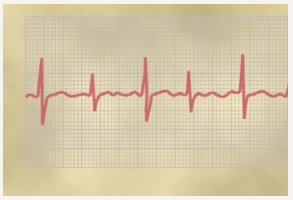
Cardiac Arrhythmias and Their Electrocardiographic Interpretation



Some of the most distressing types of heart malfunction occur not as a result of abnormal heart muscle but because of abnormal rhythm of the heart. For instance, sometimes the beat of the atria is not coordinated with the beat of the ventricles, so that the atria no longer function as primer pumps for the ventricles.

The purpose of this chapter is to discuss the physiology of common cardiac arrhythmias and their effects on heart pumping, as well as their diagnosis by electrocardiography. The causes of the cardiac arrhythmias are usually one or a combination of the following abnormalities in the rhythmicity-conduction system of the heart:

1. Abnormal rhythmicity of the pacemaker
2. Shift of the pacemaker from the sinus node to another place in the heart
3. Blocks at different points in the spread of the impulse through the heart
4. Abnormal pathways of impulse transmission through the heart
5. Spontaneous generation of spurious impulses in almost any part of the heart

Abnormal Sinus Rhythms

Tachycardia

The term "tachycardia" means *fast heart rate,* usually defined in an adult person as faster than 100 beats per minute. An electrocardiogram recorded from a patient with tachycardia is shown in Figure 13–1. This electrocardiogram is normal except that the heart rate, as determined from the time intervals between QRS complexes, is about 150 per minute instead of the normal 72 per minute.

The general causes of tachycardia include *increased body temperature, stimulation of the heart by the sympathetic nerves, or toxic conditions of the heart.*

The heart rate increases about 10 beats per minute for each degree Fahrenheit (18 beats per degree Celsius) increase in body temperature, up to a body temperature of about 105°F (40.5°C); beyond this, the heart rate may decrease because of progressive debility of the heart muscle as a result of the fever. Fever causes tachycardia because increased temperature increases the rate of metabolism of the sinus node, which in turn directly increases its excitability and rate of rhythm.

Many factors can cause the sympathetic nervous system to excite the heart, as we discuss at multiple points in this text. For instance, when a patient loses blood and passes into a state of shock or semishock, sympathetic reflex stimulation of the heart often increases the heart rate to 150 to 180 beats per minute.

Simple weakening of the myocardium usually increases the heart rate because the weakened heart does not pump blood into the arterial tree to a normal extent, and this elicits sympathetic reflexes to increase the heart rate.

Bradycardia

The term "bradycardia" means a slow heart rate, usually defined as fewer than 60 beats per minute. Bradycardia is shown by the electrocardiogram in Figure 13–2.

Bradycardia in Athletes. The athlete's heart is larger and considerably stronger than that of a normal person, which allows the athlete's heart to pump a large stroke volume output per beat even during periods of rest. When the athlete is at rest, excessive quantities of blood pumped into the arterial tree with each beat initiate feedback circulatory reflexes or other effects to cause bradycardia.

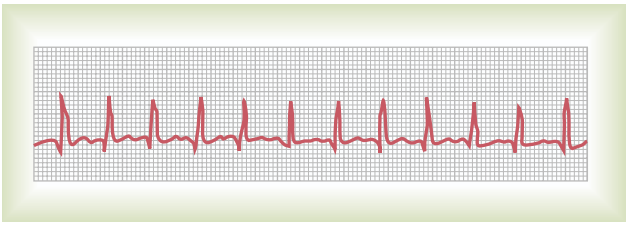


Figure 13–1

Sinus tachycardia (lead I).

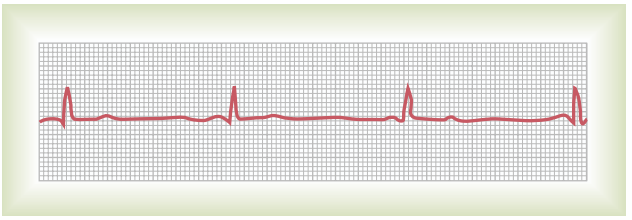


Figure 13–2

Sinus bradycardia (lead III).

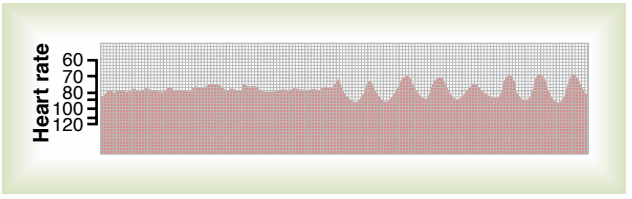


Figure 13–3

Sinus arrhythmia as recorded by a cardiotachometer. To the left is the record when the subject was breathing normally; to the right, when breathing deeply.

Vagal Stimulation as a Cause of Bradycardia. Any circulatory reflex that stimulates the vagus nerves causes release of acetylcholine at the vagal endings in the heart, thus giving a parasympathetic effect. Perhaps the most striking example of this occurs in patients with *carotid sinus syndrome.* In these patients, the pressure receptors (baroreceptors) in the carotid sinus region of the carotid artery walls are excessively sensitive. Therefore, even mild external pressure on the neck elicits a strong baroreceptor reflex, causing intense vagal-acetylcholine effects on the heart, including extreme bradycardia. Indeed, sometimes this reflex is so powerful that it actually stops the heart for 5 to 10 seconds.

Sinus Arrhythmia

Figure 13–3 shows a *cardiotachometer* recording of the heart rate, at first during normal and then (in the second half of the record) during deep respiration. A cardiotachometer is an instrument that records *by the height of successive spikes* the duration of the interval between the successive QRS complexes in the electrocardiogram. Note from this record that the heart rate increased and decreased no more than 5 per cent during quiet respiration (left half of the record). Then, *during deep respiration*, the heart rate increased and decreased with each respiratory cycle by as much as 30 per cent.

Sinus arrhythmia can result from any one of many circulatory conditions that alter the strengths of the sympathetic and parasympathetic nerve signals to the heart sinus node. In the "respiratory" type of sinus arrhythmia, as shown in Figure 13–3, this results mainly from "spillover" of signals from the medullary respiratory center into the adjacent vasomotor center during inspiratory and expiratory cycles of respiration. The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart.

