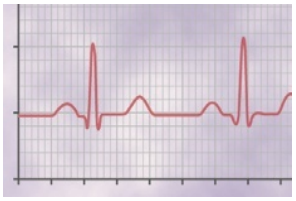
The Normal Electrocardiogram



When the cardiac impulse passes through the heart, electrical current also spreads from the heart into the adjacent tissues surrounding the heart. A small portion of the current spreads all the way to the surface of the body. If electrodes are placed on the skin on opposite sides of the heart, electrical potentials generated by the current can be recorded; the recording is known as an electrocardiogram. A normal electrocardiogram for two beats of the heart is shown in Figure 11-1.

Characteristics of the Normal Electrocardiogram

The normal electrocardiogram (see Figure 11-1) is composed of a P wave, a QRS complex, and a T wave. The QRS complex is often, but not always, three separate waves: the Q wave, the R wave, and the S wave.

The P wave is caused by electrical potentials generated when the atria depolarize before atrial contraction begins. The QRS complex is caused by potentials generated when the ventricles depolarize before contraction, that is, as the depolarization wave spreads through the ventricles. Therefore, both the P wave and the components of the QRS complex are *depolarization waves*.

The T wave is caused by potentials generated as the ventricles recover from the state of depolarization. This process normally occurs in ventricular muscle 0.25 to 0.35 second after depolarization, and the T wave is known as a *repolarization wave.*

Thus, the electrocardiogram is composed of both depolarization and repolarization waves. The principles of depolarization and repolarization are discussed in Chapter 5. The distinction between depolarization waves and repolarization waves is so important in electrocardiography that further clarification is necessary.

Depolarization Waves versus Repolarization Waves

Figure 11-2 shows a single cardiac muscle fiber in four stages of depolarization and repolarization, the color red designating depolarization. During depolarization, the normal negative potential inside the fiber reverses and becomes slightly positive inside and negative outside.

In Figure 11-2*A*, depolarization, demonstrated by red positive charges inside and red negative charges outside, is traveling from left to right. The first half of the fiber has already depolarized, while the remaining half is still polarized. Therefore, the left electrode on the outside of the fiber is in an area of negativity, and the right electrode is in an area of positivity; this causes the meter to record positively. To the right of the muscle fiber is shown a record

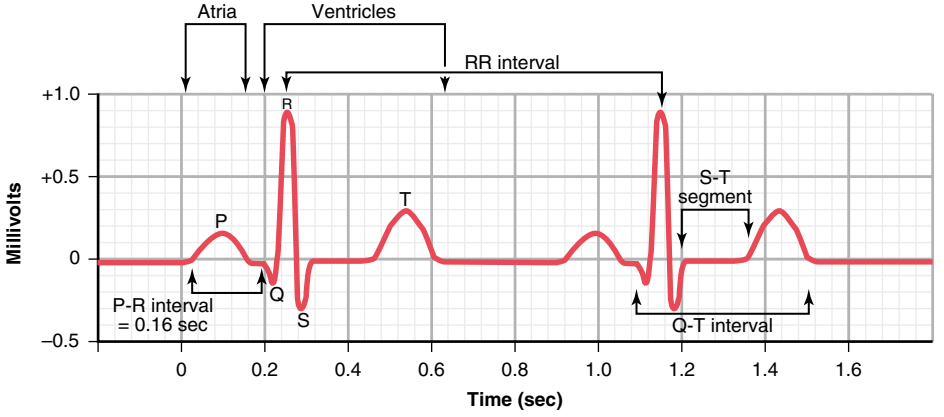


Figure 11-1 Normal electrocardiogram.

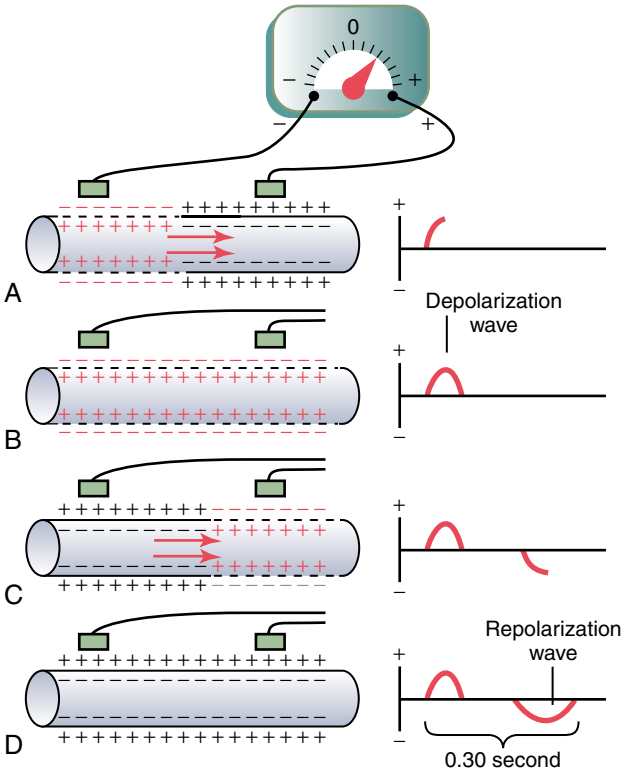


Figure 11-2 Recording the depolarization wave (*A* and *B*) and the repolarization wave (*C* and *D*) from a cardiac muscle fiber.

of changes in potential between the two electrodes, as recorded by a high-speed recording meter. Note that when depolarization has reached the halfway mark in Figure 11-2*A*, the record has risen to a maximum positive value.

In Figure 11-2*B*, depolarization has extended over the entire muscle fiber, and the recording to the right has returned to the zero baseline because both electrodes are now in areas of equal negativity. The completed wave is a depolarization wave because it results from spread of depolarization along the muscle fiber membrane.

Figure 11-2*C* shows halfway repolarization of the same muscle fiber, with positivity returning to the outside of the fiber. At this point, the left electrode is in an area of positivity, and the right electrode is in an area of negativity. This is opposite to the polarity in Figure 11-2*A*. Consequently, the recording, as shown to the right, becomes negative.

In Figure 11-2*D*, the muscle fiber has completely repolarized, and both electrodes are now in areas of positivity so that no potential difference is recorded between them. Thus, in the recording to the right, the potential returns once more to zero. This completed negative wave is a repolarization wave because it results from spread of repolarization along the muscle fiber membrane.

