LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Correlate the components of the normal ECG with physiologic events of the heart.
2. Define the ECG as a waveform that represents the cardiac electrical event in relation to the lead depicted (placement of electrodes).
3. Analyze elements of an ECG rhythm strip: ventricular and atrial rate, ventricular and atrial rhythm, QRS complex and shape, QRS duration, P wave and shape, PR interval, and P:QRS ratio.
4. Identify the ECG criteria, causes, and management of several dysrhythmias, including conduction disturbances.
5. Use the nursing process as a framework for care of patients with dysrhythmias.
6. Compare the different types of pacemakers, their uses, possible complications, and nursing implications.
7. Use the nursing process as a framework for care of patients with pacemakers.
8. Describe the key points of using a defibrillator.
9. Describe the purpose of an implantable cardioverter defibrillator (ICD), the types available, and the nursing implications.
10. Describe invasive methods to diagnose and treat recurrent dysrhythmias, and discuss the nursing implications.
Without a regular rate and rhythm, the heart may not perform efficiently as a pump to circulate oxygenated blood and other life-sustaining nutrients to all the body organs (including itself) and tissues. With an irregular or erratic rhythm, the heart is considered to be dysrhythmic (sometimes called arrhythmic). This has the potential to be a dangerous condition.

**Dysrhythmias**

Dysrhythmias are disorders of the formation or conduction (or both) of the electrical impulse within the heart. These disorders can cause disturbances of the heart rate, the heart rhythm, or both. Dysrhythmias may initially be evidenced by the hemodynamic effect they cause (eg, a change in conduction may change the pumping action of the heart and cause decreased blood pressure). Dysrhythmias are diagnosed by analyzing the electrocardiographic waveform. They are named according to the site of origin of the impulse and the mechanism of formation or conduction involved (Chart 27–1). For example, an impulse that originates in the sinoatrial (SA) node and that has a slow rate is called sinus bradycardia.

**NORMAL ELECTRICAL CONDUCTION**

The electrical impulse that stimulates and paces the cardiac muscle normally originates in the sinus node (SA node), an area located near the superior vena cava in the right atrium. Usually, the electrical impulse occurs at a rate ranging between 60 and 100 times a minute in the adult. The electrical impulse quickly travels from the sinus node through the atria to the atrioventricular (AV) node (Fig. 27–1). The electrical stimulation of the muscle cells of the atria causes them to contract. The structure of the AV node slows the electrical impulse, which allows time for the atria to contract and fill the ventricles with blood before the electrical impulse travels very quickly through the bundle of His to the right and left bundle branches and the Purkinje fibers, located in the ventricular muscle. The electrical stimulation of the muscle cells of the ventricles, in turn, causes the mechanical contraction of the ventricles (systole). The cells repolarize and the ventricles then relax (diastole). The process from sinus node electrical impulse generation through ventricular repolarization completes the electromechanical circuit, and the cycle begins again.

Sinus rhythm promotes cardiovascular circulation. The electrical impulse causes (and, therefore, is followed by) the mechanical contraction of the heart muscle. The electrical stimulation is called depolarization; the mechanical contraction is called systole. Electrical relaxation is called repolarization and mechanical relaxation is called diastole. See Chapter 26 for a more complete explanation of cardiac function.

**Glossary**

- **ablation**: purposeful destruction of heart muscle cells, usually in an attempt to control a dysrhythmia
- **antiarrhythmics**: a medication that suppresses or prevents a dysrhythmia
- **automaticity**: ability of the cardiac muscle to initiate an electrical impulse
- **cardioversion**: electrical current administered in synchrony with the patient’s own QRS to stop a dysrhythmia
- **conductivity**: ability of the cardiac muscle to transmit electrical impulses
- **defibrillation**: electrical current administered to stop a dysrhythmia, not synchronized with the patient’s QRS complex
- **depolarization**: process by which cardiac muscle cells change from a more negatively charged intracellular state to a resting state
- **dysrhythmia** (also referred to as arrhythmia): disorder of the formation or conduction (or both) of the electrical impulse within the heart, altering the heart rate, heart rhythm, or both and potentially causing altered blood flow
- **implantable cardioverter defibrillator** (ICD): a device implanted into the chest to treat dysrhythmias
- **inhibited**: in reference to pacemakers, term used to describe the pacemaker withholding an impulse (not firing)
- **P wave**: the part of an electrocardiogram (ECG) that reflects conduction of an electrical impulse from the atria to the atrioventricular (AV) node
- **paroxysmal**: a dysrhythmia that has a sudden onset and/or termination and is usually of short duration
- **PR interval**: the part of an ECG that reflects conduction of an electrical impulse from the sinoatrial (SA) node through the atrioventricular (AV) node
- **proarrhythmic**: an agent (eg, a medication) that causes or exacerbates a dysrhythmia
- **QRS complex**: the part of an ECG that reflects conduction of an electrical impulse through the ventricles; ventricular depolarization
- **QT interval**: the part of an ECG that reflects the time from ventricular depolarization to repolarization
- **repolarization**: process by which cardiac muscle cells return to a more negatively charged intracellular condition, their resting state
- **sinus rhythm**: electrical activity of the heart initiated by the sinoatrial (SA) node
- **ST segment**: the part of an ECG that reflects the end of ventricular depolarization (end of the QRS complex) through ventricular repolarization (end of the T wave)
- **supraventricular tachycardia** (SVT): a rhythm that originates in the conduction system above the ventricles
- **T wave**: the part of an ECG that reflects ventricular repolarization of the ventricles
- **triggered**: in reference to pacemakers, term used to describe the release of an impulse in response to some stimulus
- **U wave**: the part of an ECG that may reflect Purkinje fiber repolarization; usually seen when a patient’s serum potassium level is low
- **ventricular tachycardia** (VT): a rhythm that originates in the ventricles
Influences on Heart Rate and Contractility

The heart rate is influenced by the autonomic nervous system, which consists of sympathetic and parasympathetic fibers. Sympathetic nerve fibers (also referred to as adrenergic fibers) are attached to the heart and arteries as well as several other areas in the body. Stimulation of the sympathetic system increases heart rate (positive chronotropy), conduction through the AV node (positive dromotropy), and the force of myocardial contraction (positive inotropy). Sympathetic stimulation also constricts peripheral blood vessels, therefore increasing blood pressure. Parasympathetic nerve fibers are also attached to the heart and arteries. Parasympathetic stimulation reduces the heart rate (negative chronotropy), AV conduction (negative dromotropy), and the force of atrial myocardial contraction. The decreased sympathetic stimulation results in dilation of arteries, thereby lowering blood pressure.

Manipulation of the autonomic nervous system may increase or decrease the incidence of dysrhythmias. Increased sympathetic stimulation—caused, for example, by exercise, anxiety, fever, or administration of catecholamines (eg, dopamine [Intropin], aminophylline, dobutamine [Dobutrex])—may increase the incidence of dysrhythmias. Decreased sympathetic stimulation (eg, with rest, anxiety-reduction methods such as therapeutic communication or prayer, administration of beta-adrenergic blocking agents) may decrease the incidence of dysrhythmias.

**INTERPRETATION OF THE ELECTROCARDIOGRAM**

The electrical impulse that travels through the heart can be viewed by means of electrocardiography, the end product of which is an electrocardiogram (ECG). Each phase of the cardiac cycle is re-
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An ECG is obtained by slightly abrading the skin with a clean dry gauze pad and placing electrodes on the body at specific areas. Electrodes come in various shapes and sizes, but all have two components: (1) an adhesive substance that attaches to the skin to secure the electrode in place and (2) a substance that reduces the skin's electrical impedance and promotes detection of the electrical current.

The number and placement of the electrodes depend on the type of ECG needed. Most continuous monitors use two to five electrodes, usually placed on the limbs and the chest. These electrodes create an imaginary line, called a lead, that serves as a reference point from which the electrical activity is viewed. A lead is like an eye of a camera; it has a narrow peripheral field of vision, looking only at the electrical activity directly in front of it. Therefore, the ECG waveforms that appear on the paper or cardiac monitor represent the electrical current in relation to the lead (see Fig. 27-1). A change in the waveform can be caused by a change in the electrical current (where it originates or how it is conducted) or by a change in the lead.

**Obtaining an Electrocardiogram**

Electrodes are attached to cable wires, which are connected to one of the following:

- An ECG machine placed at the patient’s side for an immediate recording (standard 12-lead ECG)
- A cardiac monitor at the patient’s bedside for continuous reading; this kind of monitoring, usually called hardwire monitoring, is associated with intensive care units
- A small box that the patient carries and that continuously transmits the ECG information by radio waves to a central monitor located elsewhere (called telemetry)
- A small, lightweight tape recorder-like machine (called a Holter monitor) that the patient wears and that continuously records the ECG on a tape, which is later viewed and analyzed with a scanner

The placement of electrodes for continuous monitoring, telemetry, or Holter monitoring varies with the type of technology that is appropriate and available, the purpose of monitoring, and the standards of the institution. For a standard 12-lead ECG, 10 electrodes (six on the chest and four on the limbs) are placed on the body (Fig. 27-2). To prevent interference from the electrical activity of skeletal muscle, the limb electrodes are usually placed on areas that are not bony and that do not have significant movement. These limb electrodes provide the first six leads: leads I, II, III, aVR, aVL, and aVF. The six chest electrodes are attached to the chest at very specific areas. The chest electrodes provide the V or precordial leads, V1 through V6. To locate the fourth intercostal space and the placement of V1, locate the sternal angle and then the sternal notch, which is about 1 or 2 inches below the sternal angle. When the fingers are moved to the patient’s immediate right, the second rib can be palpated. The second intercostal space is the indentation felt just below the second rib.

Locating the specific intercostal space is critical for correct chest electrode placement. Errors in diagnosis can occur if electrodes are incorrectly placed. Sometimes, when the patient is in the hospital and needs to be monitored closely for ECG changes, the chest electrodes are left in place to ensure the same placement for follow-up ECGs.

A standard 12-lead ECG reflects the electrical activity primarily in the left ventricle. Placement of additional electrodes for other leads may be needed to obtain more complete information. For example, in patients with suspected right-sided heart damage, right-sided precordial leads are required to evaluate the right ventricle (see Fig. 27-2).

**Analysis of the Electrocardiogram**

The ECG waveform represents the function of the heart’s conduction system, which normally initiates and conducts the electrical activity, in relation to the lead. When analyzed accurately, the ECG offers important information about the electrical activity...
of the heart. ECG waveforms are printed on graph paper that is divided by light and dark vertical and horizontal lines at standard intervals (Fig. 27-3). Time and rate are measured on the horizontal axis of the graph, and amplitude or voltage is measured on the vertical axis. When an ECG waveform moves toward the top of the paper, it is called a positive deflection. When it moves toward the bottom of the paper, it is called a negative deflection. When reviewing an ECG, each waveform should be examined and compared with the others.

**WAVES, COMPLEXES, AND INTERVALS**

The ECG is composed of waveforms (including the P wave, the QRS complex, the T wave, and possibly a U wave) and of segments or intervals (including the PR interval, the ST segment, and the QT interval) (see Fig. 27-3).

The **P wave** represents the electrical impulse starting in the sinus node and spreading through the atria. Therefore, the P wave represents atrial muscle depolarization. It is normally 2.5 mm or less in height and 0.11 second or less in duration.