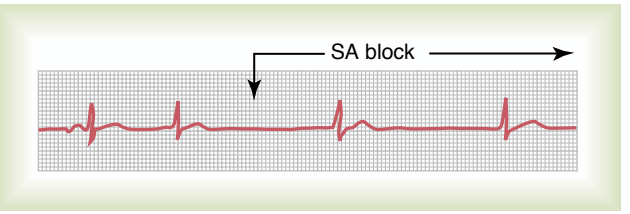


Figure 13–4

Sinoatrial nodal block, with A-V nodal rhythm during the block period (lead III).

Abnormal Rhythms That Result from Block of Heart Signals Within the Intracardiac Conduction Pathways

Sinoatrial Block

In rare instances, the impulse from the sinus node is blocked before it enters the atrial muscle. This phenomenon is demonstrated in Figure 13–4, which shows sudden cessation of P waves, with resultant standstill of the atria. However, the ventricles pick up a new rhythm, the impulse usually originating spontaneously in the atrioventricular (A-V) node, so that the rate of the ventricular QRS-T complex is slowed but not otherwise altered.

Atrioventricular Block

The only means by which impulses ordinarily can pass from the atria into the ventricles is through the *A-V bundle,* also known as the *bundle of His.* Conditions that can either decrease the rate of impulse conduction in this bundle or block the impulse entirely are as follows:

1. *Ischemia of the A-V node or A-V bundle fibers* often delays or blocks conduction from the atria to the ventricles. Coronary insufficiency can cause ischemia of the A-V node and bundle in the same way that it can cause ischemia of the myocardium.
2. *Compression of the A-V bundle* by scar tissue or by calcified portions of the heart can depress or block conduction from the atria to the ventricles.
3. *Inflammation of the A-V node or A-V bundle* can depress conductivity from the atria to the ventricles. Inflammation results frequently from

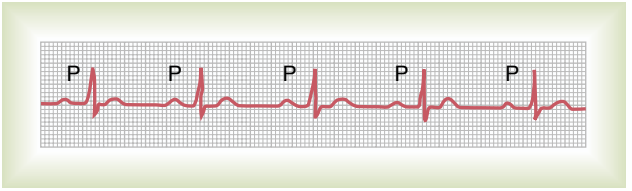


Figure 13–5

Prolonged P-R interval caused by first degree A-V heart block (lead II).

different types of myocarditis, caused, for example, by diphtheria or rheumatic fever.

4. *Extreme stimulation of the heart by the vagus nerves* in rare instances blocks impulse conduction through the A-V node. Such vagal excitation occasionally results from strong stimulation of the baroreceptors in people with *carotid sinus syndrome,* discussed earlier in relation to bradycardia.

Incomplete Atrioventricular Heart Block

Prolonged P-R (or P-Q) Interval—First Degree Block. The usual lapse of time between *beginning* of the P wave and *beginning* of the QRS complex is about 0.16 second when the heart is beating at a normal rate. This so-called *P-R interval* usually decreases in length with faster heartbeat and increases with slower heartbeat. In general, when the P-R interval increases to greater than 0.20 second, the P-R interval is said to be prolonged, and the patient is said to have *first degree incomplete heart block.*

Figure 13–5 shows an electrocardiogram with prolonged P-R interval; the interval in this instance is about 0.30 second instead of the normal 0.20 or less. Thus, first degree block is defined as a *delay* of conduction from the atria to the ventricles but not actual blockage of conduction. The P-R interval seldom increases above 0.35 to 0.45 second because, by that time, conduction through the A-V bundle is depressed so much that conduction stops entirely. One means for determining the severity of some heart diseases—*acute rheumatic heart disease*, for instance—is to measure the P-R interval.

Second Degree Block. When conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.25 to 0.45 second, the action potential sometimes is strong enough to pass through the bundle into the ventricles and sometimes is not strong enough. In this instance, there will be an atrial P wave but no QRS-T wave, and it is said that there are "dropped beats" of the ventricles. This condition is called *second degree heart block.*

Figure 13–6 shows P-R intervals of 0.30 second, as well as one dropped ventricular beat as a result of failure of conduction from the atria to the ventricles.

At times, every other beat of the ventricles is dropped, so that a "2:1 rhythm" develops, with the atria beating twice for every single beat of the ventricles. At other times, rhythms of 3:2 or 3:1 also develop.

Complete A-V Block (Third Degree Block). When the condition causing poor conduction in the A-V node or A-V bundle becomes severe, complete block of the impulse

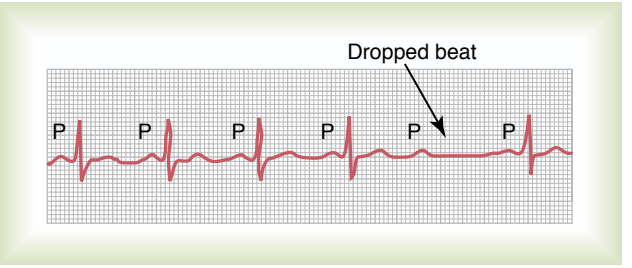


Figure 13–6

Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead V_3).

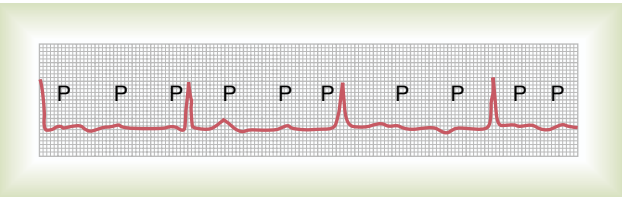


Figure 13–7

Complete A-V block (lead II).

from the atria into the ventricles occurs. In this instance, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle. Therefore, the P waves become dissociated from the QRS-T complexes, as shown in Figure 13–7. Note that the *rate of rhythm of the atria* in this electrocardiogram is about 100 beats per minute, whereas the *rate of ventricular beat* is less than 40 per minute. Furthermore, there is no relation between the rhythm of the P waves and that of the QRS-T complexes because the ventricles have "escaped" from control by the atria, and they are beating at their own natural rate, controlled most often by rhythmical signals generated in the A-V node or A-V bundle.

