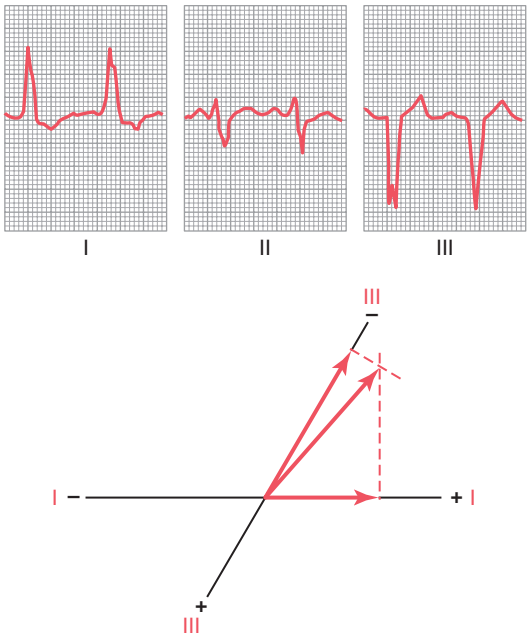

Figure 12-14. Left axis deviation caused by *left bundle branch block*. Note also the greatly prolonged QRS complex.

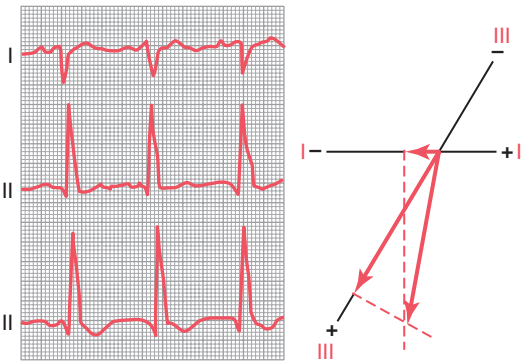

Figure 12-15. Right axis deviation caused by *right bundle branch block*. Note also the greatly prolonged QRS complex.

right axis deviation occurs. In **Figure 12-15,** right axis deviation caused by right bundle branch block is demonstrated and its vector is analyzed; this analysis shows an axis of about 105 degrees instead of the normal 59 degrees and a prolonged QRS complex because of slow conduction.

CONDITIONS THAT CAUSE ABNORMAL VOLTAGES OF THE QRS COMPLEX

INCREASED VOLTAGE IN THE STANDARD BIPOLAR LIMB LEADS

Normally, the voltages in the three standard bipolar limb leads, as measured from the peak of the R wave to the bottom of the S wave, vary between 0.5 and 2.0 millivolts, with lead III usually recording the lowest voltage and lead II the highest voltage. However, these relations are not invariable, even for the normal heart. In general, when the sum of the voltages of all the QRS complexes of the three standard leads is greater than 4 millivolts, the patient is considered to have a high-voltage ECG.

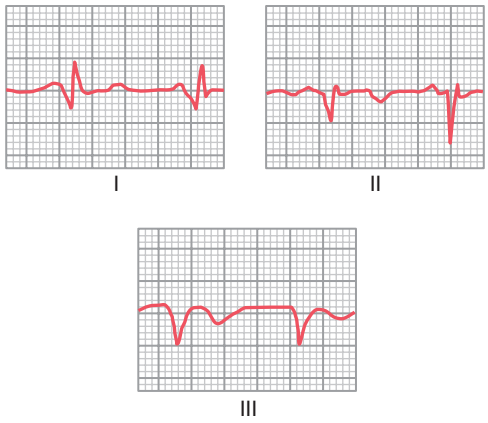

Figure 12-16. A low-voltage electrocardiogram following local damage throughout the ventricles caused by *previous myocardial infarction*.

The cause of high-voltage QRS complexes most often is increased muscle mass of the heart, which ordinarily results from *hypertrophy of the muscle* in response to excessive load on one part of the heart or the other. For example, the right ventricle hypertrophies when it must pump blood through a stenotic pulmonary valve, and the left ventricle hypertrophies when a person has high blood pressure. The increased quantity of muscle generates increased electricity around the heart. As a result, the electrical potentials recorded in the electrocardiographic leads are considerably greater than normal, as shown in **Figures 12-12** and **12-13**.

DECREASED VOLTAGE OF THE ELECTROCARDIOGRAM

Decreased Voltage Caused by Cardiac Myopathies. One of the most common causes of decreased voltage of the QRS complex is a series of *old myocardial infarctions* with resultant *diminished muscle mass.* This condition also causes the depolarization wave to move through the ventricles slowly and prevents major portions of the heart from becoming massively depolarized all at once. Consequently, this condition causes some prolongation of the QRS complex along with the decreased voltage. **Figure 12-16** shows a typical low-voltage ECG with prolongation of the QRS complex, which is common after multiple small infarctions of the heart have caused local delays of impulse conduction and reduced voltages due to loss of muscle mass throughout the ventricles.

Decreased Voltage Caused by Conditions Surrounding the Heart. One of the most important causes of decreased voltage in electrocardiographic leads is *fluid*

in the pericardium. Because extracellular fluid conducts electrical currents with great ease, a large portion of the electricity flowing out of the heart is conducted from one part of the heart to another through the pericardial fluid. Thus, this effusion effectively "short-circuits" the electrical potentials generated by the heart, decreasing the electrocardiographic voltages that reach the outside surfaces of the body. *Pleural effusion,* to a lesser extent, also can "short circuit" the electricity around the heart so that the voltages at the surface of the body and in the ECGs are decreased.

Pulmonary emphysema can decrease the electrocardiographic potentials, but for a different reason than that of pericardial effusion. In persons with pulmonary emphysema, conduction of electrical current through the lungs is depressed considerably because of an excessive quantity of air in the lungs. Also, the chest cavity enlarges, and the lungs tend to envelop the heart to a greater extent than is normal. Therefore, the lungs act as an insulator to prevent spread of electrical voltage from the heart to the surface of the body, which results in decreased electrocardiographic potentials in the various leads.

PROLONGED AND BIZARRE PATTERNS OF THE QRS COMPLEX

CARDIAC HYPERTROPHY OR DILATION PROLONG THE QRS COMPLEX

The QRS complex lasts as long as depolarization continues to spread through the ventricles—that is, as long as part of the ventricles is depolarized and part is still polarized. Therefore, *prolonged conduction* of the impulse through the ventricles always causes a prolonged QRS complex. Such prolongation often occurs when one or both ventricles are hypertrophied or dilated, owing to the longer pathway that the impulse must then travel. The normal QRS complex lasts 0.06 to 0.08 second, whereas in hypertrophy or dilation of the left or right ventricle, the QRS complex may be prolonged to 0.09 to 0.12 second.

PURKINJE SYSTEM BLOCK PROLONGS THE QRS COMPLEX

When the Purkinje fibers are blocked, the cardiac impulse must then be conducted by the ventricular muscle instead of by way of the Purkinje system. This action decreases the velocity of impulse conduction to about one third of normal. Therefore, if complete block of one of the bundle branches occurs, the duration of the QRS complex is usually increased to 0.14 second or greater.