The **QRS complex** represents ventricular muscle depolarization. Not all QRS complexes have all three waveforms. The first negative deflection after the P wave is the Q wave, which is normally less than 0.04 second in duration and less than 25% of the R wave amplitude; the first positive deflection after the P wave is the R wave; and the S wave is the first negative deflection after the R wave. When a wave is less than 5 mm in height, small letters (q, r, s) are used; when a wave is taller than 5 mm, capital letters (Q, R, S) are used. The QRS complex is normally less than 0.12 seconds in duration.

The **T wave** represents ventricular muscle repolarization (when the cells regain a negative charge; also called the resting state). It follows the QRS complex and is usually the same direction as the QRS complex.

The **U wave** is thought to represent repolarization of the Purkinje fibers, but it sometimes is seen in patients with hypokalemia (low potassium levels), hypertension, or heart disease. If present, the U wave follows the T wave and is usually smaller than the P wave. If tall, it may be mistaken for an extra P wave.

The **PR interval** is measured from the beginning of the P wave to the beginning of the QRS complex and represents the time needed for sinus node stimulation, atrial depolarization, and conduction through the AV node before ventricular depolarization. In adults, the PR interval normally ranges from 0.12 to 0.20 seconds in duration.

The **ST segment**, which represents early ventricular repolarization, lasts from the end of the QRS complex to the beginning of the T wave. The beginning of the ST segment is usually identified by a change in the thickness or angle of the terminal portion of the QRS complex. The end of the ST segment may be more difficult to identify because it merges into the T wave. The ST segment is normally isoelectric (see discussion of TP interval). It is analyzed to identify whether it is above or below the isoelectric line, which may be, among other signs and symptoms, a sign of cardiac ischemia (see Chap. 28).

The **QT interval**, which represents the total time for ventricular depolarization and repolarization, is measured from the beginning of the QRS complex to the end of the T wave. The QT interval varies with heart rate, gender, and age, and the measured interval needs to be corrected for these variables through a specific calculation. Several ECG interpretation books contain charts of these calculations. The QT interval is usually 0.32 to 0.40 seconds in duration if the heart rate is 65 to 95 beats per minute. If the QT interval becomes prolonged, the patient may be at risk for a lethal ventricular dysrhythmia called torsades de pointes.

The **TP interval** is measured from the end of the T wave to the beginning of the next P wave, an isoelectric period (see Fig 27-3).
When no electrical activity is detected, the line on the graph remains flat; this is called the isoelectric line. The ST segment is compared with the TP interval to detect changes from the line on the graph during the isoelectric period.

The PP interval is measured from the beginning of one P wave to the beginning of the next. The PP interval is used to determine atrial rhythm and atrial rate. The RR interval is measured from one QRS complex to the next QRS complex. The RR interval is used to determine ventricular rate and rhythm (Fig. 27-4).

**DETERMINING VENTRICULAR HEART RATE FROM THE ELECTROCARDIOGRAM**

Heart rate can be obtained from the ECG strip by several methods. A 1-minute strip contains 300 large boxes and 1500 small boxes. Therefore, an easy and accurate method of determining heart rate with a regular rhythm is to count the number of small boxes within an RR interval and divide 1500 by that number. If, for example, there are 10 small boxes between two R waves, the heart rate is 1500 ÷ 10, or 150; if there are 25 small boxes, the heart rate is 1500 ÷ 25, or 60 (see Fig. 27-4A).

An alternative but less accurate method for estimating heart rate, which is usually used when the rhythm is irregular, is to count the number of RR intervals in 6 seconds and multiply that number by 10. The top of the ECG paper is usually marked at 3-second intervals, which is 15 large boxes horizontally (see Fig. 27-4B). The RR intervals are counted, rather than QRS complexes, because a computed heart rate based on the latter might be inaccurately high.

The same methods may be used for determining atrial rate, using the PP interval instead of the RR interval.

**DETERMINING HEART RHYTHM FROM THE ELECTROCARDIOGRAM**

The rhythm is often identified at the same time the rate is determined. The RR interval is used to determine ventricular rhythm and the PP interval to determine atrial rhythm. If the intervals are the same or nearly the same throughout the strip, the rhythm is called regular. If the intervals are different, the rhythm is called irregular.

**ANALYZING THE ELECTROCARDIOGRAM RHYTHM STRIP**

The ECG must be analyzed in a systematic manner to determine the patient’s cardiac rhythm and to detect dysrhythmias and conduction disorders, as well as evidence of myocardial ischemia, injury, and infarction. Chart 27-2 is an example of a method that can be used to analyze the patient’s rhythm.

Once the rhythm has been analyzed, the findings are compared with and matched to the ECG criteria for dysrhythmias to determine a diagnosis. It is important for the nurse to assess the patient to determine the physiologic effect of the dysrhythmia and to identify possible causes. Treatment of dysrhythmias is based on the etiology and the effect of the dysrhythmia, not on its presence alone.

**Normal Sinus Rhythm**

Normal sinus rhythm occurs when the electrical impulse starts at a regular rate and rhythm in the sinus node and travels through the normal conduction pathway. The following are the ECG criteria for normal sinus rhythm (Fig. 27-5):

- **Ventricular and atrial rate:** 60 to 100 in the adult
- **Ventricular and atrial rhythm:** Regular
- **QRS shape and duration:** Usually normal, but may be regularly abnormal
- **P wave:** Normal and consistent shape; always in front of the QRS
- **PR interval:** Consistent interval between 0.12 and 0.20 seconds
- **P: QRS ratio:** 1:1

**Types of Dysrhythmias**

Dysrhythmias include sinus node, atrial, junctional, and ventricular dysrhythmias and their various subcategories.
SINUS NODE DYSRHYTHMIAS

Sinus Bradycardia. Sinus bradycardia occurs when the sinus node creates an impulse at a slower-than-normal rate. Causes include lower metabolic needs (e.g., sleep, athletic training, hypothermia, hypothyroidism), vagal stimulation (e.g., from vomiting, suctioning, severe pain, extreme emotions), medications (e.g., calcium channel blockers, amiodarone, beta-blockers), increased intracranial pressure, and myocardial infarction (MI), especially of the inferior wall.

The following are characteristics of sinus bradycardia (Fig. 27-6):

- Ventricular and atrial rate: Less than 60 in the adult
- Ventricular and atrial rhythm: Regular
- QRS shape and duration: Usually normal, but may be regularly abnormal
- P wave: Normal and consistent shape; always in front of the QRS
- PR interval: Consistent interval between 0.12 and 0.20 seconds
- P: QRS ratio: 1:1

All characteristics of sinus bradycardia are the same as those of normal sinus rhythm, except for the rate. The patient is assessed to determine the hemodynamic effect and the possible cause of the dysrhythmia. If the decrease in heart rate results from stimulation of the vagus nerve, such as with bearing down during defecation or vomiting, attempts are made to prevent further vagal stimulation. If the bradycardia is from a medication such as a beta-blocker, the medication may be withheld. If the slow heart rate causes significant hemodynamic changes, resulting in shortness of breath, decreased level of consciousness, angina, hypotension, ST-segment changes, or premature ventricular complexes, treatment is directed toward increasing the heart rate.

Atropine, 0.5 to 1.0 mg given rapidly as an intravenous (IV) bolus, is the medication of choice in treating sinus bradycardia. It blocks vagal stimulation, thus allowing a normal rate to occur. Rarely, catecholamines and emergency transcutaneous pacing also may be implemented.

Sinus Tachycardia. Sinus tachycardia occurs when the sinus node creates an impulse at a faster-than-normal rate. It may be caused by acute blood loss, anemia, shock, hypervolemia, hypovolemia, congestive heart failure, pain, hypermetabolic states, fever, exercise, anxiety, or sympathomimetic medications. The ECG criteria for sinus tachycardia follow (Fig. 27-7):

- Ventricular and atrial rate: Greater than 100 in the adult
- Ventricular and atrial rhythm: Regular
- QRS shape and duration: Usually normal, but may be regularly abnormal
- P wave: Normal and consistent shape; always in front of the QRS, but may be buried in the preceding T wave
- PR interval: Consistent interval between 0.12 and 0.20 seconds
- P: QRS ratio: 1:1

All aspects of sinus tachycardia are the same as those of normal sinus rhythm, except for the rate. As the heart rate increases, the diastolic filling time decreases, possibly resulting in reduced cardiac output and subsequent symptoms of syncope and low blood pressure. If the rapid rate persists and the heart cannot compensate for the decreased ventricular filling, the patient may develop acute pulmonary edema.

Treatment of sinus tachycardia is usually directed at abolishing its cause. Calcium channel blockers and beta-blockers (Table 27-1) may be used to reduce the heart rate quickly.

Sinus Arrhythmia. Sinus arrhythmia occurs when the sinus node creates an impulse at an irregular rhythm; the rate usually increases with inspiration and decreases with expiration. Nonrespiratory causes include heart disease and valvular disease, but these are rarely seen. The ECG criteria for sinus arrhythmia follow (Fig. 27-8):

- Ventricular and atrial rate: 60 to 100 in the adult
- Ventricular and atrial rhythm: Irregular
- QRS shape and duration: Usually normal, but may be regularly abnormal
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**Figure 27-6** Sinus bradycardia in lead II.

**Figure 27-7** Sinus tachycardia in lead II.

*P wave:* Normal and consistent shape; always in front of the QRS
PR interval: Consistent interval between 0.12 and 0.20 seconds
P: QRS ratio: 1:1

Sinus arrhythmia does not cause any significant hemodynamic effect and usually is not treated.

**Atrial Dysrhythmias**

**Premature Atrial Complex.** A premature atrial complex (PAC) is a single ECG complex that occurs when an electrical impulse starts in the atrium before the next normal impulse of the sinus node. The PAC may be caused by caffeine, alcohol, nicotine, stretched atrial myocardium (as in hypervolemia), anxiety, hypokalemia (low potassium level), hypermetabolic states, or atrial ischemia, injury, or infarction. PACs are often seen with sinus tachycardia. PACs have the following characteristics (Fig. 27-9):

- Ventricular and atrial rate: Depends on the underlying rhythm (eg, sinus tachycardia)
- Ventricular and atrial rhythm: Irregular due to early P waves, creating a PP interval that is shorter than the others. This is sometimes followed by a longer-than-normal PP interval, but one that is less than twice the normal PP interval. This type of interval is called a noncompensatory pause.
- QRS shape and duration: The QRS that follows the early P wave is usually normal, but it may be abnormal (aberrantly conducted PAC). It may even be absent (blocked PAC).
- P wave: An early and different P wave may be seen or may be hidden in the T wave; other P waves in the strip are consistent.
- PR interval: The early P wave has a shorter-than-normal PR interval, but still between 0.12 and 0.20 seconds.
- P: QRS ratio: usually 1:1

PACs are common in normal hearts. The patient may say, “My heart skipped a beat.” A pulse deficit (a difference between the apical and radial pulse rate) may exist.

If PACs are infrequent, no treatment is necessary. If they are frequent (more than 6 per minute), this may herald a worsening disease state or the onset of more serious dysrhythmias, such as atrial fibrillation. Treatment is directed toward the cause.

