

# HEART RHYTHM DISORDERS

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## INTRODUCTION

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Heart rhythm disorders, called arrhythmias, pose one of the paradoxes of medicine. Almost anyone's heart will occasionally produce an extra beat or two, and the distressing symptoms that may accompany the extra beats, such as palpitations or dizziness, do not necessarily signal a serious health problem. Yet an undetected arrhythmia also may set off a chain of events leading to sudden death from cardiac arrest. In the United States, more than 300,000 deaths result each year from sudden cardiac arrest. When confronted with a patient who has an arrhythmia, a physician's task is to assess the risks and need for treatment, offer a course of treatment that will prevent adverse consequences, and relieve any discomfort. Because treatment with medication may have ill effects, a decision as to whether to treat at all or how to treat requires careful weighing of the disorder and the person in whom it occurs.

Abnormal heart rhythms fall into two general classes: excessively slow heart rates, known as bradyarrhythmias or bradycardias, and overly rapid heart rates, known as tachyarrhythmias or tachycardias. (See box, "Types of Arrhythmias.")

Extra or "skipped" heartbeats most often occur in hearts that are otherwise normal. Coronary artery disease, heart valve disease, heart muscle disease,

and other cardiac disorders also may underlie more serious arrhythmias, but the immediate cause for an abnormal heart rhythm is a malfunction in the heart's electrical system. Without its electrical conduction system, the heart would be a mass of muscle incapable of coordinated pumping. The layout and timing of the heart's circuitry provide an exquisite solution. (See Figure 16.1.) This circuitry, however, can also break down.

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## THE ELECTRICAL SYSTEM OF THE HEART

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The sinus node (SN), located at the top of the right atrium near the point where blood returns from the upper body, is the heart's pacemaker. Specialized cells in the sinus node send out electrical impulses that normally range between 60 and 100 per minute. As these impulses spread, they stimulate the muscle tissue of the left and right atria, causing contractions. The electrical impulses travel to the atrioventricular node (AV node), which is located in the septum (a wall of fibrous tissue that separates the two ventricles, the heart's major pumping chambers, from each other).

Electrical current moves faster than blood, so the atrioventricular node acts as a stop sign to delay the

## Types of Arrhythmias

### Slow heart rhythms

- Sinus bradycardia: heart rate slower than 60 beats per minute
- Sick sinus syndrome: failure of the sinus node to generate or conduct impulses properly
- Heart block: malfunction of the electrical conducting system between the atria and the ventricles

### Rapid heart rhythms

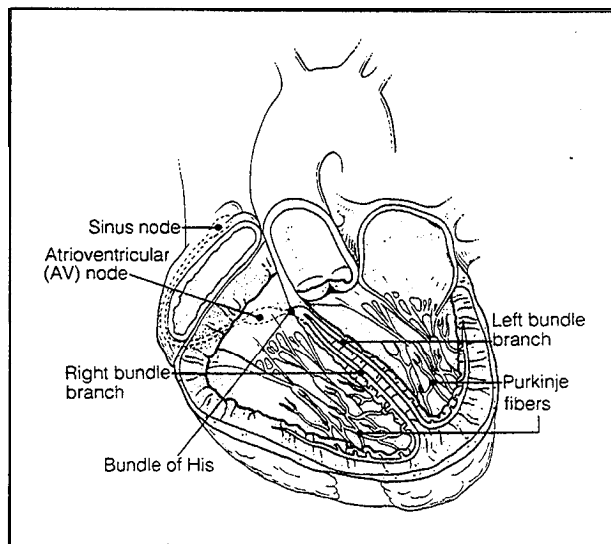
- Supraventricular tachycardias: abnormally rapid rhythms that arise in the atria or the atrioventricular node
- Atrial flutter: a "circus current," triggered by an extra or early beat, that travels in regular cycles around the atrium, pushing the atrial rate up to 250 to 350 beats per minute
- Atrial fibrillation: a chaos of uncoordinated beats, with the atrial rate at more than 350 beats per minute, that causes the atria to quiver
- Paroxysmal supraventricular tachycardia (PSVT): a supraventricular tachycardia that causes a sudden increase in the heart rate, to between 140 and 250 beats per minute
- Wolff-Parkinson-White syndrome: presence of abnormal conduction pathways that bypass the normal atrioventricular node; tends to result in recurrent tachycardias
- Premature ventricular contractions (PVCs): early or extra beats that commonly occur in normal hearts, but can cause problems in unhealthy hearts; usually safe but can cause ventricles to lapse into fatal ventricular fibrillation or ventricular tachycardia
- Ventricular tachycardias: abnormally rapid rhythms that arise in the ventricles; potentially more serious than supraventricular tachycardias
- Ventricular fibrillation: ventricles quivering and ceasing to pump blood effectively; death possible within three to four minutes if patient is not treated

impulses long enough for the blood pumped by the atria to fill the ventricles. Then the signal enters a "superhighway of conducting fibers, the His-Purkinje system, that branch left and right to direct the impulse first to the bottom and then up the sides of

the ventricles. The result is a smooth surge of muscular contraction in the ventricles that squeezes the blood up from the floors of the chambers. The blood is then forced through valves that lead to the major arteries. Thus, it begins its journey to the lungs or to the rest of the body.

