaker senses a heart rate above its programmed limit, it paces at a heart rate just above the patient’s tachycardia to take control of the heart. The pacemaker then slows the rhythm to an acceptable rate. Rate-responsive pacemakers allow pacing at accelerated heart rates when the pacemaker senses programmed indicators of increased activity such as changes in oxygen saturation, cardiac output, or blood temperature.

Pacemaker insertion can be performed with the patient under local anesthesia. Before insertion of a permanent pacemaker, the nurse thoroughly educates the patient about the indications for the pacemaker, the procedure itself, pacemaker care, and potential complications (see the Patient/Family Teaching box). Complications of pacemaker therapy include pacemaker malfunction, cardiac perforation and tamponade, pneumothorax and hemothorax, and infection. Nursing responsibilities before and after permanent pacemaker implantation are summarized in the Guidelines for Safe Practice box.

Figure 29-46 shows the ECG appearance of pacemaker-stimulated heartbeats. Paced beats are readily identifiable by the sharp spike that precedes the ECG complex. The paced QRS

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**Table 29-9**

**Common Medications for Dysrhythmias—cont’d**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Nursing Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verapamil</td>
<td>Direct depressant effect on contractility; use with caution in LV dysfunction.</td>
<td>Administer rapid IV push followed by 20 ml flush. Half-life: 10 sec.</td>
</tr>
<tr>
<td>Adenosine</td>
<td>Depresses SA node and slows conduction through AV node; can interrupt reentry pathways through AV node. Indications: SVT</td>
<td>Give slow IV push: extravasation will result in necrosis.</td>
</tr>
<tr>
<td>Atropine</td>
<td>Increases heart rate by antagonizing acetylcholine receptors, blocking vagal stimulation, increasing automaticity of SA node and conduction in AV node. Indications: symptomatic bradycardia</td>
<td>Give slow IV push: extravasation will result in necrosis.</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>Cation needed for cardiac contractility</td>
<td>Give slow IV push: extravasation will result in necrosis.</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Direct suppression of AV node; Indications: asymptomatic bradycardia</td>
<td>Give slow IV push: extravasation will result in necrosis.</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Beta, and beta, agonist increasing automaticity; Indications: asymptomatic, refractory VT/VF</td>
<td>Increases oxygen demand.</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Causes increased contractility and heart rate by acting on beta1 and beta2 receptors in heart; Indications: symptomatic bradycardia</td>
<td>Increases oxygen demand.</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Reduces SA node impulse formation, prolongs conduction time in myocardium; Indications: documented hypomagnesemia with dysrythmias, torsades de pointes</td>
<td>Monitor magnesium levels.</td>
</tr>
</tbody>
</table>

*Use of quinidine, procainamide, and disopyramide is decreasing due to newer, safer, and more efficacious drugs.†Use of these drugs, including lidocaine (historically a first-line choice for VT), is gradually decreasing in favor of safer, more efficacious antidysrhythmics.‡These drugs are not recommended for use in patients with coronary artery disease because of increased incidence of mortality and nonfatal cardiac arrest in patients after myocardial infarction.§Among the most widely used antidysrhythmics. Iden
tifying the most widely used antidysrhythmics.**
complex is wide because initiation of the impulse occurs in the ventricle (as with a PVB).

If a pacemaker should malfunction, the patient usually experiences a recurrence of symptoms. However, the nurse must also be able to diagnose the following ECG indicators of pacemaker malfunction: loss of sensing, loss of capture, and failure to pace. Table 29-11 describes common pacemaker problems and interventions to troubleshoot them. A Nursing Care Plan for a patient undergoing pacemaker insertion is on p. XXX.

Temporary pacemakers are indicated for the short-term management of dysrhythmias until the patient’s rhythm stabilizes or a permanent pacemaker can be inserted. The pacemaker wire is advanced transvenously to the right ventricle, and the leads are attached to an external pulse generator box (Figure 29-47). Transvenous pacemakers can include devices that combine pulmonary artery catheters with the pacemaker. The environment must be kept free from electrical hazards that could trigger dysrhythmias.

Research


Nurse researchers challenged the common practice of keeping patients in bed for 4 hours after electrophysiology procedures that used a femoral venous approach. Sixty-eight patients were randomized to 2 hours (n = 31) or 4 hours (n = 37) of bed rest. Patients were similar with regard to age, gender, number of sheaths used, and procedural heparin use. Medications given during the electrophysiology procedure included midazolam and fentanyl for conscious sedation. Postprocedural femoral access site care included a gauze dressing over the catheter insertion site, extension of the affected leg, and elevation of the head of bed 30 to 45 degrees. Sandbags and pressure dressings were not used. Medications for pain management included acetaminophen, 650 mg; or one to two tablets of oxycodone, 5 mg, plus acetaminophen, 325 mg.