Relation of the Monophasic Action Potential of Ventricular Muscle to the QRS and T Waves in the Standard Electrocardiogram. The monophasic action potential of ventricular muscle, discussed in Chapter 10, normally lasts between 0.25 and 0.35 second. The top part of Figure 11-3 shows a monophasic action potential

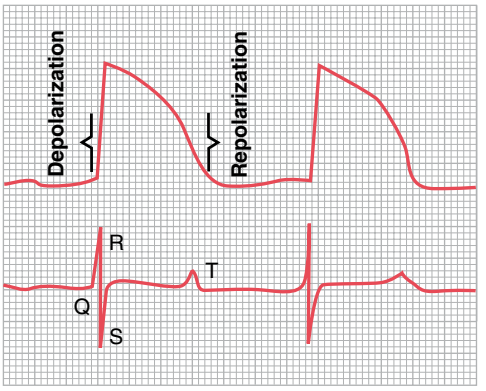


Figure 11-3 *Above,* Monophasic action potential from a ventricular muscle fiber during normal cardiac function, showing rapid depolarization and then repolarization occurring slowly during the plateau stage but rapidly toward the end. *Below,* Electrocardiogram recorded simultaneously.

recorded from a microelectrode inserted to the inside of a single ventricular muscle fiber. The upsweep of this action potential is caused by depolarization, and the return of the potential to the baseline is caused by repolarization.

Note in the lower half of the figure a simultaneous recording of the electrocardiogram from this same ventricle, which shows the QRS waves appearing at the beginning of the monophasic action potential and the T wave appearing at the end. Note especially that *no potential is recorded in the electrocardiogram when the ventricular muscle is either completely polarized or completely depolarized*. Only when the muscle is partly polarized and partly depolarized does current flow from one part of the ventricles to another part and therefore current also flows to the surface of the body to produce the electrocardiogram.

Relationship of Atrial and Ventricular Contraction to the Waves of the Electrocardiogram

Before contraction of muscle can occur, depolarization must spread through the muscle to initiate the chemical processes of contraction. Refer again to Figure 11-1; the P wave occurs at the beginning of contraction of the atria, and the QRS complex of waves occurs at the beginning of contraction of the ventricles. The ventricles remain contracted until after repolarization has occurred, that is, until after the end of the T wave.

The atria repolarize about 0.15 to 0.20 second after termination of the P wave. This is also approximately when the QRS complex is being recorded in the electrocardiogram. Therefore, the atrial repolarization wave, known as the *atrial T wave,* is usually obscured by the much larger QRS complex. For this reason, an atrial T wave seldom is observed in the electrocardiogram.

The ventricular repolarization wave is the T wave of the normal electrocardiogram. Ordinarily, ventricular muscle begins to repolarize in some fibers about 0.20 second after the beginning of the depolarization wave (the QRS complex), but in many other fibers, it takes as long as 0.35 second. Thus, the process of ventricular repolarization

extends over a long period, about 0.15 second. For this reason, the T wave in the normal electrocardiogram is a prolonged wave, but the voltage of the T wave is considerably less than the voltage of the QRS complex, partly because of its prolonged length.

Voltage and Time Calibration of the Electrocardiogram

All recordings of electrocardiograms are made with appropriate calibration lines on the recording paper. Either these calibration lines are already ruled on the paper, as is the case when a pen recorder is used, or they are recorded on the paper at the same time that the electrocardiogram is recorded, which is the case with the photographic types of electrocardiographs.

As shown in Figure 11-1, the horizontal calibration lines are arranged so that 10 of the small line divisions upward or downward in the standard electrocardiogram represent 1 millivolt, with positivity in the upward direction and negativity in the downward direction.

The vertical lines on the electrocardiogram are time calibration lines. A typical electrocardiogram is run at a paper speed of 25 millimeters per second, although faster speeds are sometimes used. Therefore, each 25 millimeters in the horizontal direction is 1 second, and each 5-millimeter segment, indicated by the dark vertical lines, represents 0.20 second. The 0.20-second intervals are then broken into five smaller intervals by thin lines, each of which represents 0.04 second.

Normal Voltages in the Electrocardiogram. The recorded voltages of the waves in the normal electrocardiogram depend on the manner in which the electrodes are applied to the surface of the body and how close the electrodes are to the heart. When one electrode is placed directly over the ventricles and a second electrode is placed elsewhere on the body remote from the heart, the voltage of the QRS complex may be as great as 3 to 4 millivolts. Even this voltage is small in comparison with the monophasic action potential of 110 millivolts recorded directly at the heart muscle membrane. When electrocardiograms are recorded from electrodes on the two arms or on one arm and one leg, the voltage of the QRS complex usually is 1.0 to 1.5 millivolts from the top of the R wave to the bottom of the S wave; the voltage of the P wave is between 0.1 and 0.3 millivolts; and that of the T wave is between 0.2 and 0.3 millivolts.

P-Q or P-R Interval. The time between the beginning of the P wave and the beginning of the QRS complex is the interval between the beginning of electrical excitation of the atria and the beginning of excitation of the ventricles. This period is called the *P-Q interval.* The normal P-Q interval is about 0.16 second. (Often this interval is called the *P-R interval* because the Q wave is likely to be absent.)

Q-T Interval. Contraction of the ventricle lasts almost from the beginning of the Q wave (or R wave, if the Q wave is absent) to the end of the T wave. This interval is called the *Q-T interval* and ordinarily is about 0.35 second.

Rate of Heartbeat as Determined from the Electrocardiogram. The rate of heartbeat can be determined easily from an electrocardiogram because the heart rate is the reciprocal of the time interval between two successive heartbeats. If the interval between two beats as determined from the time calibration lines is 1 second, the heart rate is 60 beats per minute. The normal interval between two successive QRS complexes in the adult person is about 0.83 second. This is a heart rate of 60/0.83 times per minute, or 72 beats per minute.

Methods for Recording Electrocardiograms

Sometimes the electrical currents generated by the cardiac muscle during each beat of the heart change electrical potentials and polarities on the respective sides of the heart in less than 0.01 second. Therefore, it is essential that any apparatus for recording electrocardiograms be capable of responding rapidly to these changes in potentials.

Recorders for Electrocardiographs

Many modern clinical electrocardiographs use computer-based systems and electronic display, whereas others use a direct pen recorder that writes the electrocardiogram with a pen directly on a moving sheet of paper. Sometimes the pen is a thin tube connected at one end to an inkwell, and its recording end is connected to a powerful electromagnet system that is capable of moving the pen back and forth at high speed. As the paper moves forward, the pen records the electrocardiogram. The movement of the pen is controlled by appropriate electronic amplifiers connected to electrocardiographic electrodes on the patient.

Other pen recording systems use special paper that does not require ink in the recording stylus. One such paper turns black when it is exposed to heat; the stylus itself is made very hot by electrical current flowing through its tip. Another type turns black when electrical current flows from the tip of the stylus through the paper to an electrode at its back. This leaves a black line on the paper where the stylus touches.