Figure 16.1

This rendering of the heart's electrical conduction system shows electrical impulses traveling from the sinus node through electrical pathways to the atria, causing them to contract. The impulses travel to the atrioventricular (AV) node and then to the Bundle of His, the right and left bundles, and through the Purkinje fibers to the bottom and sides of the ventricles. The result is a smooth contraction from atria to ventricles that then forces blood up and out through the valves leading to the major arteries. (See Atlas 3B for four-color rendition.)



In order to adjust its pumping rate to meet the range of physical demands encountered in daily life, the heart must be able to receive brain, hormonal, and reflex signals. Physical exertion or emotional arousal can stimulate the sympathetic nerves, a part of the involuntary (autonomic) nervous system that also tends to constrict blood vessels. Its effects can almost triple the heart rate and nearly double the heart's pumping strength. Conversely, stimulation of the other division of the autonomic nervous system, the parasympathetic, or vagal, nerves, which often occurs during sleep, slows the heart rate. However, vagal stimulation can also occur during the course of daily life. In fact, the heart rate may slow enough to cause fainting. For example, some people experience this sudden, intense parasympathetic stimulation at the sight of blood.

People are usually unaware of this ongoing adjustment of heart rate that takes place as they move from quiescence to activity, from waking to sleeping. Yogis and others trained in meditation, however, are able voluntarily to slow their own heartbeats. In contrast, a person suffering an anxiety attack may feel

his or her heart racing. The increase in heart rate during panic, to as fast as 170 beats per minute, is caused not only by strong stimulation of the sympathetic nerves, but also by a flood of adrenaline (epinephrine), secreted by the adrenal gland, that reaches the heart through the circulatory system.

Normal heart rates vary with each individual and factors such as cardiovascular conditioning, so casual comparisons of pulse rates can be misleading. For instance, a highly fit athlete at rest will have a slower pulse (45 to 60 beats per minute) than a sedentary individual (65 to 80 beats per minute). If both the sedentary person and the athlete run up a flight of stairs, both heart rates will increase, but the athlete's will not increase as much as and will return to normal sooner than that of the sedentary person, primarily because his or her muscles use oxygen much more efficiently.

Abnormal heart rates and rhythms also have variable causes and consequences in different people. The degree of symptoms alone does not necessarily indicate the seriousness of the underlying disorder. As a consequence, anyone experiencing any of the symptoms outlined should consult a physician.

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## SYMPTOMS OF ARRHYTHMIAS

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Symptoms arise from both slow or fast arrhythmias, but they may be different from person to person. The classic symptoms of arrhythmias include palpitations, dizziness, fainting, chest pain, and shortness of breath. Of course, some of these may not occur, even with serious arrhythmias. People may experience palpitations as missed beats, "skips," "thumps," "butterflies," "fluttering," or "racing"; the palpitations may come in single or multiple beats and maybe felt anywhere from the stomach to the head. People often become more aware of palpitations before going to sleep at night, particularly when they lie on the left side of the body. At this time they are free from distractions, and the bed may act like a drum, amplifying heartbeats.

Palpitations may not be especially bothersome, but light-headedness or fainting (syncope) caused by irregular, rapid, or slow rhythms is harder to ignore. These symptoms usually do not occur unless the heart rate becomes very slow (less than 35 to 45 beats per minute) or extremely rapid (more than 150 beats per minute). In other words, the heart rate rhythm disturbance usually must entail more than just a few

extra beats. The individual passes out because the erratically beating heart fails to pump enough blood to the brain.

A fainting spell caused by heart rhythm abnormalities usually begins with light-headedness rather than the spinning (vertigo) associated with dizziness. The first sensation may be of falling. If the individual recovers before actually passing out, the symptom is known as presyncope. Fainting without warning, however, may occur and may cause injury. If the person is driving a car or operating heavy machinery, fainting obviously can lead to an accident. Any sudden blackout, in the absence of a history of other causes, may indicate an arrhythmia disorder.

The chest pain and shortness of breath that may accompany an arrhythmia usually occur because a rapid heartbeat has put a strain on the heart muscle, which becomes starved for oxygen. The symptoms may be similar to those of angina—pain or pressure originating from the heart but felt anywhere from the stomach to the jaw, including the back, and sometimes associated with nausea or sweating. These symptoms are not common in younger persons who may experience irregular or rapid heartbeats. They are more frequently noticed in older persons with underlying heart disease. Some patients may feel discomfort simply because of the rapid thumping of the heart against the chest.