The groups had no significant differences with regard to bleeding incidence, hematomas, use of analgesics, or patient satisfaction. This study supported the nurses’ belief that bed rest after electrophysiological studies via a femoral vein approach could safely be reduced to 2 hours. Nurses should continue to challenge existing protocols through research. Time-in-bed studies should increasingly explore patient safety as new devices for closure of arterial and venous punctures are employed.
used, atrial wires exit to the right of the sternum and ventricular wires exit to the left. Care of the patient with a temporary pacemaker is summarized in the Guidelines for Safe Practice box.

External cardiac pacemakers are primarily used for patients with unstable rhythms in emergency situations and require the application of two electrodes to the chest wall, one over the cardiac apex and the other on the back beneath the left scapula (Figure 29-48). An electrical current flows between the electrodes and is controlled by the device operator. Most external pacing devices function in the demand mode. An oscilloscope allows monitoring of the pacemaker activity. External pacing is uncomfortable for the patient, and the nurse plans for pain management and offers encouragement and support.

Transcatheter pacing uses a transcatheter needle to place electrodes into the ventricle, which are then attached to, and controlled by, an external generator. This procedure is used only in emergency situations where other measures have failed. Transesophageal pacing utilizes a pacing electrode inserted into the lower esophagus via a nasal catheter electrode or gelatin pill electrode. Transesophageal pacing is beneficial for overdrive atrial pacing, that is, pacing the atria at a faster rate to take control and slow the rate. It has been less successful as a route for ventricular pacing.

**Implantable Cardioverter Defibrillators.** More than half the deaths from CAD in the United States each year are sudden deaths occurring within 24 hours of the onset of symptoms, commonly before the patient reaches the hospital. The pathophysiology of sudden cardiac death remains obscure, since only 20% of sudden deaths are directly associated with MI. Researchers theorize that the cause of sudden cardiac death is not occlusive thrombosis or myocardial damage but a derangement in the heart’s electrical stability, most often deteriorating into VF. The incidence of sudden death is greater in patients with cardiomyopathy, prolonged Q-T intervals, myocarditis, proaryrhythmic medications, and electrolyte imbalance.

The implantable cardioverter-defibrillator (ICD) is indicated for the treatment of clinically significant and hemodynamically important dysrhythmias that do not respond to antidysrhythmic therapy. The ICD consists of a pulse generator and two or three lead systems that continuously monitor heart activity and automatically deliver a

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**Table 29-10 Intersociety Commission for Heart Disease Codes for Pacemakers**

<table>
<thead>
<tr>
<th>Chamber(s)</th>
<th>Chamber(s)</th>
<th>Modes of Responses</th>
<th>Programmable Functions</th>
<th>Special Tachyaryrhythmia Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>V: ventricle</td>
<td>V: ventricle</td>
<td>T: triggered</td>
<td>P: programmable</td>
<td>B: bursts</td>
</tr>
<tr>
<td>D: double (dual)</td>
<td>D: double (dual)</td>
<td>D: double (dual function: T and I)</td>
<td>O: none (permanent pacemakers only)</td>
<td>S: scanning</td>
</tr>
<tr>
<td>O: none</td>
<td>O: none (continuous)</td>
<td>R: reverse</td>
<td></td>
<td>E: external</td>
</tr>
</tbody>
</table>

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**Coronary Artery Disease and Dysrhythmias Chapter 29 793**
countershock to correct a perceived dysrhythmia. The pacemaker-cardioverter-defibrillator models can pace patients out of tachycardic rhythms and can pace bradycardic rhythms. The devices can override the heart’s pacemaker to gain control or cardiovert the heart at different energy outputs if overpacing is ineffective.

Records of the dysrhythmic event can be retrieved to evaluate the sequence of events and the appropriateness of ICD therapy.

The device is implanted in a similar manner to a permanent pacemaker. The nurse teaches the patient about situations that may cause malfunction of the ICD, such as MRI or diathermy. Special

**Guidelines for Safe Practice**  
**The Patient Undergoing Permanent Pacemaker Insertion**

**Preprocedure**
- Establish assessment baselines: vital signs, 12-lead electrocardiogram (ECG), peripheral pulses, heart and lung sounds, mental status.
- Teach patient per patient/family education guidelines.
- Maintain nothing-by-mouth status for 8 hours.
- Establish intravenous (IV) access for administration of fluids, sedation, and emergency drugs.
- Assess anxiety level and intervene appropriately with active listening, reassurance, education, and sedation as needed.