Flow of Current Around the Heart during the Cardiac Cycle

Recording Electrical Potentials from a Partially Depolarized Mass of Syncytial Cardiac Muscle

Figure 11-4 shows a syncytial mass of cardiac muscle that has been stimulated at its centralmost point. Before stimulation, all the exteriors of the muscle cells had been positive and the interiors negative. For reasons presented in Chapter 5 in the discussion of membrane potentials, as soon as an area of cardiac syncytium becomes depolarized, negative charges leak to the outsides of the depolarized muscle fibers, making this part of the surface

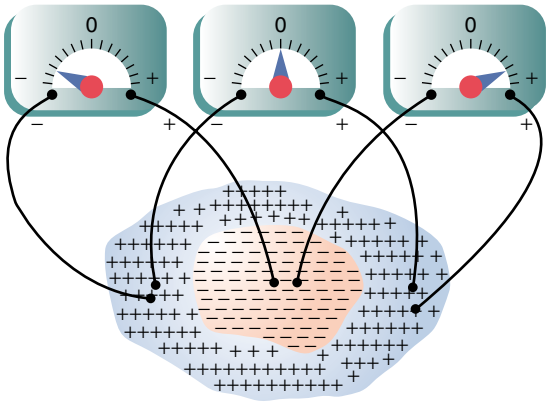


Figure 11-4 Instantaneous potentials develop on the surface of a cardiac muscle mass that has been depolarized in its center.

electronegative, as represented by the negative signs in Figure 11-4. The remaining surface of the heart, which is still polarized, is represented by the positive signs. Therefore, a meter connected with its negative terminal on the area of depolarization and its positive terminal on one of the still-polarized areas, as shown to the right in the figure, records positively.

Two other electrode placements and meter readings are also demonstrated in Figure 11-4. These should be studied carefully, and the reader should be able to explain the causes of the respective meter readings. Because the depolarization spreads in all directions through the heart, the potential differences shown in the figure persist for only a few thousandths of a second, and the actual voltage measurements can be accomplished only with a high-speed recording apparatus.

Flow of Electrical Currents in the Chest Around the Heart

Figure 11-5 shows the ventricular muscle lying within the chest. Even the lungs, although mostly filled with air, conduct electricity to a surprising extent, and fluids in other tissues surrounding the heart conduct electricity even more easily. Therefore, the heart is actually suspended in a conductive medium. When one portion of the ventricles depolarizes and therefore becomes electronegative with respect to the remainder, electrical current flows from the depolarized area to the polarized area in large circuitous routes, as noted in the figure.

It should be recalled from the discussion of the Purkinje system in Chapter 10 that the cardiac impulse first arrives in the ventricles in the septum and shortly thereafter spreads to the inside surfaces of the remainder of the ventricles, as shown by the red areas and the negative signs in Figure 11-5. This provides electronegativity on the insides of the ventricles and electropositivity on the outer walls of the ventricles, with electrical current flowing through the fluids surrounding the ventricles along elliptical paths, as demonstrated by the curving arrows in the figure. If one algebraically averages all the lines of current flow (the elliptical lines), one finds that *the average current flow occurs with negativity toward the base of the heart and with positivity toward the apex.*

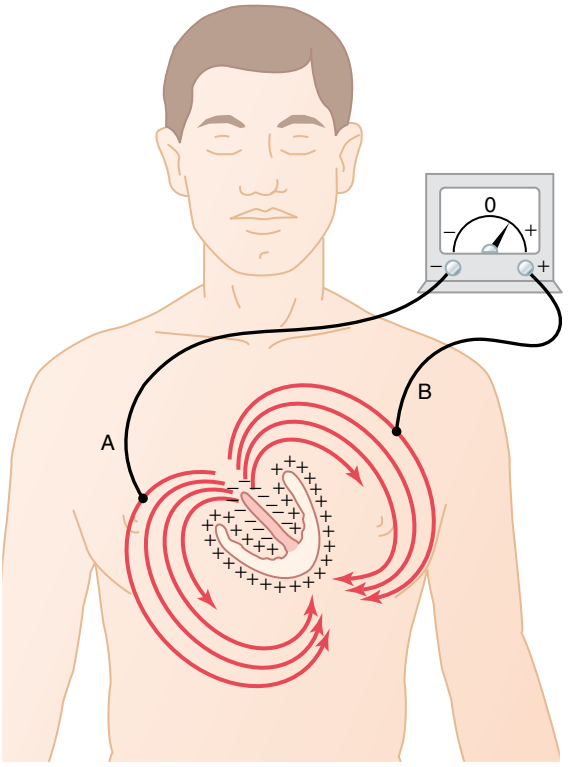


Figure 11-5 Flow of current in the chest around partially depolarized ventricles.

During most of the remainder of the depolarization process, current also continues to flow in this same direction, while depolarization spreads from the endocardial surface outward through the ventricular muscle mass. Then, immediately before depolarization has completed its course through the ventricles, the average direction of current flow reverses for about 0.01 second, flowing from the ventricular apex toward the base, because the last part of the heart to become depolarized is the outer walls of the ventricles near the base of the heart.

Thus, in normal heart ventricles, current flows from negative to positive primarily in the direction from the base of the heart toward the apex during almost the entire cycle of depolarization, except at the very end. And if a meter is connected to electrodes on the surface of the body as shown in Figure 11-5, the electrode nearer the base will be negative, whereas the electrode nearer the apex will be positive, and the recording meter will show positive recording in the electrocardiogram.

Electrocardiographic Leads

Three Bipolar Limb Leads

Figure 11-6 shows electrical connections between the patient's limbs and the electrocardiograph for recording electrocardiograms from the so-called *standard bipolar*