Stokes-Adams Syndrome—Ventricular Escape. In some patients with A-V block, the total block comes and goes; that is, impulses are conducted from the atria into the ventricles for a period of time and then suddenly impulses are not conducted. The duration of block may be a few seconds, a few minutes, a few hours, or even weeks or longer before conduction returns. This condition occurs in hearts with borderline ischemia of the conductive system.

Each time A-V conduction ceases, the ventricles often do not start their own beating until after a delay of 5 to 30 seconds. This results from the phenomenon called *overdrive suppression.* This means that ventricular excitability is at first in a suppressed state because the ventricles have been driven by the atria at a rate greater than their natural rate of rhythm. However, after a few seconds, some part of the Purkinje system beyond the block, usually in the distal part of the A-V node beyond the blocked point in the node, or in the A-V bundle, begins discharging rhythmically at a rate of 15 to 40 times per minute and acting as the pacemaker of the ventricles. This is called *ventricular escape.*

Because the brain cannot remain active for more than 4 to 7 seconds without blood supply, most patients faint a few seconds after complete block occurs because the heart does not pump any blood for 5 to 30 seconds, until the ventricles "escape." After escape, however, the slowly beating ventricles usually pump enough blood to allow rapid recovery from the faint and then to sustain the person. These periodic fainting spells are known as the *Stokes-Adams syndrome.*

Occasionally the interval of ventricular standstill at the onset of complete block is so long that it becomes detrimental to the patient's health or even causes death. Consequently, most of these patients are provided with an *artificial pacemaker,* a small battery-operated electrical stimulator planted beneath the skin, with electrodes usually connected to the right ventricle. The pacemaker provides continued rhythmical impulses that take control of the ventricles.

Incomplete Intraventricular Block—Electrical Alternans

Most of the same factors that can cause A-V block can also block impulse conduction in the peripheral ventricular Purkinje system. Figure 13–8 shows the condition known as *electrical alternans,* which results from partial intraventricular block every other heartbeat. This electrocardiogram also shows *tachycardia* (rapid heart rate), which is probably the reason the block has occurred, because when the rate of the heart is rapid, it may be impossible for some portions of the Purkinje system to recover from the previous refractory period quickly enough to respond during every succeeding heartbeat. Also, many conditions that depress the heart, such as ischemia, myocarditis, or digitalis toxicity, can cause incomplete intraventricular block, resulting in electrical alternans.

Premature Contractions

A premature contraction is a contraction of the heart before the time that normal contraction would have been expected. This condition is also called *extrasystole, premature beat,* or *ectopic beat.*

Causes of Premature Contractions. Most premature contractions result from *ectopic foci* in the heart, which emit abnormal impulses at odd times during the cardiac rhythm. Possible causes of ectopic foci are (1) local areas of ischemia; (2) small calcified plaques at different points in the heart, which press against the adjacent cardiac muscle so that some of the fibers are irritated; and (3) toxic irritation of the A-V node, Purkinje system, or myocardium caused by drugs, nicotine, or caffeine. Mechanical initiation of premature contractions is also frequent during cardiac catheterization; large numbers of premature contractions often occur when the catheter enters the right ventricle and presses against the endocardium.

Premature Atrial Contractions

Figure 13–9 shows a single premature atrial contraction. The P wave of this beat occurred too soon in the heart cycle; the P-R interval is shortened, indicating that the ectopic origin of the beat is in the atria near the A-V node. Also, the interval between the premature contraction and the next succeeding contraction is slightly prolonged, which is called a *compensatory pause.* One of the reasons for this is that the premature contraction originated in the atrium some distance from the sinus node, and the impulse had to travel through a considerable amount of atrial muscle before it discharged the sinus node. Consequently, the sinus node discharged late in the premature cycle, and this made the succeeding sinus node discharge also late in appearing.

Premature atrial contractions occur frequently in otherwise healthy people. Indeed, they often occur in athletes whose hearts are in very healthy condition. Mild toxic conditions resulting from such factors as smoking, lack of sleep, ingestion of too much coffee, alcoholism, and use of various drugs can also initiate such contractions.

Pulse Deficit. When the heart contracts ahead of schedule, the ventricles will not have filled with blood normally, and the stroke volume output during that contraction is depressed or almost absent. Therefore, the pulse wave passing to the peripheral arteries after a premature contraction may be so weak that it cannot be felt in the radial artery. Thus, a deficit in the number of radial pulses occurs when compared with the actual number of contractions of the heart.

A-V Nodal or A-V Bundle Premature Contractions

Figure 13–10 shows a premature contraction that originated in the A-V node or in the A-V bundle. The P wave is missing from the electrocardiographic record

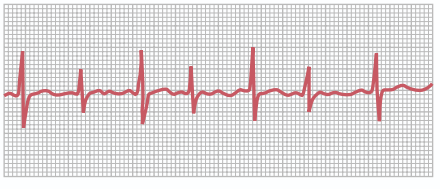


Figure 13–8

Partial intraventricular block—"electrical alternans" (lead III).

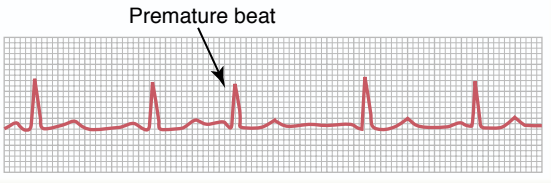


Figure 13–9

Atrial premature beat (lead I).

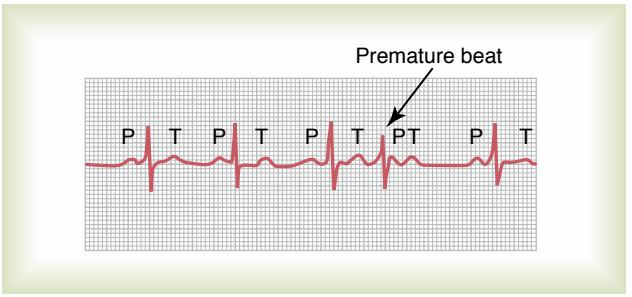


Figure 13–10

A-V nodal premature contraction (lead III).