**Intraprocedure**
- Shave and scrub access site.
- Maintain sterile field.
- Cardiac monitor at all times.
- Assess patient’s anxiety level and intervene appropriately with reassurance and sedation as needed.

**Postprocedure**
- Monitor for complications of insertion such as pneumothorax, hemothorax, perforation, tamponade.
- Be alert to lead dislodgement, manifested by ECG changes or hiccups if diaphragm is being paced.
- Control pain; provide analgesics and nonpharmacologic interventions (positioning, distraction) as needed.
- Obtain baseline ECG and monitor for loss of sensing, loss of capture, or failure to pace.
- Assess insertion site for bleeding and infection.
- Ensure bed rest for 12 hours.
- Restrict range of motion of affected arm for 12 to 24 hours.
- Apply ice pack to minimize pain and swelling for first 6 hours.
- Do not administer aspirin or heparin for 48 hours.
- If defibrillation is necessary, anteroposterior placement is preferable; avoid area surrounding generator site.
- If patient is symptomatic from pacer malfunction, enforce bed rest, follow safety precautions for syncope potential, monitor vital signs frequently, and obtain a 12-lead ECG to diagnose malfunction. —Monitor by continuous telemetry, obtain IV access with atropine at bedside, provide oxygen if needed, perform chest x-ray study to check lead position, and use pacemaker magnet to convert pacemaker to fixed mode if indicated.
- Provide discharge teaching per patient/family teaching guidelines.
precautions are also necessary during procedures such as lithotripsy and radiotherapy. Electrical interference may occur with stereo systems, high-powered motors, and arc welders. Emotional support is critical because patients and family members commonly respond to the ICD with anxiety, depression, fear, and anger. The strongest predictor of a poor quality-of-life outcome may be linked with ICD shocks. Phantom shocks, also reported in the literature, can contribute to anxiety and depression after the procedure. Teaching guidelines are included in the Patient/Family Teaching box.

**Patient/Family Teaching.** Patient and family teaching is an integral part of the nursing care for patients experiencing dysrhythmias. Lifestyle modifications may include the elimination of caffeine, alcohol, or other substances believed to contribute to the disorder. Stress reduction measures are often encouraged. The nurse directly addresses the patient’s and family’s fears and concerns, recognizing the challenges of living with a potentially life-threatening dysrhythmia. The nurse teaches the patient about the specific dysrhythmia, the treatment plan, and the importance of seeking medical attention promptly if symptoms recur. Patients should also know how to take the pulse and the types of pulse changes that need to be reported. The nurse reviews the common side effects of the antidysrhythmic agents with the patient and encourages the patient to discuss the incidence and severity of side effects with a health care professional. Patients are cautioned not to adjust the dose or discontinue the use of any prescribed medication. Follow-up is essential in monitoring medication therapy and response.

<table>
<thead>
<tr>
<th>Problem</th>
<th>Definition</th>
<th>ECG Finding</th>
<th>Physiologic Effect</th>
<th>Nursing Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of sensing— overseasing</td>
<td>Pacemaker senses extraneous signal as impulse and therefore does not pace.</td>
<td>Pause</td>
<td>Decreased cardiac output</td>
<td>Decrease sensitivity of pacemaker. Check for electromagnetic interference and proper grounding of equipment (temporary pacemaker).</td>
</tr>
<tr>
<td>Loss of sensing— undersensing</td>
<td>Pacemaker does not sense heart’s own impulse and therefore thinks it has to pace the heart.</td>
<td>Inappropriate pacing (extra beats)</td>
<td>Danger of pacing in vulnerable period, causing ventricular tachycardia</td>
<td>Increase sensitivity to heart’s rhythm.</td>
</tr>
<tr>
<td>Loss of capture</td>
<td>Pacemaker fires but does not depolarize ventricle.</td>
<td>Spike present but without QRS complex</td>
<td>Decrease in cardiac output</td>
<td>Increase milliamperes (energy delivered), turn to left side (bring lead in better contact with endocardium). Check all connections (temporary pacemaker). Determine cause of ventricle not responding and correct electrolyte abnormality, ischemia, lead dislodgement.</td>
</tr>
<tr>
<td>Failure to pace</td>
<td>Electrical impulse never initiated.</td>
<td>Pause without spikes</td>
<td>Decrease in cardiac output</td>
<td>Keep external or temporary pacemaker at bedside. Assess response and treat symptoms until cause determined and corrected (dislodged lead, battery depletion, malfunctioning pulse generator).</td>
</tr>
</tbody>
</table>