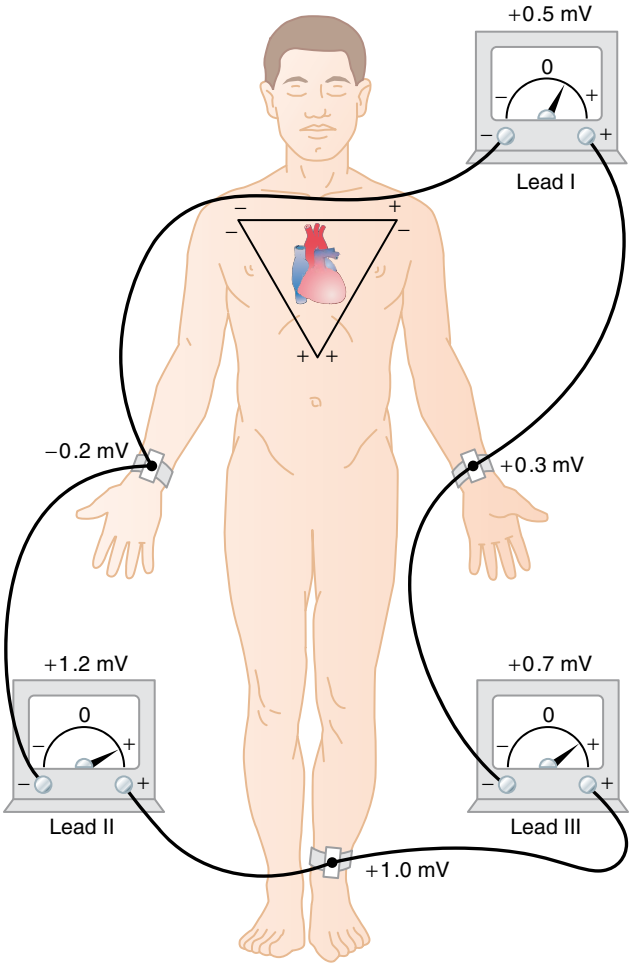


Figure 11-6 Conventional arrangement of electrodes for recording the standard electrocardiographic leads. Einthoven's triangle is superimposed on the chest.

limb leads. The term "bipolar" means that the electrocardiogram is recorded from two electrodes located on different sides of the heart—in this case, on the limbs. Thus, a "lead" is not a single wire connecting from the body but a combination of two wires and their electrodes to make a complete circuit between the body and the electrocardiograph. The electrocardiograph in each instance is represented by an electrical meter in the diagram, although the actual electrocardiograph is a high-speed recording meter with a moving paper.

Lead I. In recording limb lead I, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal to the left arm. Therefore, when the point where the right arm connects to the chest is electronegative with respect to the point where the left arm connects, the electrocardiograph records positively, that is, above the zero voltage line in the electrocardiogram. When the opposite is true, the electrocardiograph records below the line.

Lead II. To record limb lead II, the negative terminal of the electrocardiograph is connected to the right arm and the positive terminal to the left leg. Therefore, when the right arm is negative with respect to the left leg, the electrocardiograph records positively.

Lead III. To record limb lead III, the negative terminal of the electrocardiograph is connected to the left arm and the positive terminal to the left leg. This means that the electrocardiograph records positively when the left arm is negative with respect to the left leg.

Einthoven's Triangle. In Figure 11-6, the triangle, called *Einthoven's triangle,* is drawn around the area of the heart. This illustrates that the two arms and the left leg form apices of a triangle surrounding the heart. The two apices at the upper part of the triangle represent the points at which the two arms connect electrically with the fluids around the heart, and the lower apex is the point at which the left leg connects with the fluids.

Einthoven's Law. Einthoven's law states that if the electrical potentials of any two of the three bipolar limb electrocardiographic leads are known at any given instant, the third one can be determined mathematically by simply summing the first two. Note, however, that the positive and negative signs of the different leads must be observed when making this summation.

For instance, let us assume that momentarily, as noted in Figure 11-6, the right arm is −0.2 millivolts (negative) with respect to the average potential in the body, the left arm is +0.3 millivolts (positive), and the left leg is +1.0 millivolts (positive). Observing the meters in the figure, one can see that lead I records a positive potential of +0.5 millivolts because this is the difference between the −0.2 millivolts on the right arm and the +0.3 millivolts on the left arm. Similarly, lead III records a positive potential of +0.7 millivolts, and lead II records a positive potential of +1.2 millivolts because these are the instantaneous potential differences between the respective pairs of limbs.

Now, note that the sum of the voltages in leads I and III equals the voltage in lead II; that is, 0.5 plus 0.7 equals 1.2. Mathematically, this principle, called Einthoven's law, holds true at any given instant while the three "standard" bipolar electrocardiograms are being recorded.

Normal Electrocardiograms Recorded from the Three Standard Bipolar Limb Leads. Figure 11-7 shows recordings of the electrocardiograms in leads I, II, and III. It is obvious that the electrocardiograms in these three leads are similar to one another because they all record positive P waves and positive T waves, and the major portion of the QRS complex is also positive in each electrocardiogram.

On analysis of the three electrocardiograms, it can be shown, with careful measurements and proper observance of polarities, that at any given instant the sum of the potentials in leads I and III equals the potential in lead II, thus illustrating the validity of Einthoven's law.

Because the recordings from all the bipolar limb leads are similar to one another, it does not matter greatly which lead is recorded when one wants to diagnose different cardiac arrhythmias, because diagnosis of arrhythmias depends mainly on the time relations between the different waves of the cardiac cycle. But when one wants to diagnose damage in the ventricular or atrial muscle or in the Purkinje conducting system, it matters greatly which

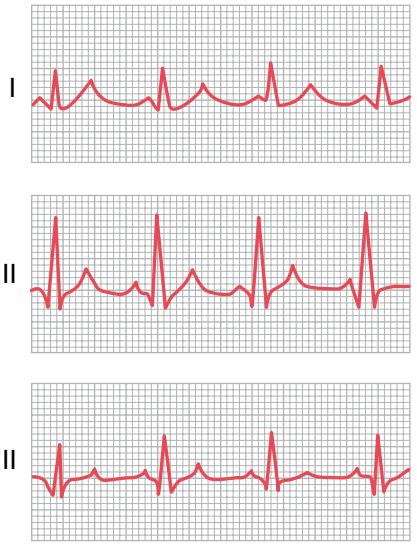


Figure 11-7 Normal electrocardiograms recorded from the three standard electrocardiographic leads.

leads are recorded, because abnormalities of cardiac muscle contraction or cardiac impulse conduction do change the patterns of the electrocardiograms markedly in some leads yet may not affect other leads. Electrocardiographic interpretation of these two types of conditions—cardiac myopathies and cardiac arrhythmias—is discussed separately in Chapters 12 and 13.

Chest Leads (Precordial Leads)

Often electrocardiograms are recorded with one electrode placed on the anterior surface of the chest directly over the heart at one of the points shown in Figure 11-8. This electrode is connected to the positive terminal of the electrocardiograph, and the negative electrode, called the *indifferent electrode,* is connected through equal electrical resistances to the right arm, left arm, and left leg all at the same time, as also shown in the figure. Usually six standard chest leads are recorded, one at a time, from the anterior chest wall, the chest electrode being placed sequentially at the six points shown in the diagram. The different recordings are known as leads V1, V2, V3, V4, V5, and V6.

Figure 11-9 illustrates the electrocardiograms of the healthy heart as recorded from these six standard chest leads. Because the heart surfaces are close to the chest wall, each chest lead records mainly the electrical potential of the cardiac musculature immediately beneath the electrode. Therefore, relatively minute abnormalities in the ventricles, particularly in the anterior ventricular wall, can cause marked changes in the electrocardiograms recorded from individual chest leads.