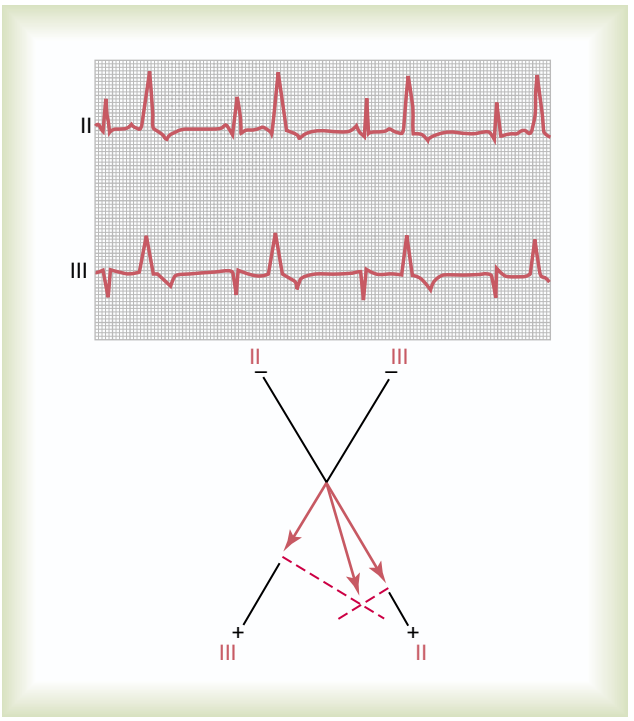


Figure 13–11

Premature ventricular contractions (PVCs) demonstrated by the large abnormal QRS-T complexes (leads II and III). Axis of the premature contractions is plotted in accordance with the principles of vectorial analysis explained in Chapter 12; this shows the origin of the PVC to be near the base of the ventricles.

of the premature contraction. Instead, the P wave is superimposed onto the QRS-T complex because the cardiac impulse traveled backward into the atria at the same time that it traveled forward into the ventricles; this P wave slightly distorts the QRS-T complex, but the P wave itself cannot be discerned as such. In general, A-V nodal premature contractions have the same significance and causes as atrial premature contractions.

Premature Ventricular Contractions

The electrocardiogram of Figure 13–11 shows a series of premature ventricular contractions (PVCs) alternating with normal contractions. PVCs cause specific effects in the electrocardiogram, as follows:

1. The QRS complex is usually considerably prolonged. The reason is that the impulse is conducted mainly through slowly conducting muscle of the ventricles rather than through the Purkinje system.
2. The QRS complex has a high voltage for the following reasons: when the normal impulse passes through the heart, it passes through both ventricles nearly simultaneously; consequently, in the normal heart, the depolarization waves of the two sides of the heart—mainly of opposite polarity to each other—partially neutralize each other in the electrocardiogram. When a PVC occurs, the impulse almost always travels in only one direction, so that there is no such neutralization effect, and one entire side or end of the ventricles is depolarized ahead of the other; this causes large electrical potentials, as shown for the PVCs in Figure 13–11.
3. After almost all PVCs, the T wave has an electrical potential polarity exactly opposite to that of the QRS complex, because the *slow conduction of the impulse* through the cardiac muscle causes the muscle fibers that depolarize first also to repolarize first.

Some PVCs are relatively benign in their effects on overall pumping by the heart; they can result from such factors as cigarettes, coffee, lack of sleep, various mild toxic states, and even emotional irritability. Conversely, many other PVCs result from stray impulses or reentrant signals that originate around the borders of infarcted or ischemic areas of the heart. The presence of such PVCs is not to be taken lightly. Statistics show that people with significant numbers of PVCs have a much higher than normal chance of developing spontaneous lethal ventricular fibrillation, presumably initiated by one of the PVCs. This is especially true when the PVCs occur during the vulnerable period for causing fibrillation, just at the end of the T wave when the ventricles are coming out of refractoriness, as explained later in the chapter.

Vector Analysis of the Origin of an Ectopic Premature Ventricular Contraction. In Chapter 12, the principles of vectorial analysis are explained. Applying these principles, one can determine from the electrocardiogram in Figure 13–11 the point of origin of the PVC as follows: Note that the potentials of the premature contractions in leads II and III are both strongly positive. Plotting these potentials on the axes of leads II and III and solving by vectorial analysis for the mean QRS vector in the heart, one finds that the vector of this premature contraction has its negative end (origin) at the base of the heart and its positive end toward the apex. Thus, the first portion of the heart to become depolarized during this premature contraction is near the base of the ventricles, which therefore is the locus of the ectopic focus.

Paroxysmal Tachycardia

Some abnormalities in different portions of the heart, including the atria, the Purkinje system, or the ventricles, can occasionally cause rapid rhythmical discharge of impulses that spread in all directions throughout the heart. This is believed to be caused most frequently by

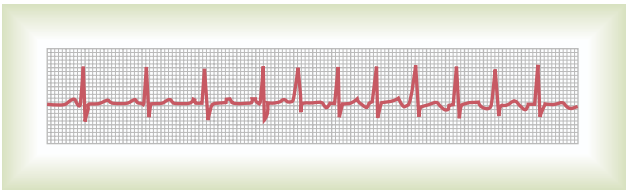


Figure 13–12

Atrial paroxysmal tachycardia—onset in middle of record (lead I).

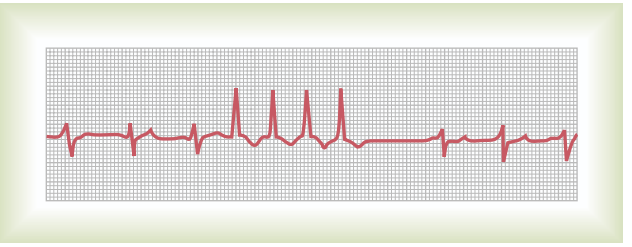


Figure 13–13

Ventricular paroxysmal tachycardia (lead III).

re-entrant circus movement feedback pathways that set up local repeated self–re-excitation. Because of the rapid rhythm in the irritable focus, this focus becomes the pacemaker of the heart.