In general, a QRS complex is considered to be abnormally long when it lasts more than 0.09 second; when it lasts more than 0.12 second, the prolongation is almost certainly caused by a pathological block somewhere in the ventricular conduction system, as shown by the ECGs for bundle branch block in **Figures 12-14** and **12-15**.

CONDITIONS THAT CAUSE BIZARRE QRS COMPLEXES

Bizarre patterns of the QRS complex most frequently are caused by two conditions: (1) destruction of cardiac muscle in various areas throughout the ventricular system, with replacement of this muscle by scar tissue, and (2) multiple small local blocks in the conduction of impulses at many points in the Purkinje system. As a result, cardiac impulse conduction becomes irregular, causing rapid shifts in voltages and axis deviations. This irregularity often causes double or even triple peaks in some of the electrocardiographic leads, such as those shown in **Figure 12-14**.

CURRENT OF INJURY

Many different cardiac abnormalities, especially those that damage the heart muscle, often cause part of the heart to remain partially or totally *depolarized all the time.* When this condition occurs, current flows between the pathologically depolarized and the normally polarized areas, even between heartbeats. This condition is called a *current of injury.* Note especially that *the injured part of the heart is negative, because this is the part that is depolarized and emits negative charges into the surrounding fluids, whereas the remainder of the heart is neutral or in positive polarity.*

Some abnormalities that can cause current of injury are (1) *mechanical trauma,* which sometimes makes the membranes remain so permeable that full repolarization cannot take place; (2) *infectious processes* that damage the muscle membranes; and (3) *ischemia of local areas of heart muscle caused by local coronary occlusions,* which is by far the most common cause of current of injury in the heart. During ischemia, not enough nutrients from the coronary blood supply are available to the heart muscle to maintain normal membrane polarization.

EFFECT OF CURRENT OF INJURY ON THE QRS COMPLEX

In **Figure 12-17**, a small area in the base of the left ventricle is newly infarcted (i.e., there is loss of coronary blood flow). Therefore, during the T-P interval—that is, when the normal ventricular muscle is totally polarized—abnormal *negative* current still flows from the infarcted area at the base of the left ventricle and spreads toward the rest of the ventricles.

The vector of this "current of injury," as shown in the first heart in **Figure 12-17**, is in a direction of about 125 degrees, with the base of the vector, the *negative end,*

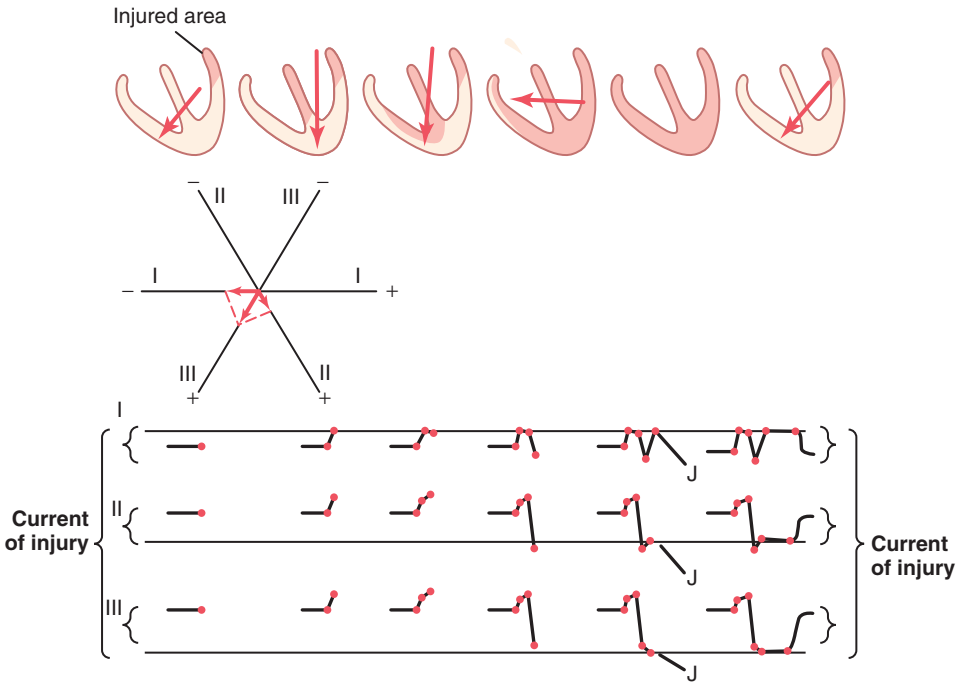

Figure 12-17. Effect of a current of injury on the electrocardiogram.

toward the injured muscle. As shown in the lower portions of the figure, even before the QRS complex begins, *this vector causes an initial record in lead I below the zero potential line,* because the projected vector of the current of injury in lead I points toward the negative end of the lead I axis. In lead II, the record is above the line because the projected vector points more toward the positive terminal of the lead. In lead III, the projected vector points in the same direction as the positive terminal of lead III so that the record is positive. Furthermore, because the vector lies almost exactly in the direction of the axis of lead III, the voltage of the current of injury in lead III is much greater than in either lead I or lead II.

As the heart then proceeds through its normal process of depolarization, the septum first becomes depolarized; then the depolarization spreads down to the apex and back toward the bases of the ventricles. The last portion of the ventricles to become totally depolarized is the base of the right ventricle, because the base of the left ventricle is already totally and permanently depolarized. By vectorial analysis, the successive stages of ECG generation by the depolarization wave traveling through the ventricles can be constructed graphically, as demonstrated in the lower part of **Figure 12-17**.

When the heart becomes totally depolarized, at the end of the depolarization process (as noted by the next-to-last stage in **Figure 12-17**), all the ventricular muscle is in a negative state. Therefore, at this instant in the ECG, no current flows from the ventricles to the electrocardiographic electrodes because now both the injured heart muscle and the contracting muscle are depolarized.

Next, as repolarization takes place, all of the heart finally repolarizes, except the area of permanent depolarization in the injured base of the left ventricle. Thus, repolarization causes a return of the current of injury in each lead, as noted at the far right in **Figure 12-17**.

THE "J POINT" IS THE ZERO REFERENCE POTENTIAL FOR ANALYZING CURRENT OF INJURY

One might think that the ECG machines could determine when no current is flowing around the heart. However, many stray currents exist in the body, such as currents resulting from "skin potentials" and from differences in ionic concentrations in different fluids of the body. Therefore, when two electrodes are connected between the arms or between an arm and a leg, these stray currents make it impossible to predetermine the exact zero reference level in the ECG.

For these reasons, the following procedure must be used to determine the zero potential level: First, one notes *the exact point at which the wave of depolarization just completes its passage through the heart,* which occurs at the end of the QRS complex. At exactly this point, all parts of the ventricles have become depolarized, including both the damaged parts and the normal parts, so no current is flowing around the heart. Even the current of injury disappears at this point. Therefore, the potential of the electrocardiogram at this instant is at zero voltage. This point is known as the "J point" in the ECG, as shown in **Figure 12-18**.