**TABLE 29-11 Troubleshooting Pacemaker Malfunction**

**Coronary Artery Disease and Dysrhythmias**

**CHAPTER 29**
In emergent treatment for a patient in ventricular fibrillation, the nurse's first action is to:
1. Administer intravenous magnesium sulfate
2. Set-up for placement of transcutaneous pacemaker
3. Prepare the patient for cardioversion
4. Defibrillate the client

Cardiopulmonary Resuscitation

The AHA estimates that 400,000 to 460,000 individuals die of heart disease each year in the emergency room or before they even reach the hospital, often from VF. Sudden death from ischemic heart disease is one of the most serious medical emergencies, and it seems reasonable to assume that many of these deaths might be prevented by prompt and appropriate intervention. Lay rescuers are increasingly being trained to use AEDs, since the time from collapse to defibrillation is the single greatest determinant of survival. Access to AEDs is increasing at sites with concentrated populations (e.g., sporting events, airlines, and shopping malls). AEDs are used only when the patient is unresponsive, has no effective breathing, and has no signs of circulation (cardiopulmonary arrest).

Cardiopulmonary arrest is characterized by the cessation of breathing and circulation and signifies a state of clinical death. It is characterized by unresponsiveness, cessation of respiration, pallor and cyanosis, absence of heart sounds and blood pressure, loss of palpable pulse, and dilation of the pupils. Immediate and definitive action must be instituted within 4 to 6 minutes after the arrest, or biologic death occurs.

Nursing Care Plan

Patient Receiving Permanent Pacemaker

Data: A 65-year-old woman is admitted to the cardiac care unit for unstable angina and suspected acute myocardial infarction. Her presenting symptoms are unusual fatigue and indigestion. Her medical history includes type 2 diabetes mellitus of 20 years’ duration and hypertension, which has been controlled with medication for the past 7 years. Before a diagnostic cardiac catheterization can be performed, the patient develops symptomatic bradycardia ranging between 40 and 50 beats/min. Electrocardiogram reveals a Mobitz type II second-degree atrioventricular block. Atropine is administered and temporary pacing initiated. Further diagnostic tests indicate that the patient has suffered an acute myocardial infarction. Because of the patient’s increased risk for heart failure as a result of bradycardia and the ischemic blood supply to her conduction system, the decision is made to proceed with permanent pacemaker placement. A DDD pacemaker is placed 3 days after her admission to the hospital. Her postoperative course is uncomplicated, and plans are made for her discharge to home. The patient’s daughter, who will be caring for her at home, voices concern about what to do should the pacemaker fail.

Nursing Diagnosis

Risk for infection related to surgical implantation of foreign device

Outcomes
- Patient will remain free from signs of wound and systemic infection.
- Patient will accurately demonstrate sterile technique in caring for surgical incision.

Related NOC Outcomes
- Immune Status
- Knowledge: Infection Control
- Risk Control

Related NIC Interventions
- Infection Control
- Infection Protection
- Surveillance
- Wound Care

Nursing Interventions/Rationales
- Assess insertion site for signs of localized wound infection (redness, swelling, tenderness, warmth). To recognize the presence of wound infection so treatment can be initiated and systemic infection prevented.
- Assess for signs of systemic infection (fever, fatigue, elevated white blood cell count). To recognize the presence of systemic infection so prompt treatment can be initiated.
- Teach patient how to maintain sterile technique when changing dressings or cleaning incisional site. Microorganisms can readily penetrate through nonintact skin such as a surgical incision. Sterile technique reduces the risk of contamination and subsequent wound infection.
- Teach patient the signs and symptoms of localized and systemic infection. So the client will recognize impending infection and seek assistance quickly.
- Encourage patient to avoid handling of or unnecessary contact with surgical site. To decrease the risk of wound contamination and subsequent wound infection.
- Encourage a high-protein, high-calorie diet. Proteins and calories are necessary for wound healing.

Nursing Diagnosis

Impaired physical mobility related to incisional site pain, activity restrictions, and fear of lead displacement

Outcomes
- Patient will verbalize prescribed restrictions.
- Patient will describe resources to assist with activities of daily living (ADLs) until physical mobility improves.

Related NOC Outcomes
- Adherence Behavior
- Knowledge: Prescribed Activity
- Mobility

Related NIC Interventions
- Pain Management
- Positioning
- Self-Care Assistance
- Teaching: Prescribed Activity/Exercise

ARE YOU READY?

In emergent treatment for a patient in ventricular fibrillation, the nurse's first action is to:
1. Administer intravenous magnesium sulfate
2. Set-up for placement of transcutaneous pacemaker
3. Prepare the patient for cardioversion
4. Defibrillate the client
immediately, and the victim is cautiously placed in the supine position on a firm surface, remembering the potential for head injury.