**Atrial Flutter.** Atrial flutter occurs in the atrium and creates impulses at an atrial rate between 250 and 400 times per minute. Because the atrial rate is faster than the AV node can conduct, not all atrial impulses are conducted into the ventricle, causing a therapeutic block at the AV node. This is an important feature of this dysrhythmia. If all atrial impulses were conducted to the ventricle, the ventricular rate would also be 250 to 400, which would result in ventricular fibrillation, a life-threatening dysrhythmia. Causes are similar to that of atrial fibrillation. Atrial flutter is characterized by the following (Fig. 27-10):

- Ventricular and atrial rate: Atrial rate ranges between 250 and 400; ventricular rate usually ranges between 75 and 150.
- Ventricular and atrial rhythm: The atrial rhythm is regular; the ventricular rhythm is usually regular but may be irregular because of a change in the AV conduction.
- QRS shape and duration: Usually normal, but may be abnormal or may be absent
- P wave: Saw-toothed shape. These waves are referred to as F waves.
- PR interval: Multiple F waves may make it difficult to determine the PR interval.
- P: QRS ratio: 2:1, 3:1, or 4:1

Atrial flutter can cause serious signs and symptoms, such as chest pain, shortness of breath, and low blood pressure. If the
### Table 27-1 • Summary of Antiarrhythmic Medications*

<table>
<thead>
<tr>
<th>CLASS</th>
<th>ACTION</th>
<th>DRUGS: GENERIC (TRADE) NAMES</th>
<th>SIDE EFFECTS</th>
<th>NURSING INTERVENTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A</td>
<td>Moderate depression of depolarization; prolongs repolarization</td>
<td>quinidine (Quinaglute, Quinalan, Quinora, Quinidex, Cardioquin), procainamide (Pronestyl), disopyramide (Norpace)</td>
<td>Decreased cardiac contractility, Prolonged QRS, QT, Proarrhythmic, Hypotension with IV administration, Lupus-like syndrome with Pronestyl, Anticholinergic effects: dry mouth, decreased urine output</td>
<td>Observe for HF, Monitor BP with IV administration, Monitor QRS duration for increase &gt;50% from baseline, Monitor for prolonged QT, Monitor N-acetyl procainamide (NAPA) laboratory values during procainamide therapy</td>
</tr>
<tr>
<td>1B</td>
<td>Minimal depression of depolarization; shortened repolarization</td>
<td>lidocaine (Xylocaine), mexiletine (Mexitil), tocainide (Tonocard)</td>
<td>CNS changes (eg, confusion, lethargy)</td>
<td>Discuss with physician decreasing the dose in elderly patients and patients with cardiac/liver dysfunction</td>
</tr>
<tr>
<td>1C</td>
<td>Marked depression of depolarization; little effect on repolarization</td>
<td>flecaïnide (Tambocor), propafenone (Rhythm)</td>
<td>Proarrhythmic, HF, Bradycardia, AV blocks</td>
<td>Discuss patient’s left ventricular function with physician</td>
</tr>
<tr>
<td>II</td>
<td>Decreases automaticity and conduction</td>
<td>acebutolol (Sectral), atenolol (Tenormin), esmolol (Brevibloc), labetalol (Normodyne), metoprolol (Lopressor, Toprol), nadolol (Corgard), propranolol (Betachron E-R, Inderal), sotalol (Betapace; also has class III actions)</td>
<td>Bradycardia, AV block, Decreased contractility, Bronchospasm, Hypotension with IV administration, Masks hypoglycemia and thyrotoxicosis, CNS disturbances</td>
<td>Monitor heart rate, PR interval, signs and symptoms of HF, Monitor blood glucose level in patients with type 2 diabetes mellitus</td>
</tr>
<tr>
<td>III</td>
<td>Prolongs repolarization</td>
<td>amiodarone (Cordarone, Pacerone), dofetilide (Tikosyn), ibutilide (Corvert)</td>
<td>Pulmonary toxicity (amiodarone), Corneal microdeposits (amiodarone), Photosensitivity (amiodarone), Hypotension with IV administration, Polymorphic ventricular dysrhythmias, Nausea and vomiting, See beta-blockers (sotalol)</td>
<td>Make sure patient is sent for baseline pulmonary function tests (amiodarone), Closely monitor patient</td>
</tr>
<tr>
<td>IV</td>
<td>Blocks calcium channel</td>
<td>verapamil (Calan, Isoptin, Verlan), diltiazem (Cardizem, Dilacor, Tiazac)</td>
<td>Bradycardia, AV blocks, Hypotension with IV administration, HF, peripheral edema</td>
<td>Monitor heart rate, PR interval, Monitor blood pressure closely with IV administration, Monitor for signs and symptoms of HF</td>
</tr>
</tbody>
</table>

*Based on Vaughan-Williams classification. AV, atrioventricular; BP, blood pressure; CNS, central nervous system; HF, heart failure; IV, intravenous.
patient is unstable, electrical cardioversion (discussed later) is usually indicated. If the patient is stable, diltiazem (eg, Cardizem), verapamil (eg, Calan, Isoptin), beta-blockers, or digitalis may be administered intravenously to slow the ventricular rate. These medications can slow conduction through the AV node. Flecainide (Tambocor), ibutilide (Corvert), dofetilide (Tikosyn), quinidine (eg, Cardioquin, Quinaglute), disopyramide (Norpace), or amiodarone (Cordarone, Pacerone) may be given to promote conversion to sinus rhythm (see Table 27-1). If medication therapy is unsuccessful, electrical cardioversion is often successful. Once conversion has occurred, quinidine, disopyramide, flecainide, propafenone (Rhythmol), amiodarone, or sotalol (Betapace) may be given to maintain sinus rhythm (see Table 27-1).

Atrial Fibrillation. Atrial fibrillation causes a rapid, disorganized, and uncoordinated twitching of atrial musculature. It is the most common dysrhythmia that causes patients to seek medical attention. It may start and stop suddenly. Atrial fibrillation may occur for a very short time (paroxysmal), or it may be chronic. Atrial fibrillation is usually associated with advanced age, valvular heart disease, coronary artery disease, hypertension, cardiomyopathy, hyperthyroidism, pulmonary disease, acute moderate to heavy ingestion of alcohol ("holiday heart" syndrome), or the aftermath of open heart surgery. Sometimes it occurs in people without any underlying pathophysiology (termed lone atrial fibrillation). Atrial fibrillation is characterized by the following (Fig. 27-11):

- **Ventricular and atrial rate**: Atrial rate is 300 to 600. Ventricular rate is usually 120 to 200 in untreated atrial fibrillation
- **Ventricular and atrial rhythm**: Highly irregular
- **QRS shape and duration**: Usually normal, but may be abnormal
- **P wave**: No discernible P waves; irregular undulating waves are seen and are referred to as fibrillatory or f waves
- **PR interval**: Cannot be measured
- **P:QRS ratio**: Many

A rapid ventricular response reduces the time for ventricular filling, resulting in a smaller stroke volume. Because this rhythm causes the atria and ventricles to contract at different times, the atrial kick (the last part of diastole and ventricular filling, which accounts for 25% to 30% of the cardiac output) is also lost. This leads to symptoms of irregular palpitations, fatigue, and malaise. There is usually a pulse deficit, a numerical difference between apical and radial pulse rates. The shorter time in diastole reduces the time available for coronary artery perfusion, thereby increasing the risk for myocardial ischemia. The erratic atrial contraction promotes the formation of a thrombus within the atria, increasing the risk for an embolic event. There is a two- to five-fold increase in the risk of stroke (brain attack).

Treatment of atrial fibrillation depends on its cause and duration and the patient’s symptoms, age, and comorbidities. In many patients, atrial fibrillation converts to sinus rhythm within 24 hours and without treatment. Both stable and unstable atrial fibrillation...
of short duration are treated the same as stable and unstable atrial flutter. Cardioversion may be indicated for atrial fibrillation that has been present for less than 48 hours, a condition termed acute-onset atrial fibrillation. Cardioversion of atrial fibrillation that has lasted longer than 48 hours should be avoided unless the patient has received anticoagulants, due to the high risk for embolization of atrial thrombi.

For atrial fibrillation of acute onset, the medications quinidine, ibutilide, flecainide, dofetilide, propafenone, procainamide (Pronestyl), disopyramide, or amiodarone (see Table 27-1) may be given to achieve conversion to sinus rhythm (McNamara et al., 2001). Intravenous adenosine (Adenocard, Adenoscan) has also been used for conversion, as well as to assist in the diagnosis. To prevent recurrence and to maintain sinus rhythm, quinidine, disopyramide, flecainide, propafenone, sotalol, or amiodarone may be prescribed. Calcium-channel blockers [diltiazem (Cardizem, Dilacor, Tiazac) and verapamil (Calan, Isoptin, Verelan)] and beta blockers (see Table 27-1) are effective in controlling the ventricular rate in atrial fibrillation, especially during exercise (McNamara, et al., 2001). Use of digoxin is recommended to control the ventricular rate in those patients with poor cardiac function (ejection fraction less than 40%) (Hauptman & Kelly, 1999). In addition, warfarin is indicated if the patient is at higher risk for a stroke (ie, is elderly or has hypertension, heart failure, or a history of stroke). Aspirin may be substituted for warfarin for those with contraindications to warfarin and those who are at lower risk of stroke. The choice of antithrombotic medication can be guided by transesophageal echocardiography. Pacemaker implantation or surgery is sometimes indicated for patients who are unresponsive to medications.

**JUNCTIONAL DYSRHYTHMIAS**

**Premature Junctional Complex.** A premature junctional complex is an impulse that starts in the AV nodal area before the next normal sinus impulse reaches the AV node. Premature junctional complexes are less common than PACs. Causes of premature junctional complex include digitalis toxicity, congestive heart failure, and coronary artery disease. The ECG criteria for premature junctional complex are the same as for PACs, except for the P wave and the PR interval. The P wave may be absent, may follow the QRS, or may occur before the QRS but with a PR interval of less than 0.12 seconds. Premature junctional complexes rarely produce significant symptoms. Treatment for frequent premature junctional complexes is the same as for frequent PACs.

**Junctional Rhythm.** Junctional or idionodal rhythm occurs when the AV node, instead of the sinus node, becomes the pacemaker of the heart. When the sinus node slows (eg, from increased vagal tone) or when the impulse cannot be conducted through the AV node (eg, because of complete heart block), the AV node automatically discharges an impulse. The following are the ECG criteria for junctional rhythm not caused by complete heart block (Fig. 27-12):**

- **Ventricular and atrial rate:** Ventricular rate 40 to 60; atrial rate also 40 to 60 if P waves are discernible
- **Ventricular and atrial rhythm:** Regular
- **QRS shape and duration:** Usually normal, but may be abnormal
- **P wave:** May be absent, after the QRS complex, or before the QRS; may be inverted, especially in lead II

**FIGURE 27-11** Atrial fibrillation in lead II.

**FIGURE 27-12** Junctional rhythm in lead II; note short PR intervals.
PR interval: If P wave is in front of the QRS, PR interval is less than 0.12 second.
P: QRS ratio: 1:1 or 0:1

Junctional rhythm may produce signs and symptoms of reduced cardiac output. If so, the treatment is the same as for sinus bradycardia. Emergency pacing may be needed.