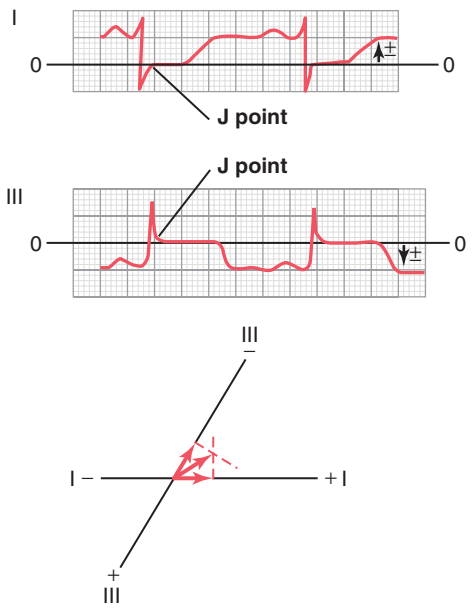

Figure 12-18. J point as the zero reference potential of the electrocardiograms for leads I and III. Also, the method for plotting the axis of the injury potential is shown in the lowermost panel.

Then, for analysis of the electrical axis of the injury potential caused by a current of injury, a horizontal line is drawn in the ECG for each lead at the level of the J point. This horizontal line is then the *zero potential level* in the ECG from which all potentials caused by currents of injury must be measured.

Use of the J Point in Plotting Axis of Injury Potential. Figure 12-18 shows ECGs (leads I and III) from an injured heart. Both records show injury potentials. In other words, the J point of each of these two ECGs is not on the same line as the T-P segment. In the figure, a horizontal line has been drawn through the J point to represent the zero voltage level in each of the two recordings. The injury potential in each lead is the difference between the voltage of the ECG immediately before onset of the P wave and the zero voltage level determined from the J point. In lead I, the recorded voltage of the injury potential is above the zero potential level and is therefore positive. Conversely, in lead III, the injury potential is below the zero voltage level and therefore is negative.

At the bottom in Figure 12-18, the respective injury potentials in leads I and III are plotted on the coordinates of these leads, and the resultant vector of the injury potential for the whole ventricular muscle mass is determined by vectorial analysis as described. In this instance, the resultant vector extends from the right side of the ventricles toward the left and slightly upward, with an axis of about –30 degrees. If one places this vector for the injury potential directly over the ventricles, *the negative end of the vector points toward the permanently depolarized, "injured" area of the ventricles.* In the example shown in Figure 12-18, the injured area would be in the lateral wall of the right ventricle.

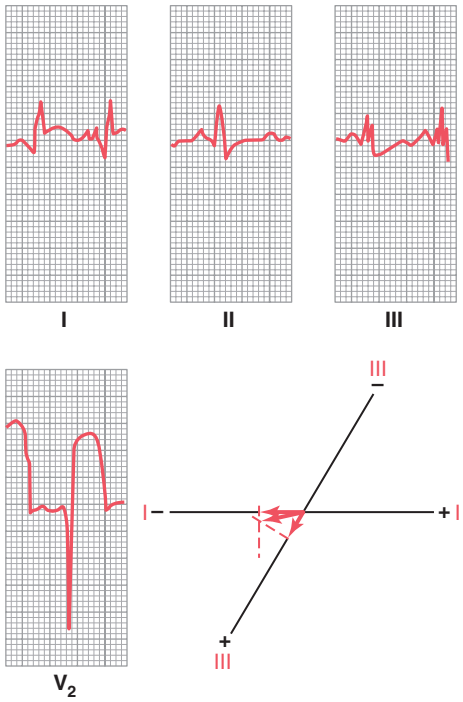

Figure 12-19. Current of injury in *acute anterior wall infarction*. Note the intense injury potential in lead V_2.

This analysis is obviously complex. However, it is essential that the student review it again and again until he or she understands it thoroughly. No other aspect of electrocardiographic analysis is more important.

CORONARY ISCHEMIA AS A CAUSE OF INJURY POTENTIAL

Insufficient blood flow to the cardiac muscle depresses the metabolism of the muscle for three reasons: (1) lack of oxygen, (2) excess accumulation of carbon dioxide, and (3) lack of sufficient food nutrients. Consequently, repolarization of the muscle membrane cannot occur in areas of severe myocardial ischemia. Often the heart muscle does not die because the blood flow is sufficient to maintain life of the muscle even though it is not sufficient to cause normal repolarization of the membranes. As long as this state exists, an injury potential continues to flow during the diastolic portion (the T-P portion) of each heart cycle.

Extreme ischemia of the cardiac muscle occurs after coronary occlusion and a strong current of injury flows from the infarcted area of the ventricles during the T-P interval between heartbeats, as shown in Figures 12-19 and 12-20. Therefore, one of the most important diagnostic features of ECGs recorded after acute coronary thrombosis is the current of injury.

Acute Anterior Wall Infarction. Figure 12-19 shows the ECG in the three standard bipolar limb leads and in one chest lead (lead V_2) recorded from a patient with acute anterior wall cardiac infarction. The most

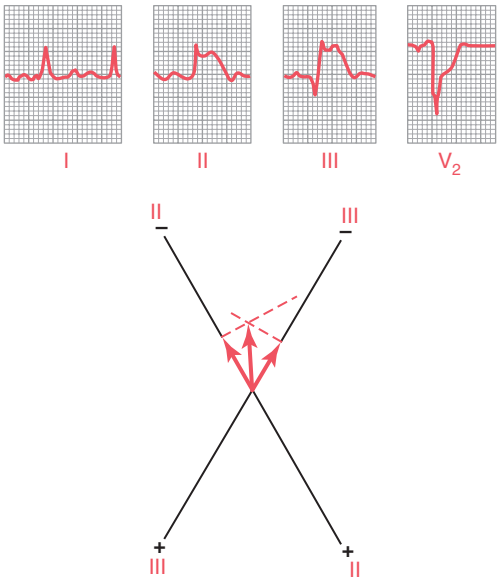

Figure 12-20. Injury potential in *acute posterior wall, apical infarction.*

important diagnostic feature of this ECG is the intense injury potential in chest lead V_2. If one draws a zero horizontal potential line through the J point of this ECG, a strong *negative* injury potential during the T-P interval is found, which means that the chest electrode over the front of the heart is in an area of strongly negative potential. In other words, the negative end of the injury potential vector in this heart is against the anterior chest wall. This means that the current of injury is emanating from the anterior wall of the ventricles, which diagnoses this condition as *anterior wall infarction*.

When analyzing the injury potentials in leads I and III, one finds a negative potential in lead I and a positive potential in lead III. This finding means that the resultant vector of the injury potential in the heart is about +150 degrees, with the negative end pointing toward the left ventricle and the positive end pointing toward the right ventricle. Thus, in this ECG, the current of injury is coming mainly from the left ventricle, as well as from the anterior wall of the heart. Therefore, one would conclude that this anterior wall infarction almost certainly is caused by thrombosis of the anterior descending branch of the left coronary artery.