Step 2: Open the Airway

The tongue is the most common cause of airway obstruction in the unconscious person. The head tilt–chin lift and the jaw thrust are the two recommended methods for opening and maintaining the airway (Figures 29-49 and 29-50). Jaw thrust (without head tilt) is the safest approach to use for a victim with a suspected neck injury. The rescuer must carefully support the head to avoid turning or tilting it backward. While maintaining an open airway, the rescuer takes 3 to 5 seconds to look, listen, and feel for spontaneous breathing. The rescuer places an ear over the victim’s nose and mouth while looking at the victim’s chest to see if it moves.

Persons who appear to be unconscious may be asleep, deaf, or intoxicated. Unconsciousness is confirmed by shaking the victim’s shoulders and shouting, “Are you OK?” If the person does not respond, the emergency response system (911) is activated immediately, and the victim is cautiously placed in the supine position on a firm surface, remembering the potential for head injury.

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with respiration, listens for air escaping during exhalation, and feels for air movement against the face.

**Step 3: Initiate Artificial Ventilation**

**Mouth-to-Mouth Ventilation.** To initiate artificial ventilation, the rescuer gives two breaths lasting 2 seconds each, and observes for adequate ventilation. If the patient does not resume breathing, the rescuer continues mouth-to-mouth ventilation, delivering one breath every 5 seconds.

1. Maintain victim in head tilt–chin lift position.
2. Pinch nostrils.
3. Take a deep breath and place mouth around outside of victim’s mouth, forming a tight seal. Use a rescue airway if available.
4. Blow into victim’s mouth.
5. Adequate ventilation is demonstrated by:
   a. Rise and fall of chest
   b. Hearing and feeling air escape as victim passively exhales
   c. Feeling the resistance of the victim’s lungs expanding

**Mouth-to-Nose Ventilation.** Mouth-to-nose ventilation is indicated when the mouth is seriously injured or a tight seal cannot be established around the mouth. The rescuer places one hand on the forehead to tilt the head back and uses the other hand to lift the lower jaw and close the mouth. After taking a deep breath, the rescuer seals the mouth around the victim’s nose and begins blowing until the lungs expand. Occasionally, when mouth-to-nose ventilation is used, it may become necessary to open the victim’s mouth or lips to allow air to escape on exhalation because the soft palate may produce nasopharyngeal obstruction.

**Mouth-to-Stoma Ventilation.** Direct mouth-to-stoma artificial ventilation is performed for the laryngectomy patient. For the patient with a temporary tracheostomy tube, mouth-to-tube ventilation should be initiated after the cuff is inflated.

**Mouth-to-Barrier Ventilation.** An alternative to direct mouth-to-mouth ventilation is use of a barrier device such as a face shield and mask device. Most mask devices have a one-way valve so that exhaled air does not enter the rescuer’s mouth; many face shields

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**GUIDELINES FOR SAFE PRACTICE The Patient With a Temporary Pacemaker**

**Assess Patient’s Tolerance of Heart Rhythm**

Perform patient assessment: mental status, blood pressure and rhythm, urinary output, skin color and warmth, pulses, heart sounds, and lung sounds.
Perform continuous electrocardiographic (ECG) monitoring.

**Check System for Proper Functioning**

Check pacing threshold (the minimum amount of milliamperes needed to pace the heart) every 12 hours; set milliamperes level two to three times the threshold as a safety margin; adjust as needed and notify physician.
Replace battery in generator or connecting cable for failure to pace as necessary.
Adjust sensitivity for undersensing or oversensing; notify physician.
Secure all connections; secure generator box to patient (preferably) or bed.

**Maintain Electrical Safety**

Wires must be connected and secured to the correct connector ports (atrial/ventricular, positive/negative).

Maintain insulation cover over uninsulated ends.
Wear rubber gloves when handling exposed terminals.
Do not touch the patient and electrical equipment at the same time.
Prevent liquids from coming in contact with the generator, cables, or insertion site.
Keep ungrounded electrical equipment from contact with the patient.

**Monitor for Complications at Insertion Site**

Inspect site daily for infection.
Change dressing every 48 hours using central line dressing sterile technique.