In leads V1 and V2, the QRS recordings of the normal heart are mainly negative because, as shown in Figure 11-8, the chest electrode in these leads is nearer to the base of the heart than to the apex, and the base of the heart is the direction of electronegativity during most of

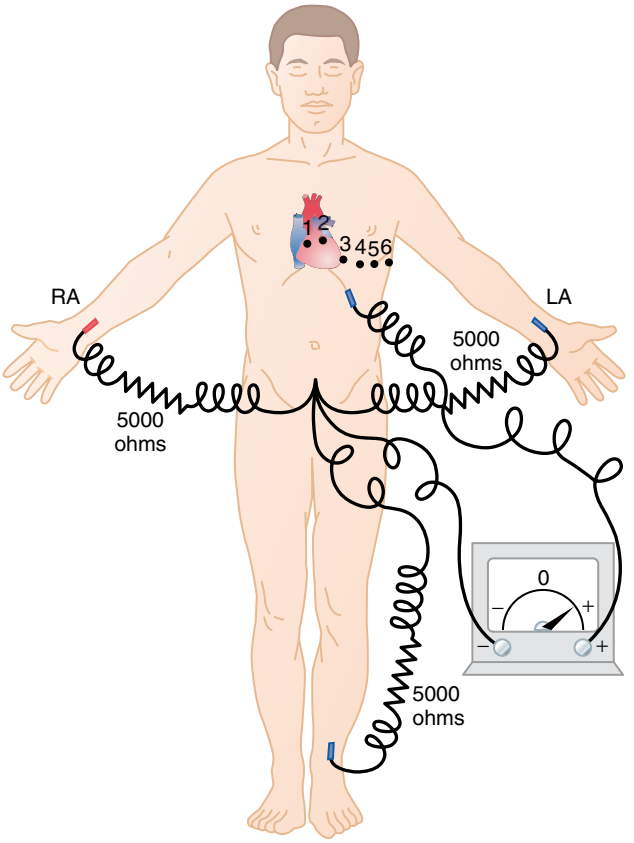


Figure 11-8 Connections of the body with the electrocardiograph for recording chest leads. LA, left arm; RA, right arm.

the ventricular depolarization process. Conversely, the QRS complexes in leads V4, V5, and V6 are mainly positive because the chest electrode in these leads is nearer the heart apex, which is the direction of electropositivity during most of depolarization.

Augmented Unipolar Limb Leads

Another system of leads in wide use is the *augmented unipolar limb lead*. In this type of recording, two of the limbs are connected through electrical resistances to the negative terminal of the electrocardiograph, and the third limb is connected to the positive terminal. When the positive terminal is on the right arm, the lead is known as the aVR lead; when on the left arm, the aVL lead; and when on the left leg, the aVF lead.

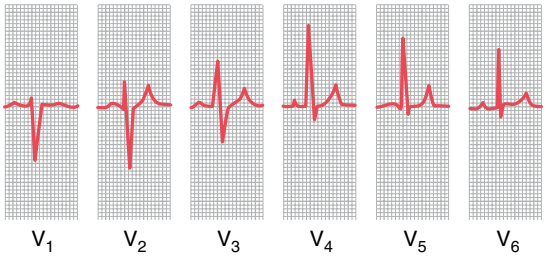


Figure 11-9 Normal electrocardiograms recorded from the six standard chest leads.

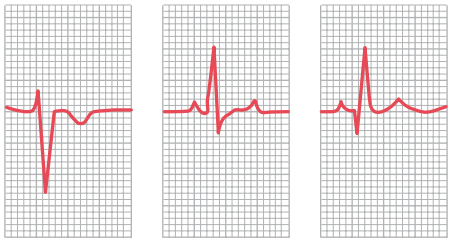


Figure 11-10 Normal electrocardiograms recorded from the three augmented unipolar limb leads.

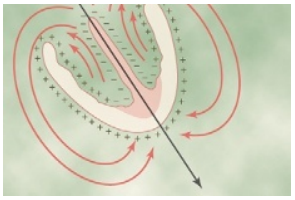
Normal recordings of the augmented unipolar limb leads are shown in Figure 11-10. They are all similar to the standard limb lead recordings, except that the recording from the aVR lead is inverted. (Why does this inversion occur? Study the polarity connections to the electrocardiograph to determine this.)

Bibliography

See bibliography for Chapter 13.

CHAPTER 12

Electrocardiographic Interpretation of Cardiac Muscle and Coronary Blood Flow Abnormalities: Vectorial Analysis



From the discussion in Chapter 10 of impulse transmission through the heart, it is obvious that any change in the pattern of this transmission can cause abnormal electrical potentials around the heart and, consequently, alter the shapes of the waves in the electrocardiogram. For this reason, most serious abnormalities of the heart muscle can be diagnosed by analyzing the contours of the waves in the different electrocardiographic leads.

Principles of Vectorial Analysis of Electrocardiograms

Use of Vectors to Represent Electrical Potentials

Before it is possible to understand how cardiac abnormalities affect the contours of the electrocardiogram, one must first become thoroughly familiar with the concept of *vectors* and *vectorial analysis* as applied to electrical potentials in and around the heart.

Several times in Chapter 11 it was pointed out that heart current flows in a particular direction in the heart at a given instant during the cardiac cycle. A vector is an arrow that points in the direction of the electrical potential generated by the current flow, *with the arrowhead in the positive direction.* Also, by convention, the length of the arrow is drawn *proportional to the voltage of the potential.*

"Resultant" Vector in the Heart at Any Given Instant. Figure 12-1 shows, by the shaded area and the negative signs, depolarization of the ventricular septum and parts of the apical endocardial walls of the two ventricles. At this instant of heart excitation, electrical current flows between the depolarized areas inside the heart and the nondepolarized areas on the outside of the heart, as indicated by the long elliptical arrows. Some current also flows inside the heart chambers directly from the depolarized areas toward the still polarized areas. Overall, considerably more current flows downward from the base of the ventricles toward the apex than in the upward direction. Therefore, the summated vector of the generated potential at this particular instant, called the *instantaneous mean vector,* is represented by the long *black* arrow drawn through the center of the ventricles in a direction from base toward apex. Furthermore, because the summated current is considerable in quantity, the potential is large and the vector is long.

Direction of a Vector Is Denoted in Terms of Degrees

When a vector is exactly horizontal and directed toward the person's left side, the vector is said to extend in the direction of 0 degrees, as shown in Figure 12-2. From this zero reference point, the scale of vectors rotates clockwise: when the vector extends from above and straight downward, it has a direction of +90 degrees; when it extends from the person's left to right, it has a direction of +180 degrees; and when it extends straight upward, it has a direction of –90 (or +270) degrees.