The term "paroxysmal" means that the heart rate becomes rapid in paroxysms, with the paroxysm beginning suddenly and lasting for a few seconds, a few minutes, a few hours, or much longer. Then the paroxysm usually ends as suddenly as it began, with the pacemaker of the heart instantly shifting back to the sinus node.

Paroxysmal tachycardia often can be stopped by eliciting a vagal reflex. A type of vagal reflex sometimes elicited for this purpose is to press on the neck in the regions of the carotid sinuses, which may cause enough of a vagal reflex to stop the paroxysm. Various drugs may also be used. Two drugs frequently used are quinidine and lidocaine, either of which depresses the normal increase in sodium permeability of the cardiac muscle membrane during generation of the action potential, thereby often blocking the rhythmical discharge of the focal point that is causing the paroxysmal attack.

Atrial Paroxysmal Tachycardia

Figure 13–12 demonstrates in the middle of the record a sudden increase in the heart rate from about 95 to about 150 beats per minute. On close study of the electrocardiogram during the rapid heartbeat, an inverted P wave is seen before each QRS-T complex, and this P wave is partially superimposed onto the normal T wave of the preceding beat. This indicates that the origin of this paroxysmal tachycardia is in the atrium, but because the P wave is abnormal in shape, the origin is not near the sinus node.

A-V Nodal Paroxysmal Tachycardia. Paroxysmal tachycardia often results from an aberrant rhythm that involves the A-V node. This usually causes almost normal QRS-T complexes but totally missing or obscured P waves.

Atrial or A-V nodal paroxysmal tachycardia, both of which are called *supraventricular tachycardias,* usually occurs in young, otherwise healthy people, and they generally grow out of the predisposition to tachycardia after adolescence. In general, supraventricular tachycardia frightens a person tremendously and may cause weakness during the paroxysm, but only seldom does permanent harm come from the attack.

Ventricular Paroxysmal Tachycardia

Figure 13–13 shows a typical short paroxysm of ventricular tachycardia. The electrocardiogram of ventricular paroxysmal tachycardia has the appearance of a series of ventricular premature beats occurring one after another without any normal beats interspersed.

Ventricular paroxysmal tachycardia is usually a serious condition for two reasons. First, this type of tachycardia usually does not occur unless considerable ischemic damage is present in the ventricles. Second, ventricular tachycardia *frequently initiates the lethal condition of ventricular fibrillation* because of rapid repeated stimulation of the ventricular muscle, as we discuss in the next section.

Sometimes intoxication from the heart treatment drug *digitalis* causes irritable foci that lead to ventricular tachycardia. Conversely, *quinidine*, which increases the refractory period and threshold for excitation of cardiac muscle, may be used to block irritable foci causing ventricular tachycardia.

Ventricular Fibrillation

The most serious of all cardiac arrhythmias is *ventricular fibrillation,* which, if not stopped within 1 to 3 minutes, is almost invariably fatal. Ventricular fibrillation results from cardiac impulses that have gone berserk within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion, then another, and eventually feeding back onto itself to re-excite the same ventricular muscle over and over—never stopping. When this happens, many small portions of the ventricular muscle will be contracting at the same time, while equally as many other portions will be relaxing. Thus, there is never a coordinate contraction of all the ventricular muscle at once, which is required for a pumping cycle of the heart. Despite massive movement of stimulatory signals throughout the ventricles, the ventricular chambers neither enlarge nor contract but remain in an indeterminate stage of partial contraction, pumping either no blood or negligible amounts. Therefore, after fibrillation begins, unconsciousness occurs within 4 to 5 seconds for lack of blood flow to the brain, and irretrievable death of tissues begins to occur throughout the body within a few minutes.

Multiple factors can spark the beginning of ventricular fibrillation—a person may have a normal heartbeat one moment, but 1 second later, the ventricles are in fibrillation. Especially likely to initiate fibrillation are (1) sudden electrical shock of the heart, or (2) ischemia of the heart muscle, of its specialized conducting system, or both.

Phenomenon of Re-entry—"Circus Movements" as the Basis for Ventricular Fibrillation

When the *normal* cardiac impulse in the normal heart has traveled through the extent of the ventricles, it has no place to go because all the ventricular muscle is refractory and cannot conduct the impulse farther. Therefore, that impulse dies, and the heart awaits a new action potential to begin in the atrial sinus node.

Under some circumstances, however, this normal sequence of events does not occur. Therefore, let us explain more fully the background conditions that can initiate re-entry and lead to "circus movements," which in turn cause ventricular fibrillation.

Figure 13–14 shows several small cardiac muscle strips cut in the form of circles. If such a strip is stimulated at the 12 o'clock position *so that the impulse travels in only one direction,* the impulse spreads progressively around the circle until it returns to the 12 o'clock position. If the originally stimulated muscle fibers are still in a refractory state, the impulse then dies out because refractory muscle cannot transmit a second impulse. But there are three different conditions that can cause this impulse to continue to travel around the circle, that is, to cause "re-entry" of the impulse into muscle that has already been excited. This is called a "circus movement."

First, if the *pathway around the circle is too long,* by the time the impulse returns to the 12 o'clock position, the originally stimulated muscle will no longer be refractory and the impulse will continue around the circle again and again.

Second, if the length of the pathway remains constant but the *velocity of conduction becomes decreased* enough, an increased interval of time will elapse before the impulse returns to the 12 o'clock position. By this time, the originally stimulated muscle might be out of the refractory state, and the impulse can continue around the circle again and again.

Third, *the refractory period of the muscle might become greatly shortened.* In this case, the impulse could also continue around and around the circle.

All these conditions occur in different pathological states of the human heart, as follows: (1) A long pathway typically occurs in dilated hearts. (2) Decreased rate of conduction frequently results from (a) blockage of the Purkinje system, (b) ischemia of the muscle, (c) high blood potassium levels, or (d) many other factors. (3) A shortened refractory period commonly occurs in response to various drugs, such as epinephrine, or after repetitive electrical stimulation. Thus, in many cardiac disturbances, re-entry can cause abnormal patterns of cardiac contraction or abnormal cardiac rhythms that ignore the pace-setting effects of the sinus node.