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## SLOW HEART RHYTHMS (BRADYCARDIAS)

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### SINUSBRADYCARDIA

Doctors define sinus bradycardia as a heart rhythm slower than 60 beats per minute that originates from the normal pacemaker. Almost anyone, however, can go about normal activities with this heart rate. During deep sleep or in young, well-conditioned people, the normal heart rate may actually be as slow as 30 to 40 beats per minute. The heart of a trained athlete can pump more than the usual volume of blood with each beat, making more rapid rates unnecessary.

A slow heart rhythm becomes abnormal when it diminishes the heart's output of blood to the rest of the body enough to cause symptoms ranging from fatigue and shortness of breath to fainting spells. Exercise and increased activity often bring on these symptoms when the heart rate fails to increase to meet the body's needs.

Failure of the sinus node to generate or conduct impulses properly (a condition often referred to as sick sinus syndrome) may underlie some slow heart rhythms. Age or disease may damage the sinus node, excess fibrous or scar tissue may accumulate and interfere with its function, or the autonomic nervous system may fail to regulate its activity properly. A number of antiarrhythmic, antihypertensive, and other drugs can also have adverse effects on sinus node function. (See Chapter 23.) Physicians have recently recognized that children and adolescents who had heart disease at birth (congenital heart disease) that has been surgically corrected may develop sinus node dysfunction in their teens or adulthood. This is a result of scarring from either intrinsic disease or the surgical procedure. (See Chapter 25.)

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## HEART BLOCK

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Slow heart rhythms may also result from the improper transmission of electrical impulses through the atrioventricular node or any of the heart's specialized conduction pathways, despite their normal generation by the sinus node. Doctors often call the condition "heart block," which should not be confused with blockage in the coronary arteries. (See Chapter 11.)

The level of impairment is expressed in degrees. First-degree heart block denotes slow conduction time in the atrioventricular node. Heart rate and rhythm are normal.

Second-degree heart block is diagnosed when some impulses from the atria intermittently fail to reach and activate the ventricles, resulting in a varying number of "dropped beats." Included in this category is a condition known as the Wenckebach phenomenon. This occurs when there is a progressive delay in each ventricular response, resulting in a periodic omission of a single ventricular contraction.

Third-degree heart block, also called complete atrioventricular block, occurs when no impulses from the atria reach the ventricles. If ventricular action is to continue, the heart must rely on an independent junctional or ventricular pacemaker. Sometimes there is a lag before this independent pacemaker takes over. During this time, there is no ventricular contraction, and the person may faint. This is called an Adams-Stokes attack. Usually, though, the ventricular pacemaker eventually establishes a slow rhythm (20 to 45 beats per minute) that is unrelated to the atrial impulses.

The most common causes of heart block are inflammation and scarring of the conducting tissue,

which often result from coronary artery disease or hypertension and the "wear and tear" associated with the aging process. These other parts of the conduction system can also be adversely affected by drugs that interfere with proper sinus node operation. Heart block can occur at any age, although it most often develops in later years. Some children may be born with the condition because of an immune response transmitted from their mothers, a defect in the conducting tissue, or small tumors that disrupt the electrical pathways. These cases are rare.

Symptoms of heart block are similar to those of sinus node disease. They vary depending on the severity and location of the block. Patients with complete block are at greatest risk for fainting or congestive heart failure.

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## RAPID HEART RHYTHM TACHYCARDIAS

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Abnormally fast heart rates are classified into two types: supraventricular (meaning "above the ventricle") tachycardias—those that arise in the atria or the atrioventricular node—and ventricular tachycardias. In both instances, an extra or early beat may trigger the rapid rhythms. Although the sinus node develops as the specialized site of impulse production, all cardiac muscle cells retain the capacity to become pacemaker cells. Normally, the pacemaking activity of the sinus node suppresses impulse production by other cells, but if conductance to some part of the heart muscle is blocked, or if the heart is overstimulated, islands of cells may express their latent impulse-production ability, resulting in extra beats. In other words, impulses are fired from one or more locations in addition to the normal pacemaker, the sinus node.

Extra or early beats arising in the atria are called premature atrial contractions (PACs), atrial premature beats, atrial ectopic beats, or atrial extrasystoles. Such extra beats often occur in normal hearts and are usually harmless. They can, however, cause palpitations, as well as trigger supraventricular tachycardias. Many of these episodes are not serious and can easily be treated.

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## ATRIAL FLUTTER AND FIBRILLATION

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Among the most common supraventricular tachycardias are atrial flutter and fibrillation. They can occur together and may arise in a heart that is otherwise normal and healthy. Flutter results when an extra or

early beat triggers a “circus circular current” that travels in regular cycles around the atrium, pushing the atrial rate up to 250 to 350 beats per minute. The atrioventricular node between the atria and ventricles will often block one of every two beats, keeping the ventricular rate at about 125 to 175 beats per minute. This is the pulse rate that will be felt, even though the atria are beating more rapidly. At this pace, the ventricles will usually continue to pump blood relatively effectively for many hours or even days. A patient with underlying heart disease, however, may experience chest pain, faintness, or even heart failure as a result of the continuing increased stress on the heart muscle. In some individuals, the ventricular rate may also be slower if there is increased block of impulses in the AV node, or faster if there is little or no block.