Atrioventricular Nodal Reentry Tachycardia. AV nodal reentry tachycardia occurs when an impulse is conducted to an area in the AV node that causes the impulse to be rerouted back into the same area over and over again at a very fast rate. Each time the impulse is conducted through this area, it is also conducted down into the ventricles, causing a fast ventricular rate. AV nodal reentry tachycardia that has an abrupt onset and an abrupt cessation with a QRS of normal duration had been called paroxysmal atrial tachycardia (PAT). Factors associated with the development of AV nodal reentry tachycardia include caffeine, nicotine, hypoxemia, and stress. Underlying pathologies include coronary artery disease and cardiomyopathy. The ECG criteria are as follows (Fig. 27-13):

Ventricular and atrial rate: Atrial rate usually ranges between 150 to 250; ventricular rate usually ranges between 75 to 250
Ventricular and atrial rhythm: Regular; sudden onset and termination of the tachycardia
QRS shape and duration: Usually normal, but may be abnormal
P wave: Usually very difficult to discern
PR interval: If P wave is in front of the QRS, PR interval is less than 0.12 seconds
P: QRS ratio: 1:1, 2:1

The clinical symptoms vary with the rate and duration of the tachycardia and the patient’s underlying condition. The tachycardia usually is of short duration, resulting only in palpitations. A fast rate may also reduce cardiac output, resulting in significant signs and symptoms such as restlessness, chest pain, shortness of breath, pallor, hypotension, and loss of consciousness.

Treatment is aimed at breaking the reentry of the impulse. Vagal maneuvers, such as carotid sinus massage (Fig. 27-14), gag reflex, breath holding, and immersing the face in ice water, increase parasympathetic stimulation, causing slower conduction through the AV node and blocking the reentry of the rerouted impulse. Some patients have learned to use some of these methods to terminate the episode on their own. Because of the risk of a cerebral embolic event, carotid sinus massage is contraindicated in patients with carotid bruits. If the vagal maneuvers are ineffective, the patient may then receive a bolus of adenosine, verapamil, or diltiazem. Cardioversion is the treatment of choice if the patient is unstable or does not respond to the medications.

If P waves cannot be identified, the rhythm may be called supraventricular tachycardia (SVT), which indicates only that it is not ventricular tachycardia (VT). SVT could be atrial fibrillation, atrial flutter, or AV nodal reentry tachycardia, among others. Vagal maneuvers and adenosine are used to slow conduction in the AV node to allow visualization of the P waves.

VENTRICULAR DYSRHYTHMIAS

Premature Ventricular Complex. Premature ventricular complex (PVC) is an impulse that starts in a ventricle and is conducted through the ventricles before the next normal sinus impulse. PVCs can occur in healthy people, especially with the use of caffeine, nicotine, or alcohol. They are also caused by cardiac ischemia or infarction, increased workload on the heart (eg, exercise, fever, hypervolemia, heart failure, tachycardia), digitalis toxicity, hypoxia, acidosis, or electrolyte imbalances, especially hypokalemia.

In the absence of disease, PVCs are not serious. In the patient with an acute MI, PVCs may indicate the need for more aggressive therapy. PVCs may indicate the possibility of ensuing VT. However, PVCs that are (1) more frequent than 6 per minute,
Ventricular tachycardia (VT) is de-

monly used for immediate, short-term therapy (see Table 27-1).

In a rhythm called bigeminy, every other complex is a PVC. Trigeminy is a rhythm in which every third complex is a PVC, and quadrigeminy is a rhythm in which every fourth complex is a PVC. PVCs have the following characteristics on the ECG (Fig. 27-15):

- **Ventricular and atrial rate**: Depends on the underlying rhythm (eg, sinus rhythm)
- **Ventricular and atrial rhythm**: Irregular due to early QRS, creating one RR interval that is shorter than the others. PP interval may be regular, indicating that the PVC did not depolarize the sinus node.
- **QRS shape and duration**: Duration is 0.12 seconds or longer; shape is bizarre and abnormal
- **P wave**: Visibility of P wave depends on the timing of the PVC; may be absent (hidden in the QRS or T wave) or in front of the QRS. If the P wave follows the QRS, the shape of the P wave may be different.
- **PR interval**: If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds.
- **P: QRS ratio**: 0:1; 1:1

The patient may feel nothing or may say that the heart “skipped a beat.” The effect of a PVC depends on its timing in the cardiac cycle and how much blood was in the ventricles when they contracted. Initial treatment is aimed at correcting the cause, if possible. Lidocaine (Xylocaine) is the medication most com-
plicated in quadrigeminy in lead V1. Note regular PP interval.

- **Ventricular Tachycardia.** Ventricular tachycardia (VT) is defined as three or more PVCs in a row, occurring at a rate exceeding 100 beats per minute. The causes are similar to those for PVC. VT is usually associated with coronary artery disease and may precede ventricular fibrillation. VT is an emergency because the patient is usually (although not always) unresponsive and pulseless. VT has the following characteristics (Fig. 27-16):
  - **Ventricular and atrial rate**: Ventricular rate is 100 to 200 beats per minute; atrial rate depends on the underlying rhythm (eg, sinus rhythm)
  - **Ventricular and atrial rhythm**: Usually regular; atrial rhythm may also be regular.
  - **QRS shape and duration**: Duration is 0.12 seconds or more; bizarre, abnormal shape

VT is an emergency because the patient is usually (although not always) unresponsive and pulseless. VT has the following characteristics (Fig. 27-16):

- **Ventricular and atrial rate**: Ventricular rate is 100 to 200 beats per minute; atrial rate depends on the underlying rhythm (eg, sinus rhythm)
- **Ventricular and atrial rhythm**: Usually regular; atrial rhythm may also be regular.
- **QRS shape and duration**: Duration is 0.12 seconds or more; bizarre, abnormal shape

**P wave**: Very difficult to detect, so atrial rate and rhythm may be indeterminable

**PR interval**: Very irregular, if P waves seen.

**P: QRS ratio**: Difficult to determine, but if P waves are apparent, there are usually more QRS complexes than P waves.

The patient’s tolerance or lack of tolerance for this rapid rhythm depends on the ventricular rate and underlying disease. If the patient is stable, continuing the assessment, especially obtaining a 12-lead ECG, may be the only action necessary. Cardioversion may be the treatment of choice, especially if the patient is unstable. Several factors determine the initial medication used for treatment, including the following: identifying the rhythm as monomorphic (having a consistent QRS shape and rate) or polymorphic (having varying QRS shapes and rates); determining the existence of a prolonged QT interval before the initiation of VT; and ascertaining the patient’s heart function (normal or decreased). VT in a patient who is unconscious and without a pulse is treated in the same manner as ventricular fibrillation: immediate defibrillation is the action of choice.

**Ventricular Fibrillation.** Ventricular fibrillation is a rapid but disorganized ventricular rhythm that causes ineffective quivering of the ventricles. There is no atrial activity seen on the ECG. Causes of ventricular fibrillation are the same as for VT; it may also result from untreated or unsuccessfully treated VT. Other causes include electrical shock and Brugada syndrome, in which the patient (frequently of Asian descent) has a structurally normal heart, few or no risk factors for coronary artery disease, and a family history of sudden cardiac death. Ventricular fibrillation has the following characteristics (Fig. 27-17):

- **Ventricular rate**: Greater than 300 per minute
- **Ventricular rhythm**: Extremely irregular, without specific pattern
- **QRS shape and duration**: Irregular, undulating waves without recognizable QRS complexes

This dysrhythmia is always characterized by the absence of an audible heartbeat, a palpable pulse, and respirations. Because there is no coordinated cardiac activity, cardiac arrest and death are imminent if ventricular fibrillation is not corrected. Treatment of choice is immediate defibrillation and activation of emergency services. The importance of defibrillation is evident in one of the recent changes in basic life support (American Heart Association, 2000): placing a call for emergency assistance and calling for a defibrillator takes precedence over initiating cardiopulmonary resuscitation in the adult victim. Also, application of an automatic external defibrillator (AED) is included in basic life support classes. After defibrillation, eradicating causes and administering vaso-active and antiarrhythmic medications alternating with defibrill-
Idioventricular Rhythm. Idioventricular rhythm, also called ventricular escape rhythm, occurs when the impulse starts in the conduction system below the AV node. When the sinus node fails to create an impulse (eg, from increased vagal tone), or when the impulse is created but cannot be conducted through the AV node (eg, due to complete AV block), the Purkinje fibers automatically discharge an impulse. The following are the ECG criteria when idioventricular rhythm is not caused by AV block (Fig. 27-18):

- **Ventricular rate:** Ranges between 20 and 40; if the rate exceeds 40, the rhythm is known as accelerated idioventricular rhythm (AIVR).
- **Ventricular rhythm:** Regular
- **QRS shape and duration:** Bizarre, abnormal shape; duration is 0.12 seconds or more

Idioventricular rhythm commonly causes the patient to lose consciousness and experience other signs and symptoms of reduced cardiac output. In such cases, the treatment is the same as for pulseless electrical activity if the patient is in cardiac arrest or for bradycardia if the patient is not in cardiac arrest. Interventions may include identifying the underlying cause, administering intravenous atropine and vasopressor medications, and initiating emergency transcutaneous pacing. In some cases, idioventricular rhythm may cause no symptoms of reduced cardiac output. However, bed rest is prescribed so as not to increase the cardiac workload.

**Ventricular Asystole.** Commonly called flatline, ventricular asystole (Fig. 27-19) is characterized by absent QRS complexes, although P waves may be apparent for a short duration in two different leads. There is no heartbeat, no palpable pulse, and no respiration. Without immediate treatment, ventricular asystole is fatal. Cardiopulmonary resuscitation and emergency services are necessary to keep the patient alive. The guidelines for advanced cardiac life support (American Heart Association, 2000) state that the key to successful treatment is rapid assessment to identify a possible cause, which may be hypoxia, acidosis, severe electrolyte imbalance, drug overdose, or hypothermia. Intubation and establishment of intravenous access are the first recommended actions. Transcutaneous pacing may be attempted. A bolus of intravenous epinephrine should be administered and repeated at 3- to 5-minute intervals, followed by 1-mg boluses of atropine at 3- to 5-minute intervals. Because of the poor prognosis associated with asystole, if the patient does not respond to these actions and others aimed at correcting underlying causes, resuscitation efforts are usually ended (“the code is called”) unless special circumstances (eg, hypothermia) exist.

**CONDUCTION ABNORMALITIES**

When assessing the rhythm strip, the nurse takes care first to identify the underlying rhythm (eg, sinus rhythm, sinus arrhythmia). Then the PR interval is assessed for the possibility of an AV block. AV blocks occur when the conduction of the impulse through the AV nodal area is decreased or stopped. These blocks can be caused by medications (eg, digitalis, calcium channel blockers, beta-blockers), myocardial ischemia and infarction, valvular disorders, or myocarditis. If the AV block is caused by increased vagal tone (eg, suctioning, pressure above the eyes or on large vessels, anal stimulation), it is commonly accompanied by sinus bradycardia.

The clinical signs and symptoms of a heart block vary with the resulting ventricular rate and the severity of any underlying dis-
ease processes. Whereas first-degree AV block rarely causes any hemodynamic effect, the other blocks may result in decreased heart rate, causing a decrease in perfusion to vital organs, such as the brain, heart, kidneys, lungs, and skin. A patient with third-degree AV block caused by digitalis toxicity may be stable; another patient with the same rhythm caused by acute MI may be unstable. Health care providers must always keep in mind the need to treat the patient, not the rhythm. The treatment is based on the hemodynamic effect of the rhythm.