Chain Reaction Mechanism of Fibrillation

In ventricular fibrillation, one sees many separate and small contractile waves spreading at the same time in different directions over the cardiac muscle. The re-entrant impulses in fibrillation are not simply a single impulse moving in a circle, as shown in Figure 13–14. Instead, they have degenerated into a series of multiple wave fronts that have the appearance of a "chain reaction." One of the best ways to explain this process in fibrillation is to describe the initiation of fibrillation by electric shock caused by 60-cycle alternating electric current.

Fibrillation Caused by 60-Cycle Alternating Current. At a central point in the ventricles of heart A in Figure 13–15, a 60-cycle electrical stimulus is applied through a stimulating electrode. The first cycle of the electrical stimulus causes a depolarization wave to spread in all directions, leaving all the muscle beneath the electrode in a refractory state. After about 0.25 second, part of this muscle begins to come out of the refractory state. Some portions come out of refractoriness before other

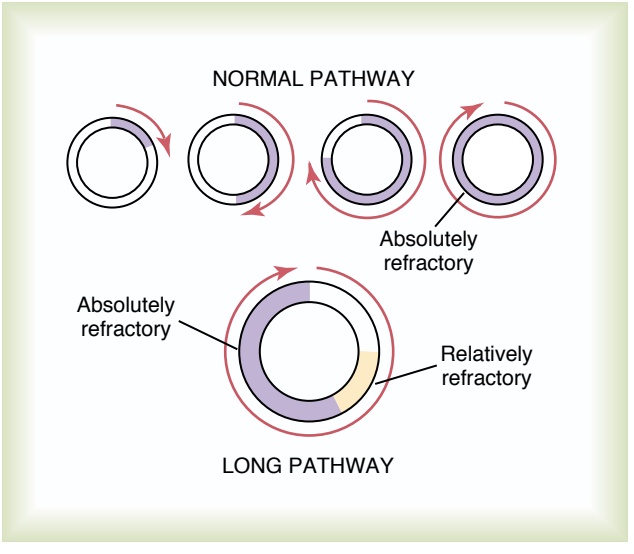


Figure 13–14

Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

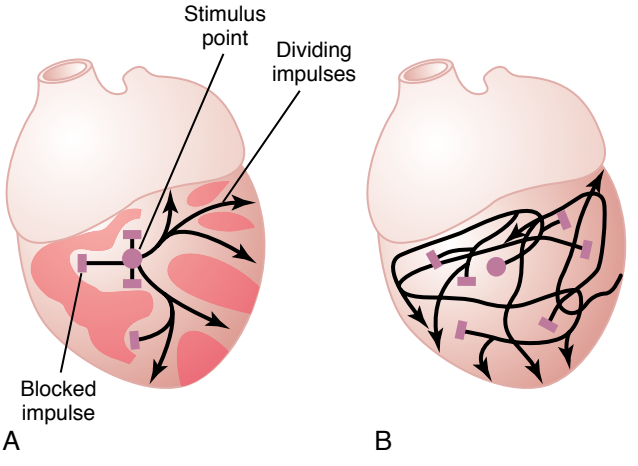


Figure 13–15

A, Initiation of fibrillation in a heart when patches of refractory musculature are present. *B,* Continued propagation of *fibrillatory impulses* in the fibrillating ventricle.

portions. This state of events is depicted in heart A by many lighter patches, which represent excitable cardiac muscle, and dark patches, which represent still refractory muscle. Now, continuing 60-cycle stimuli from the electrode can cause impulses to travel only in certain directions through the heart but not in all directions. Thus, in heart A, certain impulses travel for short distances, until they reach refractory areas of the heart, and then are blocked. But other impulses pass between the refractory areas and continue to travel in the excitable areas. Then, several events transpire in rapid succession, all occurring simultaneously and eventuating in a state of fibrillation.

First, block of the impulses in some directions but successful transmission in other directions creates one of the necessary conditions for a re-entrant signal to develop—that is, *transmission of some of the depolarization waves around the heart in only some directions but not other directions.*

Second, the rapid stimulation of the heart causes two changes in the cardiac muscle itself, both of which predispose to circus movement: (1) The *velocity of conduction through the heart muscle decreases,* which allows a longer time interval for the impulses to travel around the heart. (2) The *refractory period of the muscle is shortened,* allowing re-entry of the impulse into previously excited heart muscle within a much shorter time than normally.

Third, one of the most important features of fibrillation is the *division of impulses,* as demonstrated in heart A. When a depolarization wave reaches a refractory area in the heart, it travels to both sides around the refractory area. Thus, a single impulse becomes two impulses. Then, when each of these reaches another refractory area, it, too, divides to form two more impulses. In this way, many new wave fronts are continually being formed in the heart by progressive *chain reactions* until, finally, there are many small depolarization waves traveling in many directions at the same time. Furthermore, this irregular pattern of impulse travel causes *many circuitous routes for the impulses to travel, greatly lengthening the conductive pathway, which is one of the conditions that sustains the fibrillation.* It also results in a continual irregular pattern of patchy refractory areas in the heart.

One can readily see when a vicious circle has been initiated: More and more impulses are formed; these cause more and more patches of refractory muscle, and the refractory patches cause more and more division of the impulses. Therefore, any time a single area of cardiac muscle comes out of refractoriness, an impulse is close at hand to re-enter the area.

Heart B in Figure 13–15 demonstrates the final state that develops in fibrillation. Here one can see many impulses traveling in all directions, some dividing and increasing the number of impulses, whereas others are blocked by refractory areas. In fact, a single electric shock during this vulnerable period frequently can lead to an odd pattern of impulses spreading multidirectionally around refractory areas of muscle, which will lead to fibrillation.