Posterior Wall Infarction. **Figure 12-20** shows the three standard bipolar limb leads and one chest lead (lead V_2) from a patient with posterior wall infarction. The major diagnostic feature of this ECG is also in the chest lead. If a zero potential reference line is drawn through the J point of this lead, it is readily apparent that during the T-P interval, the potential of the current of injury is positive. This means that the positive end of the vector is in the direction of the anterior chest wall, and the negative end (the injured end of the vector) points away from the chest wall. In other words, the current of injury is coming from the back of the heart opposite to the anterior chest wall, which is the reason this type of ECG is the basis for diagnosing posterior wall infarction.

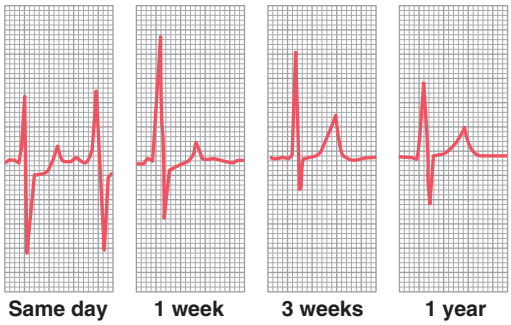

Figure 12-21. Recovery of the myocardium after *moderate posterior wall infarction*, demonstrating disappearance of the injury potential that is present on the first day after the infarction and still slightly present at 1 week.

If one analyzes the injury potentials from leads II and III of **Figure 12-20**, it is readily apparent that the injury potential is negative in both leads. By vectorial analysis, as shown in the figure, one finds that the resultant vector of the injury potential is about –95 degrees, with the negative end pointing downward and the positive end pointing upward. Thus, because the infarct, as indicated by the chest lead, is on the posterior wall of the heart and, as indicated by the injury potentials in leads II and III, is in the apical portion of the heart, one would suspect that this infarct is near the apex on the posterior wall of the left ventricle.

Infarction in Other Parts of the Heart. With use of the same procedures demonstrated in the preceding discussions of anterior and posterior wall infarctions, it is possible to determine the locus of any infarcted area emitting a current of injury, regardless of which part of the heart is involved. In making such vectorial analyses, it should be remembered that *the positive end of the injury potential vector points toward the normal cardiac muscle, and the negative end points toward the injured portion of the heart that is emitting the current of injury.*

Recovery from Acute Coronary Thrombosis. **Figure 12-21** shows a V3 chest lead from a patient with acute posterior wall infarction, demonstrating changes in the ECG from the day of the attack to 1 week later, 3 weeks later, and finally 1 year later. From this ECG, one can see that the injury potential is strong immediately after the acute attack (the T-P segment is displaced positively from the S-T segment). However, after about 1 week, the injury potential has diminished considerably, and after 3 weeks, it is gone. After that, the ECG does not change greatly during the next year. This is the usual recovery pattern after acute myocardial infarction of moderate degree, showing that the *new collateral coronary blood flow* develops enough to re-establish appropriate nutrition to most of the infarcted area.

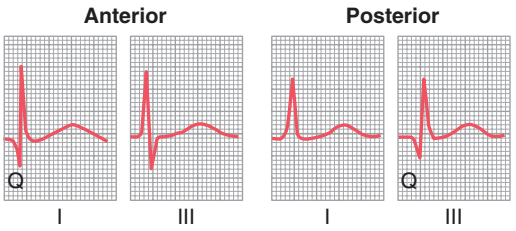

Figure 12-22. Electrocardiograms of anterior and posterior wall infarctions that occurred about 1 year previously, showing a Q wave in lead I in anterior wall infarction and a Q wave in lead III in *posterior wall infarction.*

In some patients who experience myocardial infarction, the infarcted area never redevelops adequate coronary blood supply. Often, some of the heart muscle dies, but if the muscle does not die, it will continue to show an injury potential as long as the ischemia exists, particularly during bouts of exercise when the heart is overloaded.

Old Recovered Myocardial Infarction. Figure 12-22 shows leads I and III after *anterior infarction* and leads I and III after *posterior infarction* about 1 year after the acute heart attack. The records show what might be called the "ideal" configurations of the QRS complex in these types of recovered myocardial infarction. Usually a Q wave has developed at the beginning of the QRS complex in lead I in anterior infarction because of loss of muscle mass in the anterior wall of the left ventricle, but in posterior infarction, a Q wave has developed at the beginning of the QRS complex in lead III because of loss of muscle in the posterior apical part of the ventricle.

These configurations are certainly not found in all cases of old cardiac infarction. Local loss of muscle and local points of cardiac signal conduction block can cause very bizarre QRS patterns (especially prominent Q waves, for instance), decreased voltage, and QRS prolongation.

Current of Injury in Angina Pectoris. "Angina pectoris" means pain from the heart felt in the pectoral regions of the upper chest. This pain usually also radiates into the left neck area and down the left arm. The pain is typically caused by moderate ischemia of the heart. Usually, no pain is felt as long as the person is quiet, but as soon as he or she overworks the heart, the pain appears.

An injury potential sometimes appears in the ECG during an attack of severe angina pectoris because the coronary insufficiency becomes great enough to prevent adequate repolarization of some areas of the heart during diastole.

ABNORMALITIES IN THE T WAVE

Earlier in the chapter, we pointed out that the T wave is normally positive in all the standard bipolar limb leads and that this is caused by repolarization of the apex and outer surfaces of the ventricles ahead of the intraventricular surfaces. That is, the T wave becomes abnormal when the normal sequence of repolarization does not occur. Several factors can change this sequence of repolarization.

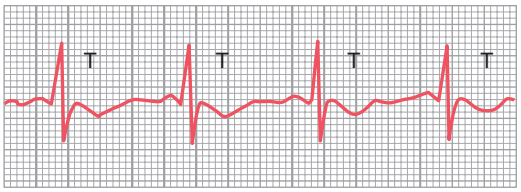

Figure 12-23. An inverted T wave resulting from mild *ischemia at the base* of the ventricles.

EFFECT OF SLOW CONDUCTION OF THE DEPOLARIZATION WAVE ON THE CHARACTERISTICS OF THE T WAVE

Referring to Figure 12-14, note that the QRS complex is considerably prolonged. The reason for this prolongation is *delayed conduction in the left ventricle* resulting from left bundle branch block. This delayed conduction causes the left ventricle to become depolarized about 0.08 second after depolarization of the right ventricle, which gives a strong mean QRS vector *to the left.* However, the refractory periods of the right and left ventricular muscle masses are not greatly different from each other. Therefore, the right ventricle begins to repolarize long before the left ventricle, which causes strong positivity in the right ventricle and negativity in the left ventricle at the time that the T wave is developing. In other words, the mean axis of the T wave is now deviated *to the right,* which is opposite the mean electrical axis of the QRS complex in the same ECG. Thus, when conduction of the depolarization impulse through the ventricles is greatly delayed, the T wave is almost always of opposite polarity to that of the QRS complex.