First-Degree Atrioventricular Block. First-degree heart block occurs when all the atrial impulses are conducted through the AV node into the ventricles at a rate slower than normal. This conduction disorder has the following characteristics (Fig. 27-20):

- **Ventricular and atrial rate:** Depends on the underlying rhythm
- **Ventricular and atrial rhythm:** Depends on the underlying rhythm
- **QRS shape and duration:** Usually normal, but may be abnormal
- **P wave:** In front of the QRS complex; shows sinus rhythm, regular shape
- **PR interval:** Greater than 0.20 seconds; PR interval measurement is constant.
- **P: QRS ratio:** 1:1

Second-Degree Atrioventricular Block, Type I. Second-degree, type I heart block occurs when all but one of the atrial impulses are conducted through the AV node into the ventricles. Each atrial impulse takes a longer time for conduction than the one before, until one impulse is fully blocked. Because the AV node is not depolarized by the blocked atrial impulse, the AV node has time to fully repolarize, so that the next atrial impulse can be conducted within the shortest amount of time. Second-degree AV block, type I has the following characteristics (Fig. 27-21):

- **Ventricular and atrial rate:** Depends on the underlying rhythm
- **Ventricular and atrial rhythm:** The PP interval is regular if the patient has an underlying normal sinus rhythm; the RR interval characteristically reflects a pattern of change. Starting from the RR that is the longest, the RR interval gradually shortens until there is another long RR interval.
- **QRS shape and duration:** Usually normal, but may be abnormal
- **P wave:** In front of the QRS complex; shape depends on underlying rhythm
- **PR interval:** PR interval becomes longer with each succeeding ECG complex until there is a P wave not followed by a QRS. The changes in the PR interval are repeated between each “dropped” QRS, creating a pattern in the irregular PR interval measurements.
- **P: QRS ratio:** 3:2, 4:3, 5:4, and so forth

Second-Degree Atrioventricular Block, Type II. Second-degree, type II heart block occurs when only some of the atrial impulses are conducted through the AV node into the ventricles. Second-degree AV block, type II has the following characteristics (Fig. 27-22):

- **Ventricular and atrial rate:** Depends on the underlying rhythm
- **Ventricular and atrial rhythm:** The PP interval is regular if the patient has an underlying normal sinus rhythm. The RR

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**FIGURE 27-18** Idioventricular rhythm in lead V1.

**FIGURE 27-19** Asystole. (Always check two different leads to confirm rhythm.)
**FIGURE 27-20** Sinus rhythm with first-degree AV block in lead II.

**FIGURE 27-21** Sinus rhythm with second-degree AV block, type I in lead II. Note progressively longer PR durations until there is a nonconducted P wave, indicated by the asterisk (*).

**FIGURE 27-22** Sinus rhythm with second-degree AV block, type II in lead V₁; note constant PR interval.
interval is usually regular but may be irregular, depending on the P: QRS ratio.
QRS shape and duration: Usually abnormal, but may be normal
P wave: In front of the QRS complex; shape depends on underlying rhythm.
PR interval: PR interval is constant for those P waves just before QRS complexes.
P: QRS ratio: 2:1, 3:1, 4:1, 5:1, and so forth

Third-Degree Atrioventricular Block. Third-degree heart block occurs when no atrial impulse is conducted through the AV node into the ventricles. In third-degree heart block, two impulses stimulate the heart: one stimulates the ventricles (eg, junctional or ventricular escape rhythm), represented by the QRS complex, and one stimulates the atria (eg, sinus rhythm, atrial fibrillation), represented by the P wave. P waves may be seen, but the atrial electrical activity is not conducted down into the ventricles to cause the QRS complex, the ventricular electrical activity. This is called AV dissociation. Complete block (third-degree AV block) has the following characteristics (Fig. 27-23):

Ventricular and atrial rate: Depends on the escape and underlying atrial rhythm.
Ventricular and atrial rhythm: The PP interval is regular and the RR interval is regular; however, the PP interval is not equal to the RR interval.
QRS shape and duration: Depends on the escape rhythm; in junctional escape, QRS shape and duration are usually normal, and in ventricular escape, QRS shape and duration are usually abnormal.
P wave: Depends on underlying rhythm
PR interval: Very irregular
P: QRS ratio: More P waves than QRS complexes

Based on the cause of the AV block and the stability of the patient, treatment is directed toward increasing the heart rate to maintain a normal cardiac output. If the patient is stable and has no symptoms, no treatment is indicated other than decreasing or eradicating the cause (eg, withholding the medication or treatment). If the patient is short of breath, complains of chest pain or lightheadedness, or has low blood pressure, an intravenous bolus of atropine is the initial treatment of choice. If the patient does not respond to atropine or has an acute MI, transcutaneous pacing should be started. A permanent pacemaker may be necessary if the block persists.

NURSING PROCESS: THE PATIENT WITH A DYSRHYTHMIA

Assessment

Major areas of assessment include possible causes of the dysrhythmia and the dysrhythmia’s effect on the heart’s ability to pump an adequate blood volume. When cardiac output is reduced, the amount of oxygen reaching the tissues and vital organs is diminished. This diminished oxygenation produces the signs and symptoms associated with dysrhythmias. If these signs and symptoms are severe or if they occur frequently, the patient may experience significant distress and disruption of daily life.

A health history is obtained to identify any previous occurrences of decreased cardiac output, such as syncope (fainting), lightheadedness, dizziness, fatigue, chest discomfort, and palpitations. Coexisting conditions that could be a possible cause of the dysrhythmia (eg, heart disease, chronic obstructive pulmonary disease) may also be identified. All medications, prescribed and over-the-counter (including herbs and nutritional supplements), are reviewed. Some medications (eg, digoxin) can cause dysrhythmias. A thorough psychosocial assessment is performed to identify the possible effects of the dysrhythmia and to determine whether anxiety is a significant contributing factor.

The nurse conducts a physical assessment to confirm the data obtained from the history and to observe for signs of diminished cardiac output during the dysrhythmic event, especially changes in level of consciousness. The nurse directs attention to the skin, which may be pale and cool. Signs of fluid retention, such as neck vein distention and crackles and wheezes auscultated in the lungs, may be detected. The rate and rhythm of apical and peripheral pulses are also assessed, and any pulse deficit is noted. The nurse auscultates for extra heart sounds (especially S3 and S4) and for heart murmurs, measures blood pressure, and determines pulse pressures. A declining pulse pressure indicates reduced cardiac output. Just one assessment may not disclose significant changes in cardiac output; therefore, the nurse compares multiple assessment findings over time, especially those that occur with and without the dysrhythmia.

Diagnosis

NURSING DIAGNOSES

Based on assessment data, major nursing diagnoses of the patient may include:
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- Decreased cardiac output
- Anxiety related to fear of the unknown
- Deficient knowledge about the dysrhythmia and its treatment

**COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS**
In addition to cardiac arrest, a potential complication that may develop over time is heart failure. Another potential complication, especially with atrial fibrillation, is a thromboembolic event. If the dysrhythmia necessitates treatment with medication, the beneficial and detrimental effects must be assessed.

**Planning and Goals**
The major goals for the patient may include eradicating or decreasing the incidence of the dysrhythmia (by decreasing contributory factors) to maintain cardiac output, minimizing anxiety, and acquiring knowledge about the dysrhythmia and its treatment.

**Nursing Interventions**

**MONITORING AND MANAGING THE DYSRHYTHMIA**
The nurse regularly evaluates blood pressure, pulse rate and rhythm, rate and depth of respirations, and breath sounds to determine the dysrhythmia’s hemodynamic effect. The nurse also asks patients about episodes of lightheadedness, dizziness, or fainting as part of the ongoing assessment. If a patient with a dysrhythmia is hospitalized, the nurse may obtain a 12-lead ECG, continuously monitor the patient, and analyze rhythm strips to track the dysrhythmia.

Control of the incidence or the effect of the dysrhythmia, or both, is often achieved by the use of antiarrhythmic medications. The nurse assesses and observes for the beneficial and adverse effects of each of the medications. The nurse also manages medication administration carefully so that a constant serum blood level of the medication is maintained at all times.

In addition to medication, the nurse assesses for factors that contribute to the dysrhythmia (e.g., caffeine, stress, nonadherence to the medication regimen) and assists the patient in developing a plan to make lifestyle changes that eliminate or reduce these factors.

**MINIMIZING ANXIETY**
When the patient experiences episodes of dysrhythmia, the nurse maintains a calm and reassuring attitude. This demeanor fosters a trusting relationship with the patient and assists in reducing anxiety (reducing the sympathetic response). Successes are emphasized with the patient to promote a sense of confidence in living with a dysrhythmia. For example, if a patient is experiencing episodes of dysrhythmia and a medication is administered that begins to reduce the incidence of the dysrhythmia, the nurse communicates that information to the patient. The nursing goal is to maximize the patient’s control and to make the unknown less threatening.

**PROMOTING HOME AND COMMUNITY-BASED CARE**

**Teaching Patients Self-Care**
When teaching patients about dysrhythmias, the nurse presents the information in terms that are understandable and in a manner that is not frightening or threatening. The nurse explains the importance of maintaining therapeutic serum levels of anti-arrhythmic medications so that the patient understands why medications should be taken regularly each day. In addition, the relationship between a dysrhythmia and cardiac output is explained so that the patient understands the rationale for the medical regimen. If the patient has a potentially lethal dysrhythmia, it is also important to establish with the patient and family a plan of action to take in case of an emergency. This allows the patient and family to feel in control and prepared for possible events.

A referral for home care usually is not necessary for the patient with a dysrhythmia unless the patient is hemodynamically unstable and has significant symptoms of decreased cardiac output. Home care is also warranted if the patient has significant comorbidities, socioeconomic issues, or limited self-management skills that could potentiate the risk for nonadherence to the therapeutic regimen.

**Evaluation**

**EXPECTED PATIENT OUTCOMES**
Expected patient outcomes may include:

1. Maintains cardiac output
   a. Demonstrates heart rate, blood pressure, respiratory rate, and level of consciousness within normal ranges
2. Experiences reduced anxiety
   a. Expresses a positive attitude about living with the dysrhythmia
   b. Expresses confidence in ability to take appropriate actions in an emergency
3. Expresses understanding of the dysrhythmia and its treatment
   a. Explains the dysrhythmia and its effects
   b. Describes the medication regimen and its rationale
   c. Explains the need for therapeutic serum level of the medication
   d. Describes a plan to eradicate or limit factors that contribute to the occurrence of the dysrhythmia
   e. States actions to take in the event of an emergency

**Adjunctive Modalities and Management**

Dysrhythmia treatments depend on whether the disorder is acute or chronic as well as on the cause of the dysrhythmia and its actual or potential hemodynamic effects.

Acute dysrhythmias may be treated with medications or with external electrical therapy. Many antiarrhythmic medications are used to treat atrial and ventricular tachydysrhythmias. These medications are summarized in Table 27-1. The choice of medication depends on the specific dysrhythmia, presence of cardiac failure and other diseases, and the patient’s response to previous treatment. The nurse is responsible for monitoring and documenting the patient’s responses to the medication and for making sure that the patient has the knowledge and ability to manage the medication regimen.