In a normal heart, the average direction of the vector during spread of the depolarization wave through the ventricles, called the *mean QRS vector,* is about +59 degrees, which is shown by vector *A* drawn through the center of Figure 12-2 in the +59-degree direction. This means that during most of the depolarization wave, the apex of the heart remains positive with respect to the base of the heart, as discussed later in the chapter.

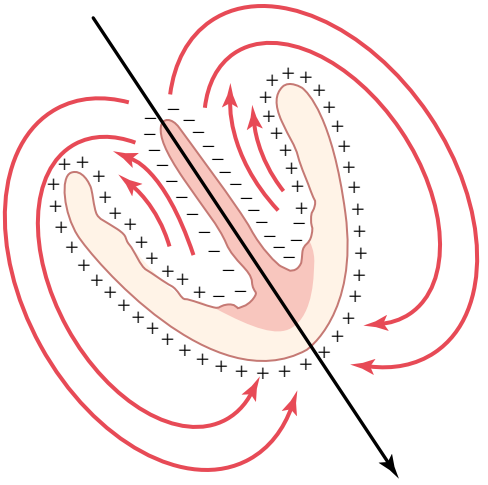


Figure 12-1 Mean vector through the partially depolarized ventricles.

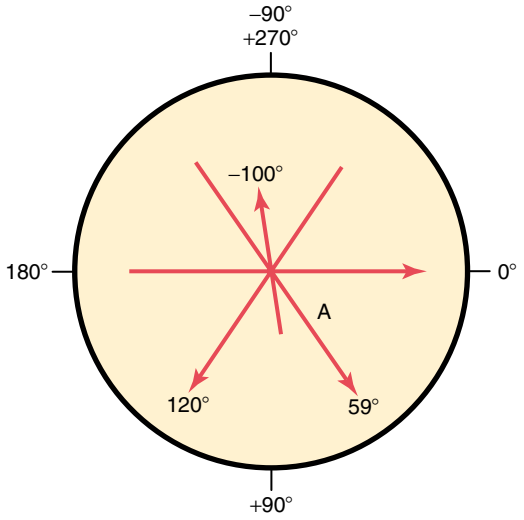


Figure 12-2 Vectors drawn to represent potentials for several different hearts, and the "axis" of the potential (expressed in degrees) for each heart.

Axis for Each Standard Bipolar Lead and Each Unipolar Limb Lead

In Chapter 11, the three standard bipolar and the three unipolar limb leads are described. Each lead is actually a pair of electrodes connected to the body on opposite sides of the heart, and the direction from negative electrode to positive electrode is called the "axis" of the lead. Lead I is recorded from two electrodes placed respectively on the two arms. Because the electrodes lie exactly in the horizontal direction, with the positive electrode to the left, the axis of lead I is 0 degrees.

In recording lead II, electrodes are placed on the right arm and left leg. The right arm connects to the torso in the upper right-hand corner and the left leg connects in the lower left-hand corner. Therefore, the direction of this lead is about +60 degrees.

By similar analysis, it can be seen that lead III has an axis of about +120 degrees; lead aVR, +210 degrees; aVF, +90 degrees; and aVL –30 degrees. The directions of the axes of all these leads are shown in Figure 12-3, which is known as the *hexagonal reference system.* The polarities of the electrodes are shown by the plus and minus signs in the figure. *The reader must learn these axes and their polarities, particularly for the bipolar limb leads I, II, and III, to understand the remainder of this chapter.*

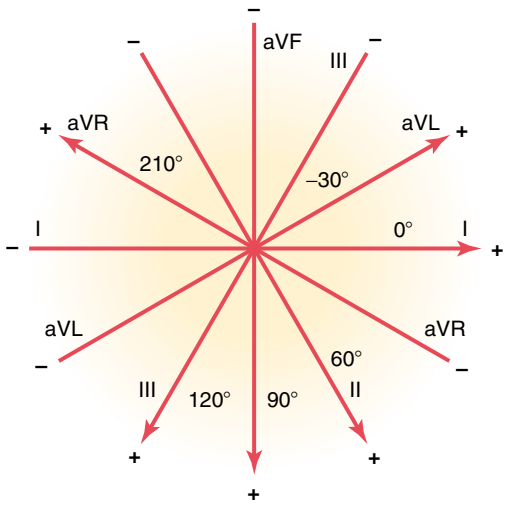


Figure 12-3 Axes of the three bipolar and three unipolar leads.

Vectorial Analysis of Potentials Recorded in Different Leads

Now that we have discussed, first, the conventions for representing potentials across the heart by means of vectors and, second, the axes of the leads, it is possible to use these together to determine the instantaneous potential that will be recorded in the electrocardiogram of each lead for a given vector in the heart, as follows.

Figure 12-4 shows a partially depolarized heart; vector *A* represents the instantaneous mean direction of current flow in the ventricles. In this instance, the direction of the vector is +55 degrees, and the voltage of the potential, represented by the length of vector *A,* is 2 mv. In the diagram below the heart, vector *A* is shown again, and a line is drawn to represent the axis of lead I in the 0-degree direction. To determine how much of the voltage in vector *A* will be recorded in lead I, a line perpendicular to the axis of lead I is drawn from the tip of vector *A* to the lead I axis, and a so-called *projected vector (B)* is drawn along the lead I axis. The arrow of this projected vector points toward the positive end of the lead I axis, which means that the record momentarily being recorded in the electrocardiogram of lead I is positive. And the instantaneous recorded voltage will be equal to the length of *B* divided by the length of *A* times 2 millivolts, or about 1 millivolt.

Figure 12-5 shows another example of vectorial analysis. In this example, vector *A* represents the electrical potential and its axis at a given instant during ventricular depolarization in a heart in which the left side of the heart depolarizes more rapidly than the right. In this instance, the instantaneous vector has a direction of 100 degrees, and its voltage is again 2 millivolts. To determine the potential actually recorded in lead I, we draw a perpendicular line from the tip of vector *A* to the lead I axis and find projected vector *B.* Vector *B* is very short and this time in the negative direction, indicating that at this

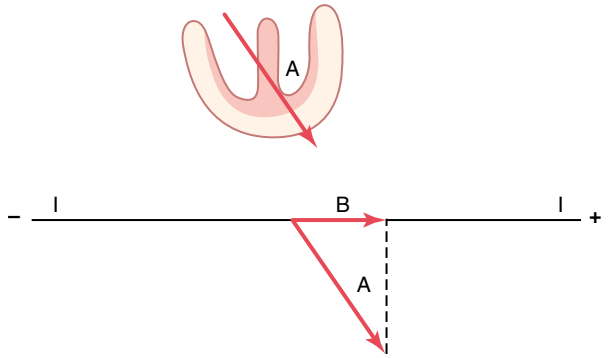


Figure 12-4 Determination of a projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