SHORTENED DEPOLARIZATION IN PORTIONS OF THE VENTRICULAR MUSCLE CAN CAUSE T-WAVE ABNORMALITIES

If the base of the ventricles should exhibit an abnormally short period of depolarization—that is, a shortened action potential—repolarization of the ventricles would not begin at the apex as it normally does. Instead, the base of the ventricles would repolarize ahead of the apex, and the vector of repolarization would point from the apex toward the base of the heart, opposite to the standard vector of repolarization. Consequently, the T wave in all three standard leads would be negative rather than the usual positive. Thus, the simple fact that the base of the ventricles has a shortened period of depolarization is sufficient to cause marked changes in the T wave, even to the extent of changing the entire T-wave polarity, as shown in Figure 12-23.

Mild ischemia is by far the most common cause of shortening of depolarization of cardiac muscle because this condition increases current flow through the potassium channels. When the ischemia occurs in only one area of the heart, the depolarization period of this area decreases out of proportion to that in other portions. As a result, definite changes in the T wave can take place. The ischemia might result from chronic, progressive coronary occlusion, acute coronary occlusion, or relative coronary insufficiency that occurs during exercise.

One means for detecting mild coronary insufficiency is to have the patient exercise and to record the ECG, noting whether changes occur in the T waves. The changes in the T waves need not be specific because any change in the T wave in any lead—inversion, for instance, or a biphasic wave—is often evidence enough that some portion of the ventricular muscle has a period of depolarization out of proportion to the rest of the heart, caused by mild to moderate coronary insufficiency.

Effect of Digitalis on the T Wave. As discussed in Chapter 22, digitalis is a drug that can be used during coronary insufficiency to increase the strength of cardiac muscle contraction. However, when an overdose of digitalis is given, depolarization duration in one part of the ventricles may be increased out of proportion to that of other parts. As a result, nonspecific changes, such as T-wave inversion or biphasic T waves, may occur in one or more of the electrocardiographic leads. A biphasic T wave caused by excessive administration of digitalis is shown in **Figure 12-24**. Therefore, changes in the T wave during digitalis administration are often the earliest signs of digitalis toxicity.

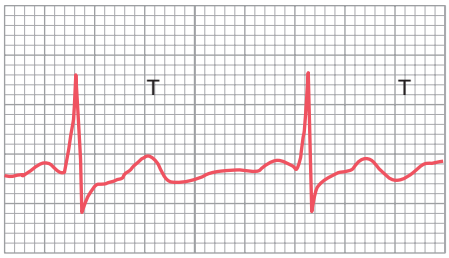

Figure 12-24. A biphasic T wave caused by *digitalis toxicity*.

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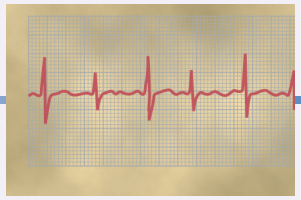
See the bibliography for Chapter 13.



CHAPTER 13

UNIT III

Cardiac Arrhythmias and Their Electrocardiographic Interpretation

Some of the most distressing types of heart malfunction occur 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 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:

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

ABNORMAL SINUS RHYTHMS

TACHYCARDIA

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

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

The heart rate usually increases about 10 beats/min for each degree (Fahrenheit) increase in body temperature (with an increase of 18 beats/min per degree Celsius), 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 sustains severe blood loss, sympathetic reflex stimulation of the heart may increase the heart rate to 150 to 180 beats/min.

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 phenomenon causes reductions in blood pressure and elicits sympathetic reflexes to increase the heart rate.

BRADYCARDIA

The term "bradycardia" means a slow heart rate, usually defined as fewer than 60 beats/min. Bradycardia is shown by the ECG in **Figure 13-2**.

Bradycardia in Athletes. The well-trained athlete's heart is often 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.

Vagal Stimulation Causes 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 phenomenon 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.

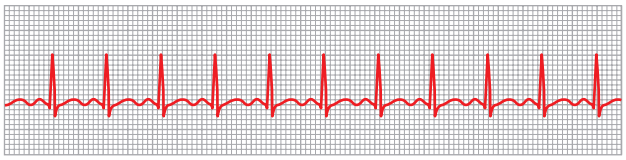


Figure 13-1. Sinus tachycardia (lead I).

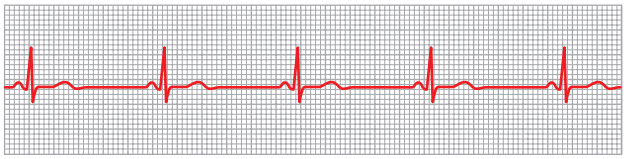


Figure 13-2. Sinus bradycardia (lead III).

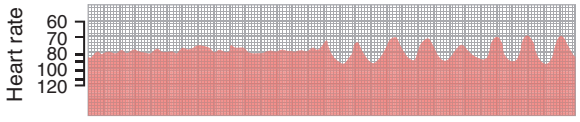


Figure 13-3. Sinus arrhythmia as recorded by a cardiotachometer. To the left is the record when the subject was breathing normally; to the right, when the subject was breathing deeply.

SINUS ARRHYTHMIA

Figure 13-3 shows a *cardiotachometer* recording of the heart rate, at first during normal respiration 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 ECG. Note from this record that the heart rate increased and decreased no more than 5 percent during quiet respiration (shown on the left half of the record). Then, *during deep respiration,* the heart rate increased and decreased with each respiratory cycle by as much as 30 percent.

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. The "respiratory" type of sinus arrhythmia, as shown in **Figure 13-3**, 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 an alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart.

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

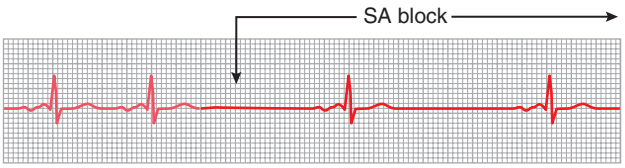


Figure 13-4. Sinoatrial *(SA)* nodal block, with atrioventricular nodal rhythm during the block period (lead III).

sudden cessation of P waves, with resultant standstill of the atria. However, the ventricles pick up a new rhythm, with the impulse usually originating spontaneously in the atrioventricular (A-V) node, so 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 conduction from the atria to the ventricles. Inflammation results frequently from different types of myocarditis that are 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 the *beginning* of the P wave and the *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 a faster heartbeat and increases with a 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 ECG with a prolonged P-R interval; the interval in this instance is about 0.30 second

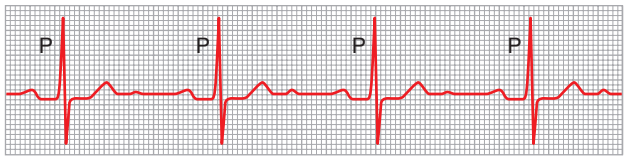


Figure 13-5. Prolonged P-R interval caused by first-degree atrioventricular heart block (lead II).

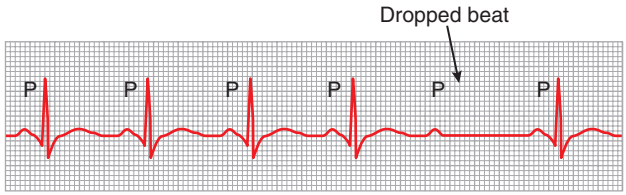


Figure 13-6. Second-degree atrioventricular block, showing occasional failure of the ventricles to receive the excitatory signals (lead V3).