A referral for home care usually is not necessary for the patient with a dysrhythmia unless the patient is hemodynamically unstable and has significant symptoms of decreased cardiac output. Home care is also warranted if the patient has significant comorbidities, socioeconomic issues, or limited self-management skills that could potentiate the risk for nonadherence to the therapeutic regimen.

If medications alone are ineffective in eradicating or decreasing the dysrhythmia, certain adjunctive mechanical therapies are available. The most common are pacemakers for bradyarrhythmias and tachycardias, elective cardioversion and defibrillation for acute tachyarrhythmias, and implantable devices for chronic tachyarrhythmia. Surgical treatments, although less common, are also available.
**PACEMAKER THERAPY**

A pacemaker is an electronic device that provides electrical stimuli to the heart muscle. Pacemakers are usually used when a patient has a slower-than-normal impulse formation or a conduction disturbance that causes symptoms. They may also be used to control some tachyarrhythmias that do not respond to medication therapy. Biventricular (both ventricles) pacing may be used to treat advanced heart failure that does not respond to medication therapy.

Pacemakers can be permanent or temporary. Permanent pacemakers are used most commonly for irreversible complete heart block. Temporary pacemakers are used (eg, after MI, after open heart surgery) to support patients until they improve or receive a permanent pacemaker.

**Pacemaker Design and Types**

Pacemakers consist of two components: an electronic pulse generator and pacemaker electrodes, which are located on leads or wires. The generator contains the circuitry and batteries that generate the rate (measured in beats per minute) and the strength (measured in milliamperes [mA]) of the electrical stimulus delivered to the heart. The pacemaker electrodes convey the heart’s electrical activity through a lead to the generator; the generator’s electrical response to the information received is then transmitted to the heart.

Leads can be threaded through a major vein into the right ventricle (endocardial leads), or they can be lightly sutured onto the outside of the heart and brought through the chest wall during open heart surgery (epicardial wires). The epicardial wires are always temporary and are removed by a gentle tug within a few days after surgery. The endocardial leads may be temporarily placed with catheters through the femoral, antecubital, brachial, or jugular vein (transvenous wires), usually guided by fluoroscopy. The endocardial and epicardial wires are connected to a temporary generator, which is about the size of a small paperback book. The energy source for a temporary generator is a common household battery; monitoring for pacemaker malfunctioning and battery failure is a nursing responsibility. This type of pacemaker therapy necessitates hospitalization of the patient.

The endocardial leads also may be placed permanently, usually through the external jugular vein, and connected to a permanent generator, which is usually implanted underneath the skin in a subcutaneous pocket in the pectoral region or below the clavicle (Fig. 27-24). Sometimes an abdominal site is selected. This procedure is usually performed in a cardiac catheterization laboratory with the patient receiving a local anesthetic. Permanent pacemaker generators are insulated to protect against body moisture and warmth. There are several different energy sources for permanent generators: mercury-zinc batteries (which last 3 to 4 years), lithium cell units (up to 10 years), and nuclear-powered sources such as plutonium 238 (up to 20 years). Some of the batteries are rechargeable. If the battery is not rechargeable and failure is impending, the old generator is removed and the new one is connected to the existing leads and reimplanted in the already existing subcutaneous pocket. This procedure is usually performed with the patient receiving a local anesthetic. Hospitalization of the patient is needed for implantation or battery replacement.

If a patient suddenly develops a bradycardia, emergency pacing may be started with transcutaneous pacing, which most defibrillators are now equipped to perform. AEDs are not able to do transcutaneous pacing (see later discussion). Large pacing ECG electrodes (sometimes the same conductive pads that are used for cardioversion and defibrillation) are placed on the patient’s chest and back. The electrodes are connected to the defibrillator, which is the temporary pacemaker generator (Fig. 27-25). Because the impulse must travel through the patient’s skin and tissue before reaching the heart, transcutaneous pacing can cause significant discomfort and is intended to be used only in emergencies. This type of pacing necessitates hospitalization. If the patient is alert, the use of sedation and analgesia should be discussed with the physician.

**Pacemaker Generator Functions**

Because of the sophistication and wide use of pacemakers, a universal code has been adopted to provide a means of safe communication about their function. The coding is referred to as the NASPE-BPEG code because it is sanctioned by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group. The complete code consists of five letters, but only the first three are commonly used.

The first letter of the code identifies the chamber or chambers being paced—that is, the chamber containing a pacing electrode. The letter characters for this code are A (atrium), V (ventricle), or D (dual, meaning both A and V).

The second letter describes the chamber or chambers being sensed by the pacemaker generator. Information from the electrode within the chamber is sent to the generator for interpretation and action by the generator. The possible letter characters are A (atrium), V (ventricle), D (dual), and O (indicating that the sensing function is turned off).

The third letter of the code describes the type of response by the pacemaker to what is sensed. The possible letter characters used to describe this response are I (inhibited), T (triggered), D (dual, inhibited and triggered), and O (none). Inhibited response means that the response of the pacemaker is controlled by the activity of the patient’s heart; that is, the pacemaker will not func-
Complications of Pacemaker Use

Complications associated with pacemakers relate to their presence within the body, and improper functioning. The following complications may arise from a pacemaker:

- Local infection at the entry site of the leads for temporary pacing, or at the subcutaneous site for permanent generator placement
- Bleeding and hematoma at the lead entry sites for temporary pacing, or at the subcutaneous site for permanent generator placement
- Hemothorax from puncture of the subclavian vein or internal mammary artery
- Ventricular ectopy and tachycardia from irritation of the ventricular wall by the endocardial electrode
- Movement or dislocation of the lead placed transvenously (perforation of the myocardium)
- Phrenic nerve, diaphragmatic (hiccuping may be a sign of this), or skeletal muscle stimulation if the lead is dislocated or if the delivered energy (mA) is set high
- Rarely, cardiac tamponade from bleeding resulting from removal of epicardial wires used for temporary pacing
In the initial hours after a temporary or permanent pacemaker is inserted, the most common complication is dislodgment of the pacing electrode. Minimizing patient activity can help to prevent this complication. If a temporary electrode is in place, the extremity through which the catheter has been advanced is immobilized. With a permanent pacemaker, the patient is instructed initially to restrict activity on the side of the implantation.

The ECG is monitored very carefully to detect pacemaker malfunction. Improper pacemaker function, which can arise from failure in one or more components of the pacing system, is outlined in Table 27-2. The following data should be noted on the patient’s record: model of pacemaker, type of generator, date and time of insertion, location of pulse generator, stimulation threshold, pacer settings (e.g., rate, energy output [mA], and duration between atrial and ventricular impulses [AV delay]). This information is important for identifying normal pacemaker function and diagnosing pacemaker malfunction.

A patient experiencing pacemaker malfunction may develop signs and symptoms of decreased cardiac output. The degree to which these symptoms become apparent depends on the severity of the malfunction, the patient’s level of dependency on the pacemaker, and the patient’s underlying condition. Pacemaker malfunction is diagnosed by analyzing the ECG. Manipulating the electrodes, changing the generator’s settings, or replacing the pacemaker generator or leads (or both) may be necessary.

Inhibition of permanent pacemakers can occur with exposure to strong electromagnetic fields (electromagnetic interference). However, recent pacemaker technology allows patients to safely use most household electronic appliances and devices (e.g., microwave ovens, electric tools) as long as they are not held close to the pacemaker generator. Gas-powered engines should be turned off before working on them. Objects that contain magnets (e.g., the earpiece of a standard phone; large stereo speakers; magnet therapy products such as mattresses, jewelry, and wraps) should not be near the generator for longer than a few seconds. Patients are advised to use digital cellular phones on the side opposite the pacemaker generator. Large electromagnetic fields, such as those produced by magnetic resonance imaging (MRI), radio and TV transmitter towers and lines, transmission power lines (these are different from the distribution lines that bring electricity into a home), and electrical substations may cause electromagnetic interference. Patients should be cautioned to avoid such situations or to simply move farther away from the area if they experience dizziness or a feeling of rapid or irregular heartbeats (palpitations). Welding and use of a chainsaw should be avoided. If such tools are used, precautionary steps such as limiting the welding current to a 60- to 130-ampere range or using electric rather than gasoline-powered chain saws are advised.

The metal of the pacemaker generator may trigger some store and airport security alarms, but these alarm systems will not interfere with the pacemaker function. However, the handheld screening devices used in airports may interfere with the pacemaker. Patients should be advised to request a hand search instead of the handheld screening device. Patients also should be instructed to wear or carry medical identification to alert personnel to the presence of the pacemaker.
Pacemaker Surveillance

Pacemaker clinics have been established to monitor patients and to test pulse generators for impending pacemaker battery failure. Several other factors, such as lead fracture, muscle inhibition, and insulation disruption, are assessed depending on the type of pacemaker and the equipment available. If indicated, the pacemaker is turned off for a few seconds, using a magnet or a programmer, while the ECG is recorded to assess the patient’s underlying cardiac rhythm.

Another follow-up method is transtelephonic transmission of the generator’s pulse rate. Special equipment is used to transmit information about the patient’s pacemaker over the telephone to a receiving system at a pacemaker clinic. The information is converted into tones, which equipment at the clinic converts to an electronic signal and records on an ECG strip. The pacemaker rate and other data concerning pacemaker function are obtained and evaluated by a cardiologist. This simplifies the diagnosis of a failing generator, reassures the patient, and improves management when the patient is physically remote from pacemaker testing facilities.

NURSING PROCESS: THE PATIENT WITH A PACEMAKER

Assessment

After a temporary or a permanent pacemaker is inserted, the patient’s heart rate and rhythm are monitored by ECG. The pacemaker’s settings are noted and compared with the ECG recordings to assess pacemaker function. Pacemaker malfunction is detected by examining the pacemaker spike and its relationship to the surrounding ECG complexes (Fig. 27-28). In addition, cardiac output and hemodynamic stability are assessed to identify the patient’s response to pacing and the adequacy of pacing. The appearance or increasing frequency of dysrhythmia is observed and reported to the physician.

The incision site where the pulse generator was implanted (or the entry site for the pacing electrode, if the pacemaker is a temporary transvenous pacemaker) is observed for bleeding, hematoma formation, or infection, which may be evidenced by swelling, unusual tenderness, unusual drainage, and increased heat. The patient may complain of continuous throbbing or pain. These symptoms are reported to the physician.

The patient with a temporary pacemaker is also assessed for electrical interference and the development of microshock. The nurse observes for potential sources of electrical hazards. All electrical equipment used in the vicinity of the patient should be grounded. Improperly grounded equipment can generate leakage of current capable of producing ventricular fibrillation. Exposed wires must be carefully covered with nonconductive material to prevent accidental ventricular fibrillation from stray currents. The nurse, working with a biomedical engineer or electrician, should make certain that the patient is in an electrically safe environment.

Patients, especially those receiving a permanent pacemaker, should be assessed for anxiety. In addition, for those receiving permanent pacemakers, the level of knowledge and learning needs of the patient and the family and the history of adherence to the therapeutic regimen should be identified.