**Assess Patient Safety and Comfort**

Explain the purpose of the pacemaker to decrease anxiety.
Position patient comfortably, avoiding accidental tension on external wires and generator.
When mobility is limited, help the patient find diversional activities.
Coronary Artery Disease and Dysrhythmias

**CHAPTER 29**

**Procedural**
- Teach patient about:
  - Indication for implantable cardioverter-defibrillator (ICD)
  - Potential complications
  - Nothing by mouth for 8 hours before the procedure
  - Pretests, including baseline 12-lead electrocardiogram and bleeding function studies
  - Cardiac monitor at all times during the procedure
  - Intravenous access for fluids, cardiac medications, and sedation
  - Prep and shave of area where generator will be implanted
  - Anesthesia of access sites
  - Sterile field during procedure
  - Analgesics available after procedure
  - Restricted arm movement for 24 hours if implant site is infraclavicular
  - Routine chest x-ray to check placement

**Discharge Instructions**
- **Insertion Site**
  - Teach patient to:
    - Monitor site for infection and bleeding the first week.
    - Avoid immersion of site in water for 3 days.
    - Remove Steri-Strips in about 1 week.
    - Wear loose covering over incision for 1 week.

- **Activity**
  - Teach patient to:
    - Avoid contact sports and ham radios.

- Increase activity gradually after implantation of device (should be at full preimplant activity level once incision has healed).
- Follow driving restrictions and discuss concerns with cardiologist.
- Seek guidance from cardiologist about: flying, excessive heights, industrial facilities, welding.
- Avoid swimming or boating alone.

**Health Care Follow-up**
- Teach patient to:
  - Adhere to schedule for important follow-up care.
  - Sit or lie down if signs or symptoms of decreasing cardiac output with dysrhythmia occur.
  - Notify health care professional for:
    - Signs or symptoms of dysrhythmia similar to those before ICD
    - Rapid, irregular heart rate
    - Chest pain or shortness of breath

**Safety**
- Teach patient to:
  - NOTE: Patient and others in physical contact with patient will experience a mild sensation with shock delivery.
  - Carry ICD information at all times—will alarm some airport security.
  - Consult with cardiologist before undergoing diagnostic or surgical interventions.
  - Move away from devices if dizziness experienced.
  - Avoid working over large, running motors.
  - Learn how to take radial pulses, notify health care professional for rates outside those programmed.

**Figure 29-48** Transcutaneous external pacing.

**PATIENT/FAMILY TEACHING** The Patient With an Implantable Cardioverter-Defibrillator
Table 29-12 Sequence of Cardiopulmonary Resuscitation

<table>
<thead>
<tr>
<th>Findings</th>
<th>Action</th>
<th>ABCs of Action</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>No response</td>
<td>Activate emergency medical services</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence of respiration; cyanosis, dilated pupils</td>
<td>Open airway</td>
<td>A—Open airway</td>
<td>3-5 sec to assess for respiration</td>
</tr>
<tr>
<td>Respiration still absent</td>
<td>Initiate artificial ventilation</td>
<td>B—Restore breathing</td>
<td>Deliver 1 breath every 5 sec, 2 sec per breath (12/min)</td>
</tr>
<tr>
<td>Carotid pulse not palpable (omitted with lay rescuers)</td>
<td>Initiate external cardiac compressions</td>
<td>C—Restore circulation</td>
<td>10 sec to establish pulselessness (lay rescuers omit)</td>
</tr>
<tr>
<td>ECG: ventricular fibrillation</td>
<td>Drug therapy; defibrillation</td>
<td>D—Provide definitive treatment</td>
<td>Compression rate of 80-100/min Compression depth 1.5-2 in</td>
</tr>
</tbody>
</table>

ECG: Electrocardiogram.

Figure 29-49 Head tilt–chin lift maneuver for opening airway. Place one hand on forehead and place tips of fingers of other hand under lower jaw near chin. Bring chin forward while pressing forehead down.

Figure 29-50 Jaw thrust maneuver for opening airway.

Figure 29-51 Locating carotid artery.
Step 4: Assess Circulation

The rescuer palpates the carotid pulse to determine whether cardiac compression is needed. The carotid pulse is located by finding the larynx and sliding the fingers laterally into the groove between the trachea and the sternocleidomastoid muscle (Figure 29-51). If the carotid pulse is not palpable in 5 to 10 seconds, the rescuer initiates cardiac compressions. The carotid pulse is palpated because it is accessible and the carotid arteries are central. Sometimes these pulses persist when more peripheral pulses are no longer palpable. If the pulse is absent, cardiac arrest is confirmed and external chest compression is initiated. Lay rescuers are no longer being taught to check for a pulse because their accuracy in pulse assessment is only about 65%. Other indicators of circulation are checked instead, including breathing, coughing, and movement.