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, such as *acute rheumatic heart disease,* for example, 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 is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough to do so. 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.*

There are two types of second-degree A-V block: type I (also known as *Wenckebach periodicity*) and type II. Type I block is characterized by progressive prolongation of the PR interval until a ventricular beat is dropped and is then followed by resetting of the PR and repeating of the abnormal cycle. A type I block is almost always caused by abnormality of the A-V node. In most cases, this type of block is benign and no specific treatment is needed.

In type II block there is usually a fixed number of nonconducted P waves for every QRS complex. For example, a 2:1 block implies that there are two P waves for every QRS complex. At other times, rhythms of 3:2 or 3:1 may develop. Type II block is generally caused by an abnormality of the bundle of His-Purkinje system and may require implantation of a pacemaker to prevent progression to complete heart block and cardiac arrest.

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.

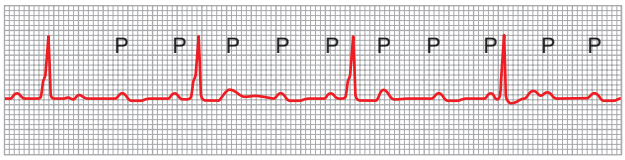


Figure 13-7. Complete atrioventricular block (lead II).

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 from the atria into the ventricles occurs. In this case, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle distal to the block. 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 ECG 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 are beating at their own natural rate, controlled most often by rhythmical signals generated distal to the A-V node or A-V bundle where the block occurs.

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 delay results from the phenomenon called *overdrive suppression.* Overdrive suppression means that ventricular excitability is at first suppressed 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 phenomenon is called *ventricular escape.*

Because the brain cannot remain active for more than 4 to 7 seconds without blood supply, most people 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 (typically beating less than 40 beats per minute) 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.*

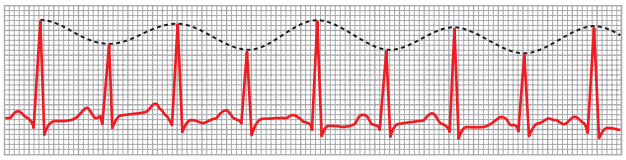


Figure 13-8. Partial intraventricular block—"electrical alternans" (lead II).

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 to 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 ECG 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 infection, 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.

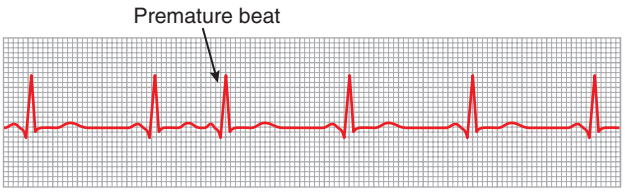


Figure 13-9. Atrial premature beat (lead I).

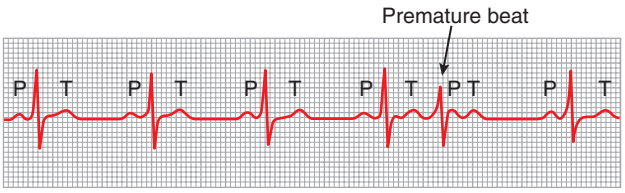


Figure 13-10. Atrioventricular nodal premature contraction (lead III).

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 compensatory pause 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, which 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 of the premature contraction. Instead, the P wave is superimposed onto the QRS-T complex because the cardiac

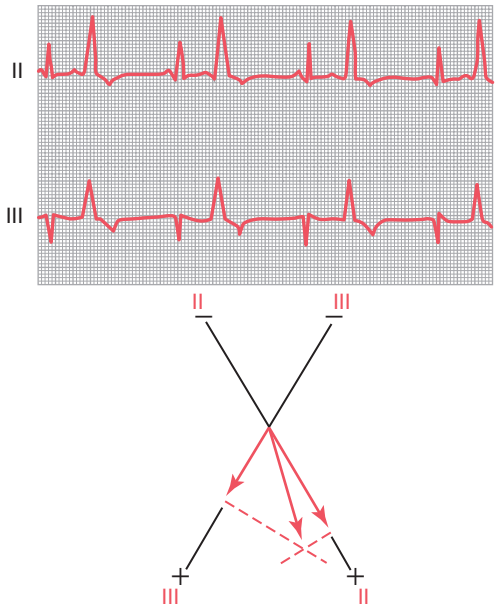


Figure 13-11. Premature ventricular contractions (PVCs) demonstrated by the large abnormal QRS-T complexes (leads II and III). The axis of the premature contractions is plotted in accordance with the principles of vectorial analysis explained in Chapter 12 and shows the origin of the PVC to be near the base of the ventricles.

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

1. *The QRS complex is usually considerably prolonged.* The reason for this prolongation 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.* 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 ECG. When a PVC occurs, the impulse almost always travels in only one direction, so there is no such neutralization effect, and one entire side or end of the ventricles is depolarized ahead of the other, which 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, excessive intake of coffee, lack of sleep, various mild toxic states, and even emotional irritability. Conversely, many other PVCs result from stray impulses or re-entrant 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. People with significant numbers of PVCs often have a much higher than normal risk of developing spontaneous lethal ventricular fibrillation, presumably initiated by one of the PVCs. This development 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 this chapter.

Vector Analysis of the Origin of an Ectopic Premature Ventricular Contraction. In Chapter 12, the principles of vectorial analysis are explained. By applying these principles, one can determine from the ECG 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. Upon 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.

Disorders of Cardiac Repolarization—The Long QT Syndromes. Recall that the Q wave corresponds to ventricular depolarization, whereas the T wave corresponds to ventricular repolarization. The Q-T interval is the time from the Q point to the end of the T wave. Disorders that delay repolarization of ventricular muscle after the action potential cause prolonged ventricular action potentials and therefore excessively long Q-T intervals on the ECG, a condition called *long QT syndrome* (LQTS).

The major reason LQTS is of concern is that delayed repolarization of ventricular muscle increases a person's susceptibility to developing ventricular arrhythmias called *torsades de pointes,* which literally means "twisting of the points." This type of arrhythmia has the features shown in **Figure 13-12**. The shape of the QRS complex may change over time with the onset of arrhythmia usually following a premature beat, a pause, and then another beat with a long Q-T interval, which may trigger arrhythmias, tachycardia, and in some instances ventricular fibrillation.