If the cardiac impulse fails to follow a regular circuit and divides along multiple pathways, a chaos of uncoordinated beats results, producing atrial fibrillation. Fibrillation commonly occurs when the atrium is enlarged (usually because of heart disease). In addition, it can occur in the absence of any apparent heart disease. The atrial rate shoots up to more than 350 beats per minute and the atria fail to pump blood effectively, quivering like “a can of worms” or “a bowl of jelly,” as it has been variously described. The ventricular beat also becomes haphazard, producing a rapid irregular pulse. Although atrial fibrillation may cause the heart to lose 20 to 30 percent of its pumping effectiveness, the volume of blood pumped by the ventricles usually remains within the margin of safety, again because the atrioventricular node blocks out many of the chaotic beats. The ventricle may contract at a rate of only 125 to 175 beats per minute.

Sleep deprivation, excessive caffeine, street drugs such as amphetamine and cocaine, and excessive alcohol consumption increase the heart’s susceptibility to developing atrial flutter or fibrillation. So can heart valve disease, overactivity of the thyroid gland, lung disease, and inflammation of the membranous sac that covers the heart (a condition known as pericarditis). Atrial flutter or fibrillation may go unrecognized by the patient, but they may cause palpitations or light-headedness. Such symptoms are generally not life-threatening, and many people live long and well despite atrial flutter or fibrillation if the rate is controlled.

Though usually harmless, atrial flutter and fibrillation can pose serious risks. In a diseased heart, such arrhythmias can diminish cardiac function and lead to heart failure. Episodes of atrial fibrillation that per-

sist more than several days also carry an additional risk of stroke, because stagnating blood in the atria may clot, producing clumps of clotted blood, which if discharged from the heart (emboli) may be carried to the brain and produce a stroke.

#### PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIAS (PSVTs)

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In this type of rapid heart rhythm, patients experience heart rates in the range of 140 to 250 beats per minute. These episodes often occur first in youth, but may also emerge later in life. While they may be distressing, such attacks are seldom life-threatening. They typically occur in patients who have been born with an extra circuit or pathway between the atria and the ventricles. Such extra circuits occur most commonly within the atrioventricular node, but in an average of 1 or 2 out of 1,000 births, so-called accessory pathways or bypass tracts (sometimes more than one is present) form separate conduction routes. These routes may link the atria and the ventricles at locations quite distant from the atrioventricular node.

Paroxysmal supraventricular tachycardias may be triggered by an ectopic (literally, “out-of-place”) beat, originating in either the atria or the ventricles. If this tachycardia is started by a premature atrial contraction, because the extra atrial beat comes prematurely in the heart’s rhythm cycle, the atrioventricular node or the extra circuit may be blocked. The impulse takes the available route and the ventricles contract. But now the previously blocked path has regained its ability to conduct, and the impulse that has just activated the ventricles is passed back to the atria. Impulses begin to travel around the circuit loop formed by the bypass tract and the atrioventricular node, and a rapid heart rate ensues. The resulting heart rate determines the time required for the impulse to travel around the circuit.

When evidence of a different conduction pathway between the atria and ventricle shows upon the ECG of a patient who experiences symptoms of this type of arrhythmia or atrial fibrillation, the condition is often referred to as Wolff-Parkinson-White syndrome, or WPW. (It is named for the three physicians who first described its most common form.) It should be emphasized that Wolff-Parkinson-White syndrome and related conditions may pose no serious threat if properly treated. Wolff-Parkinson-White syndrome may also be associated with recurrent tachycardias despite medical therapy. In unusual situations, a more serious form of abnormal heartbeat may occur in people with Wolff-Parkinson-White

syndrome. This occurs as ventricular fibrillation where the main pumping chamber beats irregularly at more than 200 beats per minute, and it may result in death.

Like those of other supraventricular tachycardias, the symptoms of this syndrome may not emerge until later in life as the normal conduction system and bypass tract undergo changes. Triggering beats also become more common with age, and may result in more frequent episodes of tachycardia.

### VENTRICULAR ARRHYTHMIAS

In contrast to supraventricular arrhythmias, ventricular arrhythmias are potentially more serious and are more often, but not always, associated with structural heart disease. Premature ventricular contractions (PVCs) are the most common form. Like premature atrial contractions, premature ventricular contractions are early or extra beats that commonly occur and are innocuous in normal hearts, but can cause problems in unhealthy hearts. In rare circumstances, premature ventricular contractions can cause the ventricles to lapse into ventricular fibrillation the heart quivers and ceases to pump blood effectively, and death can occur within 3 to 4 minutes.

Prevention of these potentially dangerous contractions is crucial, because few victims of sudden cardiac arrest survive without immediate first aid. In cities such as Seattle, Washington, vigorous promotion of citizen training in cardiopulmonary resuscitation (CPR) has improved survival rates for victims of ventricular fibrillation. Even so, only 20 to 30 percent of such patients recover and continue to lead normal lives.