Step 5: Initiate External Cardiac Compression

External cardiac compression (sometimes called external cardiac massage) is the rhythmic compression of the heart between the lower half of the sternum and the thoracic vertebra. This intermittent pressure compresses the heart, raises intrathoracic pressure, and produces an artificial pulsatile circulation. Correctly performed cardiac compressions can produce a peak systolic blood pressure of more than 100 mm Hg. The diastolic pressure is close to 0 mm Hg, however, and the mean blood pressure in the carotid arteries is approximately 40 mm Hg, or about one-fourth normal. The technique for performing external cardiac compression is as follows:

1. Take a position close to the victim’s side. Using the middle finger of one hand, locate the xiphoid process (Figure 29-52, A). Place the index finger of the same hand on the sternum directly next to the middle finger. Using the index finger as a landmark, place the heel of the opposite hand on the sternum next to the index finger (Figure 29-52, B). Then place the first hand on top of the hand on the sternum (Figure 29-52, C). Fingers may be interlocked to avoid pressure on the patient’s ribs (Figure 29-52, D).

2. To perform effective external cardiac compression, take a position directly over the victim’s shoulders, keeping the elbows locked in a straight position, and depress the lower sternum 1½ to 2 inches. The compressions are regular, smooth, and uninterrupted. After each compression, release the pressure completely to allow the heart to refill. Establish a compression rate of 80 to 100/min with a ratio of compressions to breaths of 15:2. Deliver two full breaths after every 15 compressions (Figure 29-53).
Equipment needs include an ECG machine, suction device, oxygen, defibrillator, breathing bag, laryngoscope, variety of endotracheal tubes, cuffed down set, intravenous fluids, and tracheostomy set. Medications administered during a cardiac arrest are usually stored on an emergency cart. Most hospitals have trained teams, including physicians, nurses, anesthesiologists, and technicians, who provide immediate care in the event of a cardiac arrest. In recent years nursing research has explored the effects of cardiac arrest on family members, including the effects of witnessing the cardiac arrest of a loved one29 (see Research box).

Complications of Cardiopulmonary Resuscitation

The most common complication of external cardiac compression is fracture of the ribs. This may occur even when external cardiac compression is performed correctly. Other possible complications include fractured sternum, costochondral separation, and lung contusions. Any indication of labored respiration, paradoxical pulse, muffled heart sounds, tachycardia, decreased breath sounds, or a drop in blood pressure may indicate pericardial tamponade from the injection of intracardiac medications and is reported to the physician immediately. Laceration of the liver also may occur as a result of compressions performed over the xiphoid process.

Advanced Cardiac Life Support

The AHA regularly reviews and updates algorithms for advanced life support. The AHA website (www.americanheart.org) provides the most current practice guidelines and information about training sessions for beginning and advanced cardiac life support. Equipment needs include an ECG machine, suction device, oxy-

3. When two rescuers are available to administer CPR, one rescuer is positioned at the victim’s side and performs external cardiac compression while the second rescuer remains at the victim’s head to perform artificial ventilation. The cardiac compression rate remains the same, but a 5:1 ratio of cardiac compressions to ventilation is established. The rescuer who is ventilating the victim quickly delivers one full breath (2 seconds) after every five compressions; compressions are paused to allow for a full breath to be delivered. Two-rescuer CPR is advocated only for skilled providers because of the coordination required to appropriately time the interventions.

4. After the first minute of CPR, again palpate the carotid pulse to assess the effectiveness of CPR and to check for the return of spontaneous circulation. If two rescuers are performing CPR, the person ventilating the victim also assesses pulses and monitors for the return of spontaneous breathing. Continue to perform CPR until one of the following takes place:
   a. Spontaneous circulation and ventilation return.
   b. Another rescuer takes over basic life support.
   c. The victim is transported to an emergency facility.
   d. The victim is pronounced dead by a physician.
   e. The rescuer is exhausted and unable to continue.

Research


The presence of family members during cardiopulmonary resuscitation (CPR) is now a focus of nursing research. Using a qualitative design, Wagner interviewed six family members who were present at the onset of CPR in the coronary care unit at one 700-bed urban community hospital. Interviews were conducted within 24 hours of the event. Open-ended questions addressed where the family was at the time of the event, feelings and emotions experienced, support during the event, and communication with the health care team during the event. The overall theme from the research was: “Should we go or should we stay?” In the themes of CPR, two subthemes emerged: “What is going on?” and “You do your job.” At this point, family members negotiated to stay in the room or acquiesced to the request that they leave. Family members wanted information and expected answers, yet trusted the health care team to do their job. After the crisis, family members moved into a phase Wagner calls “breaking the rules.” During this time family members increased their vigilance regarding their loved ones. Formal and informal permission of the health care workers allowed a renegotiation of visiting hours and family presence, but only after the patient’s condition was fully stabilized.