Disorders of cardiac repolarization that lead to LQTS may be inherited or acquired. The congenital forms of

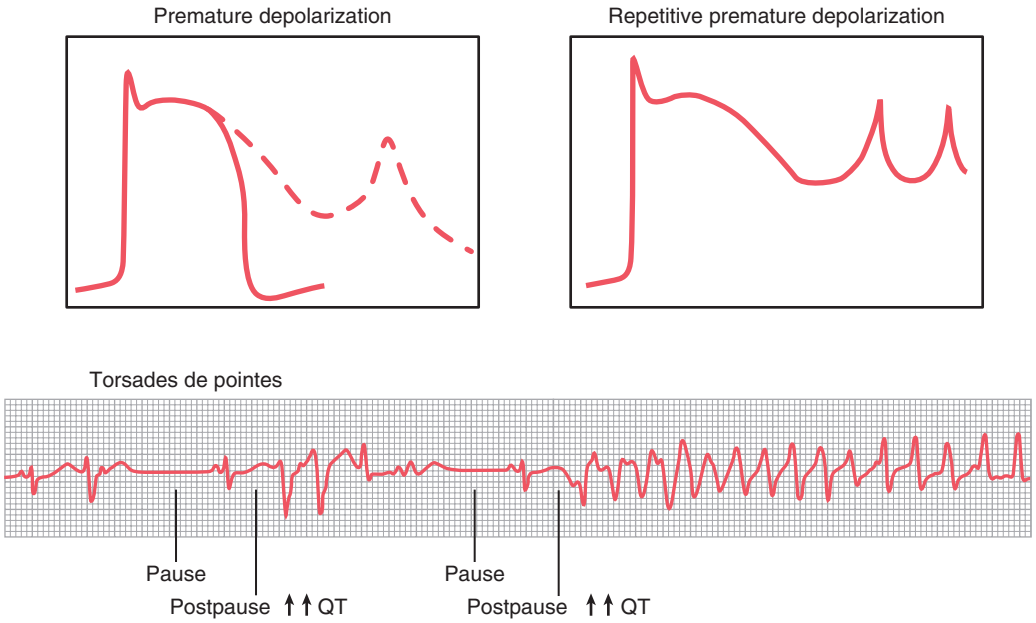


Figure 13-12. Development of arrhythmias in long QT syndrome (LQTS). When the ventricular muscle fiber action potential is prolonged as a result of delayed repolarization, a premature depolarization *(dashed line in top left figure)* may occur before complete repolarization. Repetitive premature depolarizations *(top right figure)* may lead to multiple depolarizations under certain conditions. In torsades de pointes *(bottom figure)*, premature ventricular beats lead pauses, postpause prolongation of the Q-T interval, and arrhythmias. *(Modified from Murray KT, Roden DM: Disorders of cardiac repolarization: the long QT syndromes. In: Crawford MG, DiMarco JP [eds]: Cardiology. London: Mosby, 2001.)*

LQTS are rare disorders caused by mutations of sodium or potassium channel genes. At least 10 different mutations of these genes causing variable degrees of Q-T prolongation have been identified.

More common are the acquired forms of LQTS that are associated with plasma electrolyte disturbances, such as hypomagnesemia, hypokalemia, or hypocalcemia, or with administration of excess amounts of antiarrhythmic drugs such as quinidine or some antibiotics such as fluoroquinolones or erythromycin that prolong the Q-T interval.

Although some people with LQTS show no major symptoms (other than the prolonged Q-T interval), other people exhibit fainting and ventricular arrhythmias that may be precipitated by physical exercise, intense emotions such as fright or anger, or being startled by a noise. The ventricular arrhythmias associated with LQTS can, in some cases, deteriorate into ventricular fibrillation and sudden death.

Treatment may include magnesium sulfate for acute LQTS and antiarrhythmia medications such as beta-adrenergic blockers or surgical implantation of a cardiac defibrillator for long-term LQTS.

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 phenomenon is believed to be caused most frequently by 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.

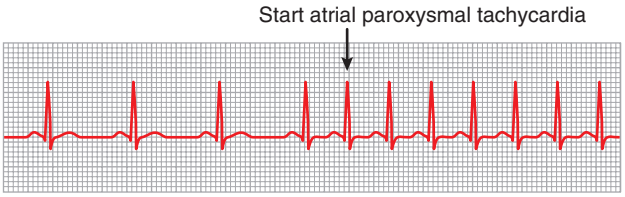


Figure 13-13. Atrial paroxysmal tachycardia—onset in the middle of the record (lead I).

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. 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. Antiarrhythmic drugs may also be used to slow conduction or prolong the refractory period in cardiac tissues.

ATRIAL PAROXYSMAL TACHYCARDIA

Figure 13-13 demonstrates a sudden increase in the heart rate from about 95 to about 150 beats per minute

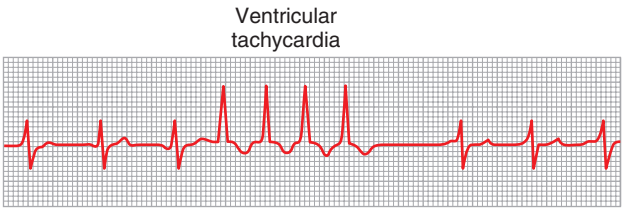


Figure 13-14. Ventricular paroxysmal tachycardia (lead III).

in the middle of the record. On close study of the ECG, an inverted P wave is seen during the rapid heartbeat before each QRS-T complex, and this P wave is partially superimposed onto the normal T wave of the preceding beat. This finding 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 involving the A-V node that 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 it usually does not cause permanent harm from the attack.

VENTRICULAR PAROXYSMAL TACHYCARDIA

Figure 13-14 shows a typical short paroxysm of ventricular tachycardia. The ECG 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. Antiarrhythmic drugs such as *amiodarone* or *lidocaine* can be used to treat ventricular tachycardia. Lidocaine 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. Amiodarone has multiple actions such as prolonging the action potential and refractory period in cardiac muscle and slowing A-V conduction. In some cases, *cardioversion* with an electric shock to the heart is needed for restoration of normal heart rhythm.

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 phenomenon occurs, 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 coordinated 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 because of 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 13-15 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. However,

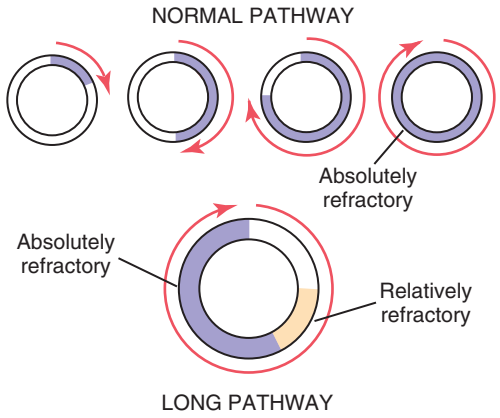


Figure 13-15. Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

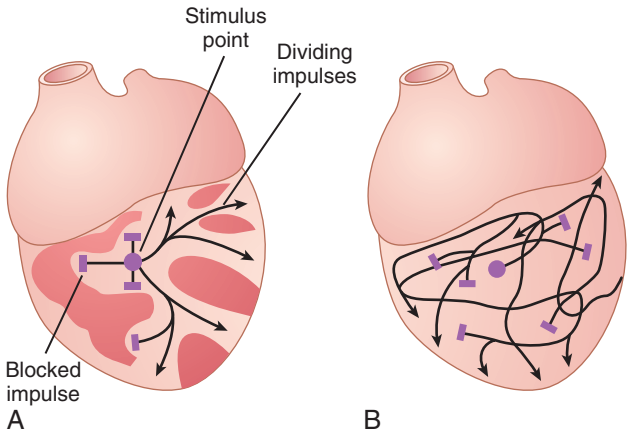


Figure 13-16. A, Initiation of fibrillation in a heart when patches of refractory musculature are present. **B,** Continued propagation of *fibrillatory impulses* in the fibrillating ventricle.

three different conditions 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 (circus movement).