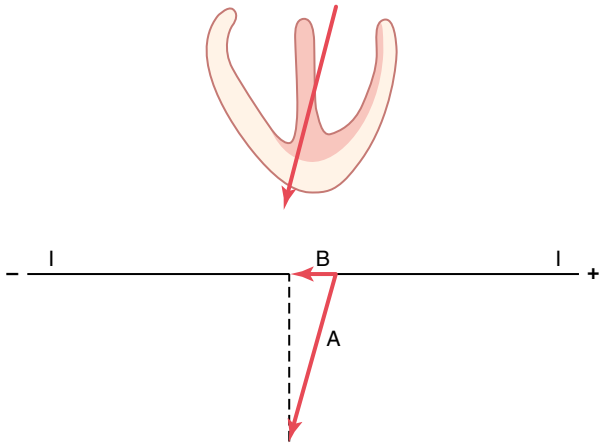


Figure 12-5 Determination of the projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

particular instant, the recording in lead I will be negative (below the zero line in the electrocardiogram), and the voltage recorded will be slight, about –0.3 millivolts. This figure demonstrates that *when the vector in the heart is in a direction almost perpendicular to the axis of the lead, the voltage recorded in the electrocardiogram of this lead is very low.* Conversely, *when the heart vector has almost exactly the same axis as the lead axis, essentially the entire voltage of the vector will be recorded.*

Vectorial Analysis of Potentials in the Three Standard Bipolar Limb Leads. In Figure 12-6, vector *A* depicts the instantaneous electrical potential of a partially depolarized heart. To determine the potential recorded at this instant in the electrocardiogram for each one of the three standard bipolar limb leads, perpendicular lines (the dashed lines) are drawn from the tip of vector *A* to the three lines representing the axes of the three different standard leads, as shown in the figure. The projected vector *B* depicts the potential recorded at that instant in lead I, projected vector *C* depicts the potential in lead II, and projected vector *D* depicts the potential in lead III. In each of these, the record in the electrocardiogram is positive—that is, above the zero line—because the projected vectors point in the positive directions along the axes of all the leads. The potential in lead I (vector *B*) is about one-half that of the actual potential in the heart (vector *A*); in lead II (vector *C*), it is almost equal to that in the heart; and in lead III (vector *D*), it is about one-third that in the heart.

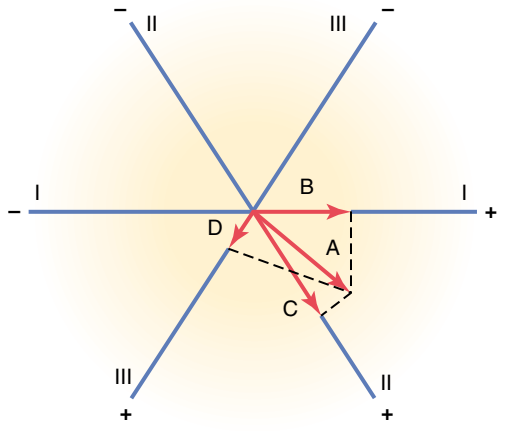


Figure 12-6 Determination of projected vectors in leads I, II, and III when vector A represents the instantaneous potential in the ventricles.

An identical analysis can be used to determine potentials recorded in augmented limb leads, except that the respective axes of the augmented leads (see Figure 12-3) are used in place of the standard bipolar limb lead axes used for Figure 12-6.

Vectorial Analysis of the Normal Electrocardiogram

Vectors That Occur at Successive Intervals during Depolarization of the Ventricles—the QRS Complex

When the cardiac impulse enters the ventricles through the atrioventricular bundle, the first part of the ventricles to become depolarized is the left endocardial surface of the septum. Then depolarization spreads rapidly to involve both endocardial surfaces of the septum, as demonstrated by the darker shaded portion of the ventricle in Figure 12-7*A*. Next, depolarization spreads along the endocardial surfaces of the remainder of the two ventricles, as shown in Figure 12-7*B* and *C*. Finally, it spreads through the ventricular muscle to the outside of the heart, as shown progressively in Figure 12-7*C, D,* and *E.*

At each stage in Figure 12-7, parts *A* to *E,* the instantaneous mean electrical potential of the ventricles is represented by a red vector superimposed on the ventricle in each figure. Each of these vectors is then analyzed by the method described in the preceding section to determine the voltages that will be recorded at each instant in each of the three standard electrocardiographic leads. To the right in each figure is shown progressive development of the electrocardiographic QRS complex. *Keep in mind that a positive vector in a lead will cause recording in the electrocardiogram above the zero line, whereas a negative vector will cause recording below the zero line.*

Before proceeding with further consideration of vectorial analysis, it is essential that this analysis of the successive normal vectors presented in Figure 12-7 be understood. Each of these analyses should be studied in detail by the procedure given here. A short summary of this sequence follows.

In Figure 12-7*A,* the ventricular muscle has just begun to be depolarized, representing an instant about 0.01 second after the onset of depolarization. At this time, the vector is short because only a small portion of the ventricles—the septum—is depolarized. Therefore, all electrocardiographic voltages are low, as recorded to the right of the ventricular muscle for each of the leads. The voltage in lead II is greater than the voltages in leads I and III because the heart vector extends mainly in the same direction as the axis of lead II.