Wagner concluded that the health care team takes control during CPR simply by determining whether family members are allowed to stay during CPR. This health care team control leads to the sense of a loss of autonomy by families who seek to be with their critically ill family member. Control by the health care team denies the family the opportunity to be vigilant, a need expressed by families after observing CPR. A lack of communication during resuscitation intensifies this loss of autonomy. Wagner suggests that further study of family members’ perceptions during CPR needs to occur, including the study of a liaison role between family members and the health care team during CPR.
Coronary Artery Disease and Dysrhythmias  Chapter 29  803

Critical Thinking

1. A 49-year-old man is admitted to the emergency department to rule out MI. His chest pain began at an intensity of 3 out of 10 at approximately 7:45 AM this morning on his drive to work. He reported that he had to make a presentation for which he did not feel adequately prepared and had gotten little rest the night before. Once at work, he drank some coffee and took ibuprofen for the pain. He tried to give the ibuprofen time to work, but the unrelenting pain finally caused him to confide in a co-worker at approximately 9 AM. He asked his co-worker if he thought Maalox or “something else” might get rid of the pain. It took an additional 30 minutes before the co-worker was able to convince him that he should go to the emergency department. He tried to “tough it out” with pain rated at an 8 out of 10. On arrival in the emergency department at 10:30 AM, his pain had reached 10/10, radiated to his left arm and up into his jaw. It was described as “gnawing and unrelenting.”

a. What diagnostic tests will be done in the emergency department to rule out MI? What is being looked for on these diagnostic tests to confirm or rule out MI?

b. The patient is being considered for reperfusion therapy. What collaborative interventions are indicated for patients experiencing MI and being screened for reperfusion therapy? Consider the eligibility requirements for both thrombolytic therapy and primary PCIs.

c. The patient receives a stent of the right coronary artery. You are with his wife as she awaits news of the procedure. How will you explain the procedure to his wife? What will you teach her regarding his postprocedure care?

d. The patient is to be discharged today after successful stenting. He lives with his wife of 22 years, a 17-year-old son, and a 13-year-old daughter. He works in middle management at a local company that has been downsizing. His wife is a physical therapist at a long-term nursing facility. Their home is two stories and has all modern conveniences. He and his family attend church weekly and are involved in various activities. A cholesterol screen done on admission showed a total cholesterol of 202. He does not routinely take any medications. He smokes one half to one pack of cigarettes a day, but only at the office, since his wife does not wish him to smoke in their home. He smoked a pack and a half a day until 5 years ago. He does not use alcohol.

Preparing for Practice

Patient: Sally Begay, Room 304

Sally Begay, a 58-year-old Navajo woman was admitted with a rule out diagnosis of Hantavirus that was subsequently determined to be pneumonia. Ms. Begay has a history of hypertension, coronary disease, and myocardial infarction (MI).

History

View the patient’s Report.

Review Sally Begay’s Medical Record; examine the History & Physical report in detail.

Assessment

Conduct a Patient Interview. As you conduct your interview, focus primarily on data that will be helpful in planning care for this patient. Record the data you collect.

Nursing Diagnoses, Outcomes, and Interventions

1. When obtaining a health history to determine risk of cardiac disease, identify and describe six important clinical manifestations to inquire about. *Hint:* use information on p. XXX if needed.

2. Sally Begay is a woman of the Navajo Indian tribe. What role does race play in the development of coronary artery disease?

3. The medical record clearly indicates that Sally Begay is experiencing pain in her chest. How might you differentiate chest pain of cardiac origin from the chest pain she is experiencing because of pneumonia? *Hint:* refer to p. XXX.

4. Sally Begay’s significant medical history lists “MI five years ago, mild CHF, and stable angina” in the section on heart disease in the Physical and History. What is angina?

5. Describe what occurs during an episode of angina. *Hint:* see p. XXX.

6. According to Sally Begay’s history, how frequently does she experience chest pain?

7. Formulate a plan of care that reflects your knowledge of the factors that could provoke Ms. Begay’s angina during this admission. Identify nursing interventions that address those factors. *Hint:* refer to p. XXX discussion of increased myocardial oxygen demands.

8. Sally Begay had an MI 5 years ago. What is an MI, and what happens during the process?
As the nurse responsible for his discharge today, develop a comprehensive teaching plan based on what you would expect the medical orders to be for this patient. Consider diet, medications, activity, and risk factor modification.

2. The rhythm strip illustrates which of the following rhythms? shows:
   a. Sinus tachycardia
   b. Atrial tachycardia
   c. Ventricular tachycardia

Discuss possible causes of this rhythm and its treatment.

References