Electrocardiogram in Ventricular Fibrillation

In ventricular fibrillation, the electrocardiogram is bizarre (Figure 13–16) and ordinarily shows no tendency toward a regular rhythm of any type. During the first few seconds of ventricular fibrillation, relatively large masses of muscle contract simultaneously, and this causes coarse, irregular waves in the electrocardiogram. After another few seconds, the coarse contractions of the ventricles disappear, and the electrocardiogram changes into a new pattern of low-voltage, very irregular waves. Thus, no repetitive electrocardiographic pattern can be ascribed to ventricular fibrillation. Instead, the ventricular muscle contracts at as many as 30 to 50 small patches of muscle at a time, and electrocardiographic potentials change constantly and spasmodically because the electrical currents in the heart flow first in one direction and then in another and seldom repeat any specific cycle.

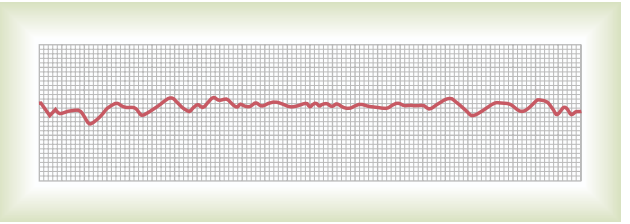


Figure 13–16

Ventricular fibrillation (lead II).

The voltages of the waves in the electrocardiogram in ventricular fibrillation are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly so that after 20 to 30 seconds, they are usually only 0.2 to 0.3 millivolt. Minute voltages of 0.1 millivolt or less may be recorded for 10 minutes or longer after ventricular fibrillation begins. As already pointed out, because no pumping of blood occurs during ventricular fibrillation, this state is lethal unless stopped by some heroic therapy, such as immediate electroshock through the heart, as explained in the next section.

Electroshock Defibrillation of the Ventricles

Although a moderate alternating-current voltage applied directly to the ventricles almost invariably throws the ventricles into fibrillation, a strong high-voltage alternating electrical current passed through the ventricles for a fraction of a second can stop fibrillation by throwing all the ventricular muscle into refractoriness simultaneously. This is accomplished by passing intense current through large electrodes placed on two sides of the heart. The current penetrates most of the fibers of the ventricles at the same time, thus stimulating essentially all parts of the ventricles simultaneously and causing them all to become refractory. All action potentials stop, and the heart remains quiescent for 3 to 5 seconds, after which it begins to beat again, usually with the sinus node or some other part of the heart becoming the pacemaker. However, the same re-entrant focus that had originally thrown the ventricles into fibrillation often is still present, in which case fibrillation may begin again immediately.

When electrodes are applied directly to the two sides of the heart, fibrillation can usually be stopped using 110 volts of 60-cycle alternating current applied for 0.1

second or 1000 volts of direct current applied for a few thousandths of a second. When applied through two electrodes on the chest wall, as shown in Figure 13–17, the usual procedure is to charge a large electrical capacitor up to several thousand volts and then to cause the capacitor to discharge for a few thousandths of a second through the electrodes and through the heart. In our laboratory, the heart of a single anesthetized dog was defibrillated 130 times through the chest wall, and the animal lived thereafter in perfectly normal condition.

Hand Pumping of the Heart (Cardiopulmonary Resuscitation) as an Aid to Defibrillation

Unless defibrillated within 1 minute after fibrillation begins, the heart is usually too weak to be revived by defibrillation because of the lack of nutrition from coronary blood flow. However, it is still possible to revive the heart by preliminarily pumping the heart by hand (intermittent hand squeezing) and then defibrillating the heart later. In this way, small quantities of blood are delivered into the aorta and a renewed coronary blood supply develops. Then, after a few minutes of hand pumping, electrical defibrillation often becomes possible. Indeed, fibrillating hearts have been pumped by hand for as long as 90 minutes followed by successful defibrillation.

A technique for pumping the heart without opening the chest consists of intermittent thrusts of pressure on the chest wall along with artificial respiration. This, plus defibrillation, is called *cardiopulmonary resuscitation,* or CPR.

Lack of blood flow to the brain for more than 5 to 8 minutes usually causes permanent mental impairment or even destruction of brain tissue. Even if the heart is revived, the person may die from the effects of brain damage or may live with permanent mental impairment.

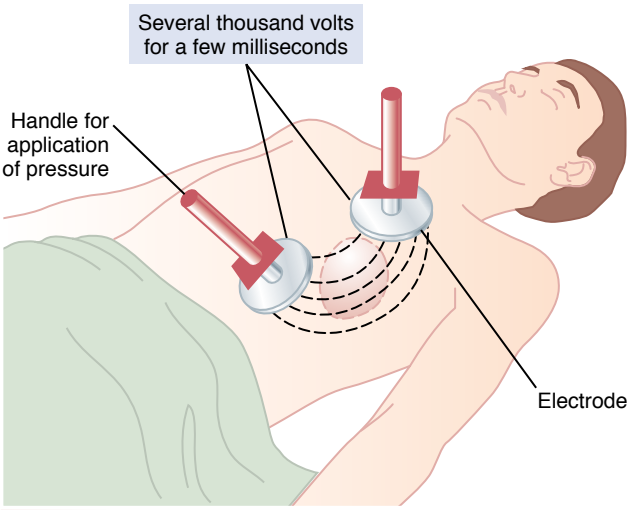


Figure 13–17

Application of electrical current to the chest to stop ventricular fibrillation.

Atrial Fibrillation

Remember that except for the conducting pathway through the A-V bundle, the atrial muscle mass is separated from the ventricular muscle mass by fibrous tissue. Therefore, ventricular fibrillation often occurs without atrial fibrillation. Likewise, fibrillation often occurs in the atria without ventricular fibrillation (shown to the right in Figure 13–19).

The mechanism of atrial fibrillation is identical to that of ventricular fibrillation, except that the process occurs only in the atrial muscle mass instead of the ventricular mass. A frequent cause of atrial fibrillation is atrial enlargement resulting from heart valve lesions that prevent the atria from emptying adequately into the ventricles, or from ventricular failure with excess damming of blood in the atria. The dilated atrial walls provide ideal conditions of a long conductive pathway as well as slow conduction, both of which predispose to atrial fibrillation.