Long-term prevention of ventricular fibrillation remains difficult. Unlike atrial arrhythmias that have no symptoms, ventricular arrhythmias or premature ventricular contractions that cause no discomfort can indicate an increased risk of life-threatening ventricular tachycardia or fibrillation, especially in patients with heart disease or a family history of sudden death, although most of the time, these individual contractions are not serious. A physician may need to perform certain tests to aid in the assessment of the risk of these extra beats.

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## DIAGNOSIS

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The techniques used to diagnose and monitor cardiac arrhythmias have become increasingly sophisticated.

The most basic tool is the electrocardiogram (ECG). Adhesive electrodes applied to the chest and limbs connect to a machine that can detect the pattern of minute electric currents in the cardiac muscle and print it out on a strip chart. Electrocardiograms performed to evaluate arrhythmias are most useful if done while symptoms are occurring, which may not be possible if symptoms are brief, infrequent, or absent. Because activity often provokes arrhythmias, an exercise test with electrocardiographic monitoring may prove helpful. (See Figures 16.2, 16.3, 16.4, 16.5, and 16.6 for ECGs showing different heart rhythm disorders.)

The use of computers to enhance and process the electrocardiogram signal (signal-averaged ECG) has improved the test as a means of predicting the risk of potentially dangerous ventricular arrhythmias. Transtelephonic electrocardiograms enable the patient to record his or her own electrocardiographic signal during symptoms and to send the recording to a doctor by telephone. Electrocardiograms using electrodes that are swallowed or inserted through the mouth into the esophagus are called transesophageal ECGs. This technique maybe useful in more difficult cases to diagnose atrial arrhythmias, because the esophagus lies directly behind the atria.

Helter monitors are portable electrocardiogram recorders that patients wear for extended periods, usually 24 to 48 hours. Recorded on tape, the test results are then analyzed by computer. Helter monitors enable a physician to obtain a record of the patient's heartbeat during ordinary activities and may be especially useful for detecting the more serious types of premature ventricular contractions that may be associated with an increased risk of ventricular fibrillation.

Electrophysiology studies form the leading edge of arrhythmia diagnosis and treatment. These studies are not necessary in the vast majority of patients with arrhythmias, but in special cases, they can be extremely useful. Guided by an X-ray picture, physicians thread electrodes via a catheter (a thin, flexible tube) through veins in the arm, neck, shoulder, or groin into the heart, where they can be used to make detailed recordings of the heart's electrical activity. The electrodes can also be used to mimic patterns of extra beats that normally occur in everyday experience to see if they provoke arrhythmias and to assess the effectiveness of therapy.

Electrophysiology studies are usually recommended for survivors of sudden cardiac arrest in order to determine the best means of preventing a recurrence. Other likely candidates include patients

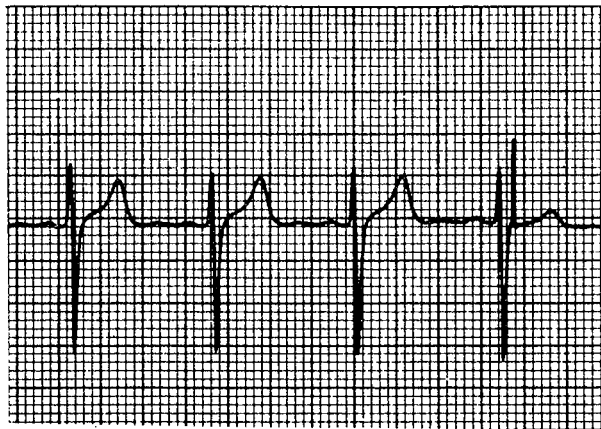


Figure 16.2  
ECG Showing Normal Heart Rhythm

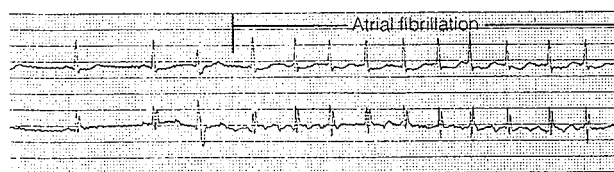


Figure 16.3  
ECGs Showing Atrial Fibrillation

In the top panel, the first two beats are normal, the third is a premature atrial contraction, and the fourth marks the beginning of atrial fibrillation during which the heart rate averages 130 beats per minute and the pattern of the beats is irregular. The lower panel shows sustained atrial fibrillation.