Diagnosis

NURSING DIAGNOSES

Based on assessment data, major nursing diagnoses of the patient may include the following:

- Risk for infection related to pacemaker lead or generator insertion
- Risk for ineffective coping
- Deficient knowledge regarding self-care program

**Table 27-2 • Assessing Pacemaker Malfunction**

<table>
<thead>
<tr>
<th>PROBLEM</th>
<th>POSSIBLE CAUSE</th>
<th>INTERVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of capture—complex does not follow pacing spike</td>
<td>Inadequate stimulus, catheter malposition, battery depletion, electronic insulation break</td>
<td>Check security of all connections; increase milliamperage. Reposition extremity; turn patient to left side. Change battery. Change generator.</td>
</tr>
<tr>
<td>Undersensing—pacing spike occurs at preset interval despite patient’s intrinsic rhythm</td>
<td>Sensitivity too high, electrical interference (eg, by a magnet), faulty generator</td>
<td>Decrease sensitivity. Eliminate interference. Replace generator.</td>
</tr>
<tr>
<td>Oversensing—loss of pacing artifact; pacing does not occur at preset interval despite lack of intrinsic rhythm</td>
<td>Sensitivity too low, electrical interference, battery depletion</td>
<td>Increase sensitivity. Eliminate interference. Change battery.</td>
</tr>
<tr>
<td>Loss of pacing—Total absence of pacing spikes</td>
<td>Battery depletion, loose or disconnected wires, perforation</td>
<td>Change battery. Check security of all connections. Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Call physician.</td>
</tr>
<tr>
<td>Change in pacing QRS shape</td>
<td>Septal perforation</td>
<td>Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Call physician.</td>
</tr>
<tr>
<td>Rhythmic diaphragmatic or chest wall twitching or hiccuping</td>
<td>Output too high, myocardial wall perforation</td>
<td>Decrease milliamperage. Turn pacer off. Call physician at once. Monitor closely for decreased cardiac output.</td>
</tr>
</tbody>
</table>
COLLABORATIVE PROBLEMS/ POTENTIAL COMPLICATIONS

Based on the assessment findings, potential complications that may develop include decreased cardiac output related to pacemaker malfunction.

Planning and Goals

The major goals for the patient may include absence of infection, adherence to a self-care program, effective coping, and maintenance of pacemaker function.

Nursing Interventions

PREVENTING INFECTION

The nurse changes the dressing regularly and inspects the insertion site for redness, swelling, soreness, or any unusual drainage. An increase in temperature should be reported to the physician. Changes in wound appearance are also reported to the physician.

PROMOTING EFFECTIVE COPING

The patient treated with a pacemaker experiences not only lifestyle and physical changes but also emotional changes. At different times during the healing process, the patient may feel angry, depressed, fearful, anxious, or a combination of these emotions. Although each patient uses individual coping strategies (eg, humor, prayer, communication with a significant other) to manage emotional distress, some strategies may work better than others. Signs that may indicate ineffective coping include social isolation, increased or prolonged irritability or depression, and difficulty in relationships.

To promote effective coping strategies, the nurse must recognize the patient’s emotional state and assist the patient to explore his or her feelings. The nurse may help the patient to identify perceived changes (eg, loss of ability to participate in contact sports), the emotional response to the change (eg, anger), and how the patient responded to that emotion (eg, quickly became angry when talking with spouse). The nurse reassures the patient that the responses are normal, then assists the patient to identify realistic goals (eg, develop interest in another activity) and to develop a plan to attain those goals. The nurse may also teach the patient easy-to-use stress reduction techniques (eg, deep-breathing exercises) to facilitate coping. Education (Chart 27-3) may assist a patient to cope with changes that occur with pacemaker treatment.

PROMOTING HOME AND COMMUNITY-BASED CARE

Teaching Patients Self-Care

After pacemaker insertion, the patient’s hospital stay may be less than 1 day, and follow-up in an outpatient clinic or office is common. The patient’s anxiety and feelings of vulnerability may interfere with the ability to learn information provided. Nurses often need to include home caregivers in the teaching and provide printed materials for use by the patient and caregiver. Priorities for learning are established with the patient and caregiver. Teaching may include the importance of periodic pacemaker monitoring, promoting safety, avoiding infection, and sources of electromagnetic interference (see Chart 27-3).

Evaluation

EXPECTED PATIENT OUTCOMES

Expected patient outcomes may include:

1. Remains free of infection
   a. Has normal temperature
   b. Has white blood cell count within normal range (5,000 to 10,000/mm$^3$)
c. Exhibits no redness or swelling of pacemaker insertion site
2. Adheres to a self-care program
   a. Responds appropriately when queried about the signs and symptoms of infection
   b. Identifies when to seek medical attention (as demonstrated in responses to signs and symptoms)
   c. Adheres to monitoring schedule
   d. Describes appropriate methods to avoid electromagnetic interference
3. Maintains pacemaker function (see Chart 27-3)
   a. Measures and records pulse rate at regular intervals
   b. Experiences no abrupt changes in pulse rate or rhythm

**CARDIOVERSION AND DEFIBRILLATION**

Cardioversion and defibrillation are treatments for tachydysrhythmias. They are used to deliver an electrical current to depolarize a critical mass of myocardial cells. When the cells repolarize, the sinus node is usually able to recapture its role as the heart’s pacemaker. One major difference between cardioversion and defibrillation has to do with the timing of the delivery of electrical current. Another major difference concerns the circumstance: defibrillation is usually performed as an emergency treatment, whereas cardioversion is usually, but not always, a planned procedure.

Electrical current may be delivered through paddles or conductor pads. Both paddles may be placed on the front of the chest (Fig. 27-29), which is the standard paddle placement, or one paddle may be placed on the front of the chest and the other connected to an adapter with a long handle and placed under the patient’s back, which is called an anteroposterior placement (Fig. 27-30).
Instead of paddles, defibrillator multifunction conductor pads may be used (Fig. 27-31). The pads, which contain a conductive medium, are placed in the same position as the paddles. They are connected to the defibrillator and allow for hands-off defibrillation. This method reduces the risks of touching the patient during the procedure and increases electrical safety. AEDs use this type of delivery for the electrical current.

Whether using pads or paddles, the nurse must observe two safety measures. First, maintain good contact between the pads or paddles (with a conductive medium) and the patient’s skin to prevent electrical current from leaking into the air (arching) when the defibrillator is discharged. Second, ensure that no one is in contact with the patient or with anything that is touching the patient when the defibrillator is discharged, to minimize the chance that electrical current will be conducted to anyone other than the patient.

When performing defibrillation or cardioversion, the nurse should remember these key points:

- Use multifunction conductor pads or paddles with a conducting agent between the paddles and the skin (the conducting agent is available as a sheet, gel, or paste).
- Place paddles or pads so that they do not touch the patient’s clothing or bed linen and are not near medication patches or direct oxygen flow.
- If cardioverting, ensure that the monitor leads are attached to the patient and that the defibrillator is in sync mode. If defibrillating, ensure that the defibrillator is not in sync mode (most machines default to the “not-sync” mode).
- Do not charge the device until ready to shock; then keep thumbs and fingers off the discharge buttons until paddles or pads are on the chest and ready to deliver the electrical charge.
- Exert 20 to 25 pounds of pressure on the paddles to ensure good skin contact.
- Before pressing the discharge button, call “Clear!” three times: As “Clear” is called the first time, ensure that you are not touching the patient, bed or equipment; as “Clear” is called the second time, ensure that no one is touching the bed, the patient, or equipment, including the endotracheal tube or adjuncts; and as “Clear” is called the third time, perform a final visual check to ensure you and everyone else are clear of the patient and anything touching the patient.
- Record the delivered energy and the results (cardiac rhythm, pulse).
- After the event is complete, inspect the skin under the pads or paddles for burns; if any are detected, consult with the physician or a wound care nurse about treatment.

**Cardioversion**

Cardioversion involves the delivery of a “timed” electrical current to terminate a tachydysrhythmia. In cardioversion, the defibrillator is set to synchronize with the ECG on a cardiac monitor so that the electrical impulse discharges during ventricular depolarization (QRS complex). Because there may be a short delay until recognition of the QRS, the discharge buttons must be held down until the shock has been delivered. The synchronization prevents the discharge from occurring during the vulnerable period of repolarization (T wave), which could result in VT or ventricular fibrillation. When the synchronizer is on, no electrical current will be delivered if the defibrillator does not discern a QRS complex. Sometimes the lead and the electrodes must be changed for the monitor to recognize the patient’s QRS complex.

If the cardioversion is elective, anticoagulation for a few weeks before cardioversion may be indicated. Digoxin is usually withheld for 48 hours before cardioversion to ensure the resumption of sinus rhythm with normal conduction. The patient is instructed not to eat or drink for at least 8 hours before the procedure. Gel-covered paddles or conductor pads are positioned front and back (anteroposteriorly) for cardioversion. Before cardio-
conversion, the patient receives intravenous sedation as well as an analgesic medication or anesthesia. Respiration is then supported with supplemental oxygen delivered by a bag-mask-valve device with suction equipment readily available. Although patients rarely require intubation, equipment is nearby if it is needed. The amount of voltage used varies from 25 to 360 joules, depending on the defibrillator’s technology and the type of dysrhythmia. If ventricular fibrillation occurs after cardioversion, the defibrillator is used to defibrillate the patient (sync mode is not used). Indications of a successful response are conversion to sinus rhythm, adequate peripheral pulses, and adequate blood pressure. Because of the sedation, airway patency must be maintained and the patient’s state of consciousness assessed. Vital signs and oxygen saturation are monitored and recorded until the patient is stable and recovered from sedation and the effects of analgesic medications or anesthesia. ECG monitoring is required during and after cardioversion.

**Defibrillation**

Defibrillation is used in emergency situations as the treatment of choice for ventricular fibrillation and pulseless VT. Defibrillation depolarizes a critical mass of myocardial cells at once; when they repolarize, the sinus node usually recaptures its role as the pacemaker. The electrical voltage required to defibrillate the heart is usually greater than that required for cardioversion. If three defibrillations of increasing voltage have been unsuccessful, cardio-pulmonary resuscitation is initiated and advanced life support treatments are begun.

The use of epinephrine or vasopressin may make it easier to convert the dysrhythmia to a normal rhythm with defibrillation. These drugs may also increase cerebral and coronary artery blood flow. After the medication is administered and 1 minute of cardio-pulmonary resuscitation is performed, defibrillation is again administered. Antiarrhythmic medications such as amiodarone (Cordarone, Pacerone), lidocaine (Xylocaine), magnesium, or procainamide (Pronestyl) are given if ventricular dysrhythmia persists (see Table 27-1). This treatment continues until a stable rhythm resumes or until it is determined that the patient cannot be revived.

The first defibrillator, which was implanted in 1980 at Johns Hopkins University, simply defibrillated the heart. Today, however, several devices are available, and many are programmed for multiple treatments (Atlee & Bernstein, 2001). Each device offers a different delivery sequence, but all are capable of delivering high-energy (high-intensity) defibrillation to treat a tachycardia (atrial or ventricular). The device may deliver up to six shocks if necessary. Some ICDs can respond with antitachycardia pacing, in which the device delivers electrical impulses at a fast rate in an attempt to disrupt the tachycardia, by low-energy (low-intensity) cardioversion, by defibrillation, or all three (Atlee & Bernstein, 2001). Some also have pacemaker capability if the patient develops bradycardia, which sometimes occurs after treatment of the tachycardia. Usually the mode is VVI (V, paces the ventricle; V, senses ventricular activity; I, paces only if the ventricles do not depolarize) (Atlee & Bernstein, 2001). Some ICDs also deliver low-energy cardioversion, and some also treat atrial fibrillation (Bubien & Sanchez, 2001; Daoud et al., 2000). Which device is used and how it is programmed depends on the patient’s dysrythmia.