First, if the *pathway around the circle is much longer than normal,* 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 blockage of the Purkinje system, ischemia of the muscle, high blood potassium levels, or 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 13-15**. 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 with a 60-cycle alternating electric current.

Fibrillation Caused by 60-Cycle Alternating Current. At a central point in the ventricles of heart A in **Figure 13-16,** 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 portions. This state of events is depicted in heart A by many lighter patches, which represent excitable cardiac muscle, and dark patches, which represent muscle that is still refractory. 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 they are blocked. However, 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, 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, and (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 in **Figure 13-16**. 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 impulses reaches another refractory area, it 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, many small depolarization waves are 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 impulses 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 13-16** demonstrates the final state that develops in fibrillation. Here one can see many impulses traveling in all directions, with some dividing and increasing the number of impulses and others 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 ECG is bizarre (**Figure 13-17**) 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, which causes coarse, irregular waves in the ECG. After another few seconds, the coarse contractions of the ventricles disappear, and the ECG 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.

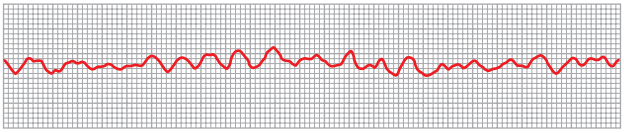


Figure 13-17. Ventricular fibrillation (lead II).

The voltages of the waves in the ECG in ventricular fibrillation are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly, and thus 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 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 feat 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, if the same re-entrant focus that had originally thrown the ventricles into fibrillation is still present, fibrillation may begin again immediately.

When electrodes are applied directly to the two sides of the heart, fibrillation can usually be stopped using 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 13-18**, 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 most cases, defibrillation current is delivered to the heart in biphasic waveforms, alternating the direction of the current pulse through the heart. This form of delivery substantially reduces the energy needed for successful defibrillation, thereby decreasing the risk for burns and cardiac damage.

In patients with high risk for ventricular fibrillation, a small battery-powered implantable cardioverter-defibrillator (ICD) with electrode wires lodged in the right

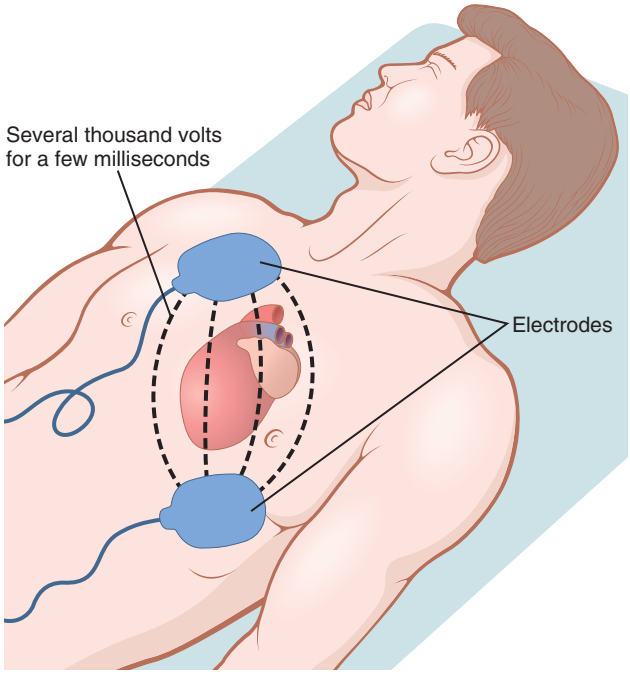


Figure 13-18. Application of electrical current to the chest to stop ventricular fibrillation.

ventricle may be implanted in the patient. The device is programmed to detect ventricular fibrillation and revert it by delivering a brief electrical impulse to the heart. Recent advances in electronics and batteries have permitted development of ICDs that can deliver enough electrical current to defibrillate the heart through electrode wires implanted subcutaneously, outside the rib cage near the heart rather than in or on the heart itself. These devices can be implanted with a minor surgical procedure.

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 process, plus defibrillation, is called *cardiopulmonary resuscitation* (CPR).

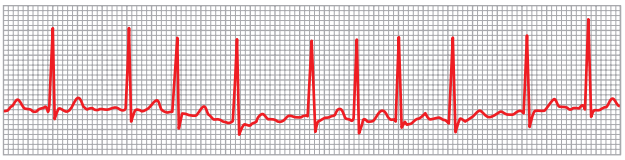


Figure 13-19. Atrial fibrillation (lead II). The waves that can be seen are ventricular QRS and T waves.

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.

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 13-20**).

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, which can result, for example, 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.

Impaired Pumping 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 percent. Therefore, in contrast to the lethality of ventricular fibrillation, a person can live for years with atrial fibrillation, although at reduced efficiency of overall heart pumping.

ELECTROCARDIOGRAM IN ATRIAL FIBRILLATION

Figure 13-19 shows the ECG 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 ECG,

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 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 ECG of **Figure 13-19**, 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 generating a normal rhythm.*

ATRIAL FLUTTER

Atrial flutter is another condition caused by a circus movement in the atria. Atrial flutter 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 13-20**. 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,

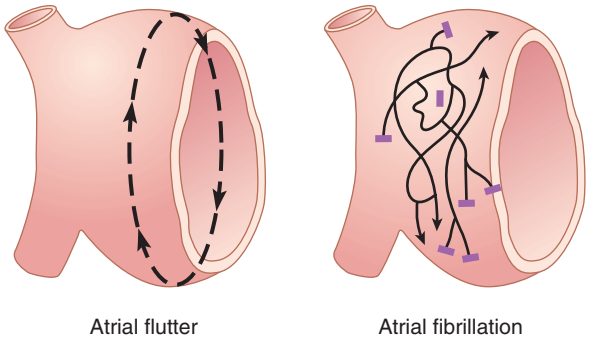


Figure 13-20. Pathways of impulses in atrial flutter and atrial fibrillation.

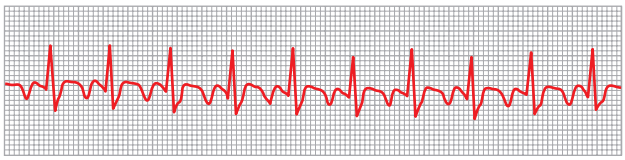


Figure 13-21. Atrial flutter—2:1 atrial to ventricle rhythm (lead II).

there are usually two to three beats of the atria for every single beat of the ventricles.

Figure 13-21 shows a typical ECG 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.

CARDIAC ARREST

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

Cardiac arrest may occur *during deep anesthesia,* when severe hypoxia may develop 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 many instances of cardiac arrest from anesthesia, prolonged CPR (for many minutes or even hours) is quite successful in re-establishing 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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