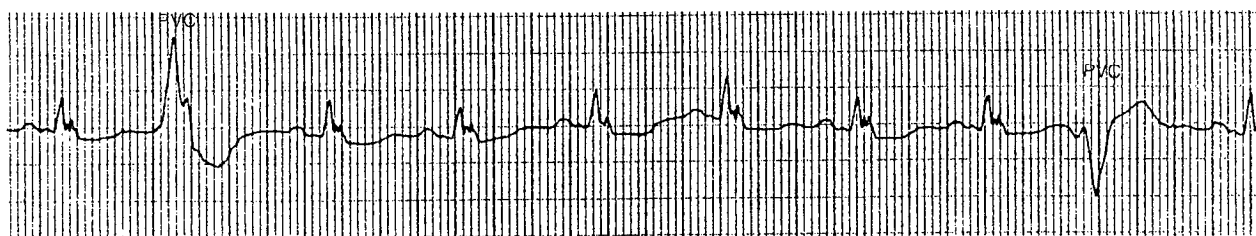


Figure 16.4  
Premature Ventricular Contractions

This electrocardiogram shows a regular rhythm that is punctuated on two occasions (indicated by arrows) by premature ventricular contractions (PVCs). Because these beats arise in the bottom pumping chambers and activate the heart in abnormal fashion, they appear on the ECG as bizarre, wide complexes that appear much different from the normal beats.

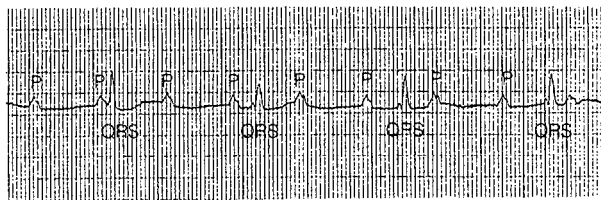


Figure 16.5  
Heart Block

This electrocardiogram shows “complete heart block.” The P waves, representing electrical activity of the natural pacemaker and upper heart chambers (atria), occur at a rate of 94 beats per minute. The QRS complexes, representing contraction of the lower pumping chambers (ventricles), occur at a rate of 44 beats per minute. None of the signals from the upper chambers are getting through to the lower chambers because of a “block” of the electrical circuits connecting them. The lower chambers are beating at a slow rate, which, fortunately, they are capable of generating on their own when no signals come from above. This backup or reserve rhythm is slow and not coordinated with the upper chambers, so pumping of blood becomes inefficient and reduced. There is no reserve pumping capability when needed, such as with physical exertion. This causes the symptoms of fatigue and exhaustion. Implantation of an artificial pacemaker usually restores a normally coordinated heart rhythm.

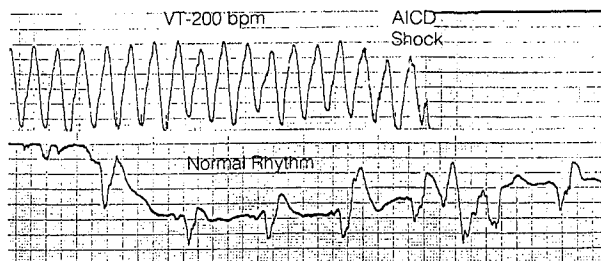


Figure 16.6  
Electrical Conversion of Ventricular Tachycardia

This electrocardiogram demonstrates an attack of ventricular tachycardia, a dangerously rapid heart rhythm that can lead to fainting or, in some instances, death. In this case, the patient did not respond to rhythm-regulating medication, and an automatic defibrillator was surgically implanted. The defibrillator detects the abnormal rhythm and delivers an electric shock that terminates the irregularity and restores normal rhythms.

at high risk for sudden death, those with paroxysmal supraventricular tachycardia or syncope, and those with persistent symptoms whose suspected arrhythmias have eluded detection by other means.

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## DECIDING TO TREAT

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The development of electrophysiology studies has spurred the continuing improvement in treatments for heart rhythm disorders, but the ultimate decision as to whether or how to treat an arrhythmia still rests on an understanding of the whole patient. The patients' overall health, age, life-style, and tolerance of symptoms as well as the arrhythmia itself all weigh into the choice of therapy. Because of such considerations, two people with the same arrhythmia may well receive entirely different treatments. Some patients may not need any treatment at all, and can live long and comfortably with an irregular heart rhythm, confident that the occasional symptom does not signal a serious health problem. (See box, "Self-Help for Arrhythmias.") But because the symptoms do not tell

### **Self-Help for Arrhythmias**

People can often slow down their own heart rates, and possibly suppress supraventricular arrhythmias, by controlling certain nerve impulses. This can be achieved in several ways: holding the breath, taking a slow drink of water, or bathing the face in cold water. Rubbing the neck may also help. If all of this fails, holding the nostrils closed and trying to blow through the nose (making the eardrums "pop") should provide some relief. Simply coughing may stop premature ventricular contractions (PVCs), those extra beats that cause palpitations or thumping in the chest.

Preventative measures include avoiding emotional distress and anxiety. Cutting down on stimulants such as caffeine may help, but an occasional candy bar, a couple of cups of coffee or tea a day, or an occasional caffeinated soda are generally safe to enjoy. Decongestants, often used in cold and allergy medications, can cause rapid heart rates. Also people should abstain from using cigarettes and alcohol because they may increase susceptibility to arrhythmias. With proper professional and self-care, most people with arrhythmias are not held back from living normal, productive lives.

the whole story, anyone who experiences the warning signs of a heart rhythm disorder should be sure to see a doctor.