Pumping Characteristics of the Atria During Atrial Fibrillation. For the same reasons that the ventricles will not pump blood during ventricular fibrillation, neither do the atria pump blood in atrial fibrillation. Therefore, the atria become useless as primer pumps for the ventricles. Even so, blood flows passively through the atria into the ventricles, and the efficiency of ventricular pumping is decreased only 20 to 30 per cent. Therefore, in contrast to the lethality of ventricular fibrillation, a person can live for months or even years with atrial fibrillation, although at reduced efficiency of overall heart pumping.

Electrocardiogram in Atrial Fibrillation. Figure 13–18 shows the electrocardiogram during atrial fibrillation. Numerous small depolarization waves spread in all directions through the atria during atrial fibrillation. Because the waves are weak and many of them are of opposite polarity at any given time, they usually almost completely electrically neutralize one another. Therefore, in the electrocardiogram, one can see either no P waves from the atria or only a fine, high-frequency, very low voltage wavy record. Conversely, the QRS-T complexes are normal unless there is some pathology of the ventricles, but their timing is irregular, as explained next.

Irregularity of Ventricular Rhythm During Atrial Fibrillation. When the atria are fibrillating, impulses arrive from the atrial muscle at the A-V node rapidly but also irregularly. Because the A-V node will not pass a second impulse for about 0.35 second after a previous one, at least 0.35 second must elapse between one ventricular contraction and the next. Then an additional but

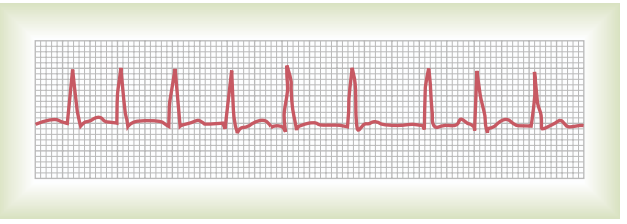


Figure 13–18

Atrial fibrillation (lead I). The waves that can be seen are ventricular QRS and T waves.

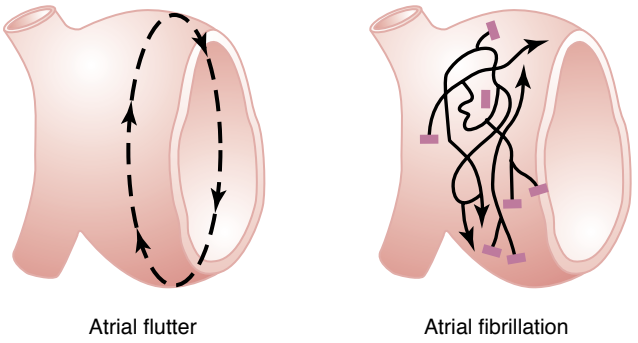


Figure 13–19

Pathways of impulses in atrial flutter and atrial fibrillation.

variable interval of 0 to 0.6 second occurs before one of the irregular atrial fibrillatory impulses happens to arrive at the A-V node. Thus, the interval between successive ventricular contractions varies from a minimum of about 0.35 second to a maximum of about 0.95 second, causing a very irregular heartbeat. In fact, this irregularity, demonstrated by the variable spacing of the heartbeats in the electrocardiogram of Figure 13–18, is one of the clinical findings used to diagnose the condition. Also, because of the rapid rate of the fibrillatory impulses in the atria, the ventricle is driven at a fast heart rate, usually between 125 and 150 beats per minute.

Electroshock Treatment of Atrial Fibrillation. In the same manner that ventricular fibrillation can be converted back to a normal rhythm by electroshock, so too can atrial fibrillation be converted by electroshock. The procedure is essentially the same as for ventricular fibrillation conversion—passage of a single strong electric shock through the heart, which throws the entire heart into refractoriness for a few seconds; a normal rhythm often follows *if the heart is capable of this.*

Atrial Flutter

Atrial flutter is another condition caused by a circus movement in the atria. It is different from atrial fibrillation, in that the electrical signal travels as a single large wave always in one direction around and around the atrial muscle mass, as shown to the left in Figure 13–19. Atrial flutter causes a rapid rate of contraction of the atria, usually between 200 and 350 beats per minute. However, because one side of the atria is contracting while the other side is relaxing, the amount of blood pumped by the atria is slight. Furthermore, the signals reach the A-V node too rapidly for all of them to be passed into the ventricles, because the refractory periods of the A-V node and A-V bundle are too long to pass more than a fraction of the atrial signals. Therefore, there are usually two to three beats of the atria for every single beat of the ventricles.

Figure 13–20 shows a typical electrocardiogram in atrial flutter. The P waves are strong because of contraction of semicoordinate masses of muscle. However, note in the record that a QRS-T complex follows an atrial P wave only once for every two to three beats of the atria, giving a 2:1 or 3:1 rhythm.

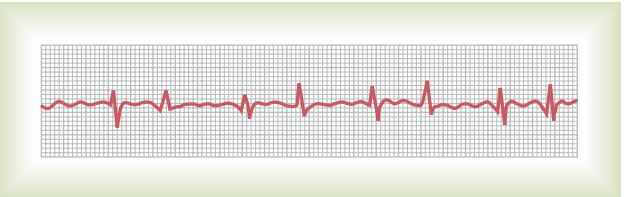


Figure 13–20

Atrial flutter—2:1 and 3:1 atrial to ventricle rhythm (lead I).

Cardiac Arrest

A final serious abnormality of the cardiac rhythmicity-conduction system is *cardiac arrest.* This results from cessation of all electrical control signals in the heart. That is, no spontaneous rhythm remains.

Cardiac arrest is especially likely to occur *during deep anesthesia,* when many patients develop severe hypoxia because of inadequate respiration. The hypoxia prevents the muscle fibers and conductive fibers from maintaining normal electrolyte concentration differentials across their membranes, and their excitability may be so affected that the automatic rhythmicity disappears.

In most instances of cardiac arrest from anesthesia, prolonged cardiopulmonary resuscitation (many minutes or even hours) is quite successful in reestablishing a normal heart rhythm. In some patients, severe myocardial disease can cause permanent or semipermanent cardiac arrest, which can cause death. To treat the condition, rhythmical electrical impulses from an *implanted electronic cardiac pacemaker* have been used successfully to keep patients alive for months to years.

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