Complications are similar to those associated with pacemaker insertion. The primary complication associated with the ICD is surgery-related infection. There are a few complications associated with the technical aspects of the equipment, such as premature battery depletion and dislodged or fractured leads. Despite the possible complications, the consensus among clinicians is that the benefits of ICD therapy exceed the risks.

Nursing interventions for the patient with an ICD are provided throughout the preoperative, perioperative, and postoperative phases. In addition to providing the patient and family with explanations regarding implantation of the ICD in the preoperative phase, the nurse may need to manage acute episodes of life-threatening dysrhythmias. In the perioperative and postoperative phases, the nurse carefully observes the patient’s responses to the ICD and provides the patient and family with further teaching and explanations regarding implantation of the ICD in the preoperative phase. The nurse may need to manage acute episodes of life-threatening dysrhythmias. In the perioperative and postoperative phases, the nurse carefully observes the patient’s responses to the ICD and provides the patient and family with further teaching and explanations regarding implantation of the ICD in the preoperative phase.

**IMPLANTABLE CARDIOVERTER DEFIBRILLATOR**

The implantable cardioverter defibrillator (ICD) is a device that detects and terminates life-threatening episodes of VT or ventricular fibrillation in high-risk patients. Patients at high risk are those who have survived sudden cardiac death syndrome, usually caused by ventricular fibrillation, or have experienced symptomatic VT (syncope secondary to VT). In addition, an ICD may be indicated for patients who have survived an MI but are at high risk for cardiac arrest.

An ICD consists of a generator and at least one lead that can sense intrinsic electrical activity and deliver an electrical impulse. The device is usually implanted much like a pacemaker (Fig. 27-32). ICDs are designed to respond to two criteria: a rate that exceeds a predetermined level, and a change in the isoelectric line segments. When a dysrhythmia occurs, rate sensors take 5 to 10 seconds to sense the dysrhythmia. Then the device takes several seconds to charge and deliver the programmed charge through the lead to the heart. Battery life is about 5 years but varies depending on use of the ICD over time. The battery is checked during follow-up visits.

Antiarrhythmic medication usually is administered with this technology to minimize the occurrence of the tachydysrhythmia and to reduce the frequency of ICD discharge.
ELECTROPHYSIOLOGIC STUDIES

An electrophysiology (EP) study is used to evaluate and treat various dysrhythmias that have caused cardiac arrest or significant symptoms. It also is indicated for patients with symptoms that suggest a dysrhythmia that has gone undetected and undiagnosed by other methods. An EP study is used to:

- Identify the impulse formation and propagation through the cardiac electrical conduction system
- Assess the function or dysfunction of the SA and AV nodal areas
- Identify the location (called mapping) and mechanism dysrhythmogenic foci
- Assess the effectiveness of antiarrhythmic medications and devices for the patient with a dysrhythmia
- Treat certain dysrhythmias through the destruction of the causative cells (ablation)

An EP procedure is a type of cardiac catheterization that is performed in a specially equipped cardiac catheterization laboratory. The patient is awake but lightly sedated. Usually a catheter with multiple electrodes is inserted through the femoral vein, threaded through the inferior vena cava, and advanced into the heart. The electrodes are positioned within the heart at specific locations—for instance, in the right atrium near the sinus node, in the coronary sinus, near the tricuspid valve, and at the apex of the right ventricle. The number and placement of electrodes depend on the type of study being conducted. These electrodes allow the electrical signal to be recorded from within the heart (intracardiac).

The electrodes also allow the clinician to introduce a pacing stimulus to the intracardiac area at a precisely timed interval and rate, thereby stimulating the area (programmed stimulation). An area of the heart may be paced at a rate much faster than the normal rate of automaticity, the rate at which impulses are spon-
taneously formed (eg, in the sinus node). This allows the pacemaker to become an artificial focus of automaticity and to assume control (overdrive suppression). Then the pacemaker is stopped suddenly, and the time it takes for the sinus node to resume control is assessed. A prolonged time indicates dysfunction of the sinus node.

One of the main purposes of programmed stimulation is to assess the ability of the area surrounding the electrode to cause a reentry dysrhythmia. One or a series of premature impulses is delivered to an area in an attempt to cause the tachydysrhythmia. Because the precise location of the suspected area and the specific timing of the pacing needed are unknown, the electrophysiologist uses several different techniques to cause the dysrhythmia during the study. If the dysrhythmia can be reproduced by programmed stimulation, it is called inducible. Once a dysrhythmia is induced, a treatment plan is determined and implemented. If, on the follow-up EP study, the tachydysrhythmia cannot be induced, then the treatment is determined to be effective. Different medications may be administered and combined with electrical devices (pacemaker, ICD) to determine the most effective treatment to suppress the dysrhythmia.

Complications of an EP study are the same as those that can occur with cardiac catheterization. Because an artery is not always used, there is a lower incidence of vascular complications than with other catheterization procedures. Cardiac arrest may occur, but the incidence is low (less than 1%).

Patients who are to undergo an EP study may be anxious about the procedure and about its outcome. A detailed discussion involving the patient, the family, and the electrophysiologist usually occurs to ensure that the patient is able to give informed consent and to reduce anxiety about the procedure. Before the procedure, patients should receive instructions about the procedure and its usual duration, the environment where the procedure is performed, and what to expect. Although an EP study is not painful, it does cause discomfort and can be tiring. It may also cause feelings that were experienced when the dysrhythmia occurred in the past. In addition, patients also are taught what will be expected of them (eg, lying very still during the procedure, reporting symptoms or concerns).

Patients need to know that the dysrhythmia may occur during the procedure, but under very controlled circumstances. It often stops on its own; if it does not, treatment is given to restore the patient’s normal rhythm. During the procedure, patients benefit from a calm, reassuring approach.

Postprocedural care includes restriction of activity to promote hemostasis at the insertion site. To identify any complications and to ensure healing, the patient’s vital signs and the appearance of the insertion site are assessed frequently.

**CARDIAC CONDUCTION SURGERY**

Atrial tachycardias and ventricular tachycardias that do not respond to medications and are not suitable for antitachycardia pacing may be treated by methods other than medications and devices. Such methods include endocardial isolation, endocardial resection, and ablation. An ICD may be used with these surgical interventions.

**Endocardial Resection**

In endocardial resection, the origin of the dysrhythmia is identified, and that area of the endocardium is peeled away. No reconstruction or repair is necessary.

**Catheter Ablation Therapy**

Catheter ablation destroys specific cells that are the cause or central conduction method of a tachydysrhythmia. It is performed with or after an EP study. Usual indications for ablation are AV nodal reentry tachycardia, atrial fibrillation, or VT unresponsive to previous therapy (or for which the therapy produced significant side effects).

Ablation is also indicated to eliminate accessory AV pathways or bypass tracts that exist in the hearts of patients with preexcitation syndromes such as Wolff-Parkinson-White (WPW) syndrome. During normal embryonic development, all connections between the atrium and ventricles disappear, except for that between the AV node and the bundle of His. In some people, embryonic connections of normal heart muscle between the atrium and ventricles remain, providing an accessory pathway or a tract through which the electrical impulse can bypass the AV node. These pathways can be located in several different areas. If the patient develops atrial fibrillation, the impulse may be conducted into the ventricle at a rate of 300 times per minute or more, which can lead to ventricular fibrillation and sudden cardiac death. Preexcitation syndromes are identified by specific ECG findings. For example, in WPW syndrome there is a shortened PR interval, slurring (called a delta wave) of the initial QRS deflection, and prolonged QRS duration (Fig. 27-33).

Ablation may be accomplished by three different methods: radiofrequency ablation, cryoablation, or electrical ablation. The most often used method is radiofrequency, which involves placing a special catheter at or near the origin of the dysrhythmia. High-frequency, low-energy sound waves are passed through the catheter, causing thermal injury and cellular changes that result in localized destruction and scarring. The tissue damage is more specific to the dysrhythmic tissue, with less trauma to the surrounding cardiac tissue than occurs with cryoablation or electrical ablation.

Cryoablation involves placing a special probe, cooled to a temperature of −60°C (−76°F), on the endocardium at the site of the dysrhythmia’s origin for 2 minutes. The tissue freezes and is later replaced by scar tissue, eliminating the origin of the dysrhythmia.

In electrical ablation, a catheter is placed at or near the origin of the dysrhythmia, and one to four shocks of 100 to 300 joules are administered through the catheter directly to the endocardium and surrounding tissue. The cardiac tissue burns and scars, thus eliminating the source of the dysrhythmia.

During the ablation procedure, defibrillation pads, an automatic blood pressure cuff, and a pulse oximeter are used on the patient, and an indwelling urinary catheter is inserted. The patient is given light sedation. An EP study is performed and attempts to induce the dysrhythmia are made. The ablation catheter is placed at the origin of the dysrhythmia, and the ablation procedure is performed. Multiple ablations may be necessary. Successful ablation is achieved when the dysrhythmia can no longer be induced. The patient is monitored for another 30 to 60 minutes and then retested to ensure that the dysrhythmia will not recur.

Postprocedural care is similar to that for an EP study, except that the patient is monitored more closely, depending on the time needed for recovery from sedation.
Critical Thinking Exercises

1. You are caring for a 40-year-old male physician who had experienced a cardiac arrest at home, witnessed by his 9-year-old son and 15-year-old daughter. After having an ICD implanted, he appears sullen and withdrawn. On inquiry about how he feels, he replies, “I don’t know if this device is a blessing or a punishment!” How would you respond? What other factors are important to assess? Discuss the impact that his children may have on his perception of the device. How would you alter your plan of care to address this patient’s psychosocial concerns because he is a physician? How would the plan of care change if, instead of appearing sullen and withdrawn, he appeared irritable and confrontational?

2. Your patient is an active 80-year-old woman who has heart failure and chronic atrial fibrillation. She is taking an angiotensin-converting enzyme inhibitor, a beta-blocker, a diuretic, and digoxin. During your assessment, she tells you that she felt very dizzy this morning. How would you focus your assessment, and why? Identify some of the key assessment factors. What nursing interventions are needed? How would you modify your assessment and interventions if your patient also had chronic obstructive pulmonary disease and renal insufficiency?

REFERENCES AND SELECTED READINGS

Books and Pamphlets


**Journals**

*Asterisks indicate nursing research articles.*


**RESOURCES AND WEBSITES**


American College of Cardiology, 911 Old Georgetown Road, Bethesda, MD 20814; 800-253-4636; http://www.acc.org.

American Heart Association, National Center, 7272 Greenville Ave., Dallas, TX 75231; 1-800-242-8721; http://www.americanheart.org.

American Heart, Lung, Blood Institute, Health Information Center, National Institutes of Health, PO Box 30105, Bethesda, MD 20824; 301-592-8573; http://www.nhlbi.nih.gov.

National Institute on Aging, Building 31, Room 5C27, 31 Center Drive, MSC 2292, Bethesda, MD 20892; 301-496-1752; http://www.nia.nih.gov.

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