In selected cases, electrophysiology studies can determine the cause of symptoms, such as fainting spells, or the need for a permanent artificial pacemaker. By administering antiarrhythmic drugs and attempting to induce arrhythmias, cardiologists can directly test the effectiveness of medications without waiting for spontaneous episodes to occur. This offers an advantage in devising safe, effective treatment, because not every antiarrhythmic drug is effective in every patient, and in some circumstances an antiarrhythmic drug may actually worsen the arrhythmia it is intended to suppress. (See Chapter 23.)

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## ANTIARRHYTHMIC DRUGS AND ARTIFICIAL PACEMAKERS

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When used as an antiarrhythmic drug, digitalis, also known as digoxin (Lanoxin), slows impulse conduction through the atrioventricular node, thereby reducing the ventricular rate in order to treat atrial fibrillation or other supraventricular tachycardias.

Beta blockers are drugs used to inhibit the effects of hormones that cause the heart rate to increase. Beta blockers can also enhance effects of other antiarrhythmics. Propranolol (Inderal and others) is a commonly used beta blocker.

The effect of another class of drugs, calcium channel blockers, is similar to that of beta blockers. They change the electrical properties of heart tissues by inhibiting the flow of calcium in and out of cells. A small amount of calcium circulates constantly in the blood and regulates muscle contractions, among other functions. Diltiazem (Cardizem) and verapamil (Calan) are the primary calcium channel blockers used to treat arrhythmias. They slow the sinus rate, but not as effectively as beta blockers. They also slow conduction through the atrioventricular node. Calcium channel blockers, beta blockers, and digitalis are useful in treating atrial fibrillation and paroxysmal supraventricular tachycardias.

Quinidine (Quinidex, Quinora, and others) is a drug that works directly on the heart, as well as through the nerves that lead to heart muscles, to help stabilize irregular heartbeats. Procainamide (Procan), disopyramide (Norpace), and moricizine (Ethmozine)

are synthetic drugs that have much the same uses as quinidine.

Antiarrhythmic drugs that work directly on the heart to suppress ventricular arrhythmias are tocainide (Tonocard) and mexiletine (Mexitil). They are often used in combination with other antiarrhythmic drugs. Flecainide (Tambocor) and propafenone (Rythmol) slow atrioventricular conduction and are effective against both supraventricular and ventricular arrhythmias. All of these antiarrhythmic drugs can worsen arrhythmias in some cases and are generally not prescribed unless careful testing has been done.

Amiodarone (Cordarone) is the most potent antiarrhythmic drug in use. In addition to suppressing virtually all types of arrhythmias, it acts as a beta blocker, an alpha blocker (blocks responses from the alpha-adrenergic nerve receptors), and a calcium channel blocker. Because of its many side effects, amiodarone is approved only for the treatment of serious arrhythmias that do not respond to other drugs. Researchers are seeking a less toxic form of amiodarone that may one day prove to be an antiarrhythmic agent with wider applications.

In some cases, instead of or in addition to drug therapy, a person will need an artificial pacemaker to correct an arrhythmia. Artificial pacemakers work in much the same way as the heart's natural pacemaker. They are small, surgically implanted units, about the size of a cigarette lighter, that use batteries to produce the electrical impulses that stimulate the pumping chambers of the heart. Tiny wires deliver the impulses to the heart muscle. Pacemakers are individually programmed to maintain a person's natural heart rate, and various types of pacemakers, pacing modes, and pacing rates are available to best suit individual needs.

Pacemakers are implanted while the recipient is under local anesthesia, but at least one day of hospitalization is required. Minor surgery is also necessary when the batteries run down and need to be replaced. (See Chapter 26 for more information about pacemakers.)

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## TREATMENT FOR SPECIFIC ARRHYTHMIAS

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In the absence of other heart disease, the prognosis for sinus node dysfunction, the underlying cause for

some slower heart rhythms, is good. When the symptoms of a slow heart rhythm are severe or debilitating, a pacemaker usually will help. Treatment of heart block is similar to that of sinus node disease. That is, patients with complete heart block usually require a pacemaker.

Treatment of atrial flutter and fibrillation is usually aimed at correcting the abnormal rhythm, but if this is not possible, medication, such as a beta blocker, digitalis, or verapamil, can be given to increase the degree of block between the atria and ventricle and slow the heart rate to within the normal range. Even though the heartbeat remains irregular, it is efficient enough to do its job. (People with Wolff-Parkinson-White syndrome who have atrial fibrillation, however, should not take digitalis or verapamil, because these drugs can paradoxically increase the heart rate and the likelihood of ventricular fibrillation.)

The prognosis depends on the overall health of the heart in which atrial flutter or fibrillation occurs. A decision to treat atrial flutter or fibrillation usually rests on how much the symptoms bother the patient. Paradoxically, slow heart rhythms may coexist with atrial flutter or fibrillation, a condition known as tachy-brady (fast-slow) syndrome, which can require treatment with both medicines and a pacemaker.

Chronic and distressing arrhythmias may be treated with electrical cardioversion. Cardioversion is used to treat atrial flutter and other arrhythmias, such as atrial tachycardia, atrial fibrillation, and ventricular tachycardia, when drug therapy fails. In this procedure, the patient is given a short-acting intravenous anesthetic, and an electrical current is delivered to the heart from a defibrillator through conducting paddles applied to the chest. The voltage varies according to the situation. The shock temporarily halts all electrical activity in the heart, allowing it to reestablish a normal heart rhythm by, in effect, starting over. When ventricular fibrillation occurs, electrical defibrillation is an emergency measure. The procedure is safe and effective.

Because atrial fibrillation may cause blood to stagnate in the atria, causing clotting, a physician may recommend an antiarrhythmic drug to maintain normal rhythm, a blood thinner (anticoagulant) to decrease the likelihood of clotting, or both. Though some studies suggest that an aspirin a day may decrease the risk of stroke associated with chronic atrial fibrillation, a more potent blood-thinning drug, such as warfarin (Coumadin), may be required.

People who experience paroxysmal supraventricular tachycardias (PSVTs) may require antiarrhythmic drugs, administered either at the time of an

attack or on a daily basis. Accessory conducting pathways that mediate PSVTs may be surgically cut, preventing further arrhythmias.

A relatively new technique for treating this particular tachycardia, and especially Wolff-Parkinson-White syndrome, without surgery is called *radiofrequency catheter ablation*. In this procedure, a physician inserts a catheter into a blood vessel and threads it, under X-ray guidance, up to the area of the heart muscle where the accessory pathway is located. A mild current, produced by very-high-frequency alternating current—that is, radiofrequency current—is then transmitted from the catheter electrode tip to the site of the pathway. (This same current is the familiar “electric needle” used in various electrocautery procedures.) The resistance of the heart muscle to the current generates a small amount of heat. An increase of 10 degrees is all that is necessary to cause the death of the heart muscle cells in a very small area, about  $\frac{1}{8}$  inch in diameter. Once this occurs, the pathways can no longer conduct the extra impulses. The procedure produces little or no discomfort. It is done under mild sedation with local anesthesia, and the patient can return to normal activities within a few days.

Drug treatment aimed at suppressing premature ventricular contractions (PVCs) to prevent serious ventricular arrhythmias often fails to reduce the risk of sudden death. Prospects for effective treatments have brightened, however, with the recent development of electrophysiology studies and treatment programs that combine drug therapy with surgery and antiarrhythmic devices, such as implantable defibrillator. (See Chapter 26.) Individuals who have ventricular fibrillation or ventricular tachycardia (especially combined with fainting) should probably undergo full evaluation to determine the best treatment.

A newer and increasingly used option for treating life-threatening ventricular arrhythmias is the automatic implantable cardioverter-defibrillator (AICD). Unlike other types of treatment, this does not prevent arrhythmias but instead stops them within seconds. An electrode lead system is attached directly to the

heart, leading to a pulse generator that is implanted under the skin in the upper abdomen. The pulse generator continuously monitors heart rate and rhythm through signals from the leads. When a tachyarrhythmia is detected, the pulse generator responds. It sends a series of up to five shocks via patch electrodes that are sewn directly onto the outside of the heart. This direct electric current should restore proper rhythm.

The automatic implantable cardioverter-defibrillator has proved extremely effective. The sudden death rate within the first year is 1 to 2 percent in patients who receive this device, compared with 20 to 50 percent for people who go untreated. The device is appropriate for people who have ventricular arrhythmias that cannot be controlled with drug therapy, but at present it can be implanted only in people who can withstand chest surgery.

Surgery, like ablation, has the potential to cure a person suffering from arrhythmias. This approach, however, should be taken only by people who have extremely serious arrhythmias that still occur despite antiarrhythmic medications, or by younger people who otherwise face a lifetime of drug therapy.

Surgery can provide a cure for atrial arrhythmias that occur when more than one electrical pathway exists; extra electrical pathways are destroyed or cut out. Traditionally people with Wolff-Parkinson-White syndrome are likely candidates for this surgery, although this now is accomplished most often with radiofrequency ablation. In the case of ventricular arrhythmias, sometimes the starting point for the abnormal impulses can be determined through electrophysiology testing and can be cut out, but the surgical mortality rate is 10 to 15 percent, and there may be recurrences in 20 to 30 percent of the cases. Often other necessary operations, such as coronary artery bypasses, are performed at the same time as antiarrhythmic surgery, and in some cases, part or all of an automatic implantable cardioverter-defibrillator system is attached as a backup. This eliminates the need for a second chest operation should the system be needed later.