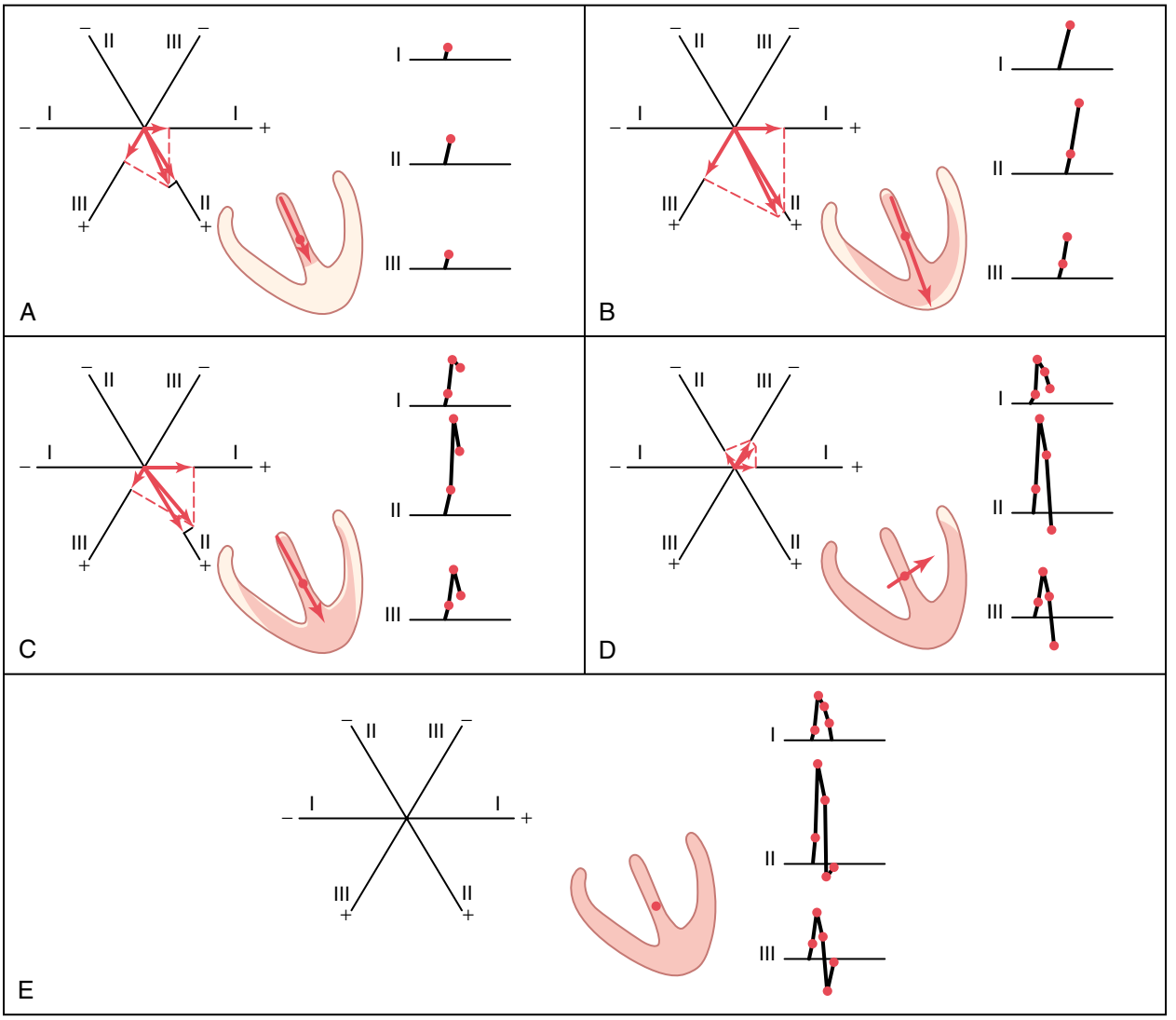


Figure 12-7 Shaded areas of the ventricles are depolarized (–); nonshaded areas are still polarized (+). The ventricular vectors and QRS complexes 0.01 second after onset of ventricular depolarization *(A)*; 0.02 second after onset of depolarization *(B)*; 0.035 second after onset of depolarization *(C)*; 0.05 second after onset of depolarization *(D)*; and after depolarization of the ventricles is complete, 0.06 second after onset *(E)*.

In Figure 12-7*B*, which represents about 0.02 second after onset of depolarization, the heart vector is long because much of the ventricular muscle mass has become depolarized. Therefore, the voltages in all electrocardiographic leads have increased.

In Figure 12-7*C*, about 0.035 second after onset of depolarization, the heart vector is becoming shorter and the recorded electrocardiographic voltages are lower because the outside of the heart apex is now electronegative, neutralizing much of the positivity on the other epicardial surfaces of the heart. Also, the axis of the vector is beginning to shift toward the left side of the chest because the left ventricle is slightly slower to depolarize than the right. Therefore, the ratio of the voltage in lead I to that in lead III is increasing.

In Figure 12-7*D*, about 0.05 second after onset of depolarization, the heart vector points toward the base of the left ventricle, and it is short because only a minute portion of the ventricular muscle is still polarized positive. Because of the direction of the vector at this time, the voltages recorded in leads II and III are both negative—that is, below the line—whereas the voltage of lead I is still positive.

In Figure 12-7*E*, about 0.06 second after onset of depolarization, the entire ventricular muscle mass is depolarized so that no current flows around the heart and no electrical potential is generated. The vector becomes zero, and the voltages in all leads become zero.

Thus, the QRS complexes are completed in the three standard bipolar limb leads.

Sometimes the QRS complex has a slight negative depression at its beginning in one or more of the leads, which is not shown in Figure 12-7; this depression is the Q wave. When it occurs, it is caused by initial depolarization of the left side of the septum before the right side, which creates a weak vector from left to right for a fraction of a second before the usual base-to-apex vector occurs. The major positive deflection shown in Figure 12-7 is the R wave, and the final negative deflection is the S wave.

Electrocardiogram during Repolarization—the T Wave

After the ventricular muscle has become depolarized, about 0.15 second later, repolarization begins and proceeds until complete at about 0.35 second. This repolarization causes the T wave in the electrocardiogram.

Because the septum and endocardial areas of the ventricular muscle depolarize first, it seems logical that these areas should repolarize first as well. However, this is not the usual case because the septum and other endocardial areas have a longer period of contraction than most of the external surfaces of the heart. Therefore, *the greatest portion of ventricular muscle mass to repolarize first is the entire outer surface of the ventricles, especially near the apex of the heart.* The endocardial areas, conversely, normally repolarize last. This sequence of repolarization is postulated to be caused by the high blood pressure inside the ventricles during contraction, which greatly reduces coronary blood flow to the endocardium, thereby slowing repolarization in the endocardial areas.

Because the outer apical surfaces of the ventricles repolarize before the inner surfaces, the positive end of the overall ventricular vector during repolarization is toward the apex of the heart. *As a result, the normal T wave in all three bipolar limb leads is positive, which is also the polarity of most of the normal QRS complex.*

In Figure 12-8, five stages of repolarization of the ventricles are denoted by progressive increase of the light tan areas—the repolarized areas. At each stage, the vector extends from the base of the heart toward the apex until it disappears in the last stage. At first, the vector is relatively small because the area of repolarization is small. Later, the vector becomes stronger because of greater degrees of repolarization. Finally, the vector becomes weaker again because the areas of depolarization still persisting become so slight that the total quantity of current flow decreases. These changes also demonstrate that the vector is greatest when about half the heart is in the polarized state and about half is depolarized.

The changes in the electrocardiograms of the three standard limb leads during repolarization are noted under each of the ventricles, depicting the progressive stages of repolarization. Thus, over about 0.15 second, the period of time required for the whole process to take place, the T wave of the electrocardiogram is generated.

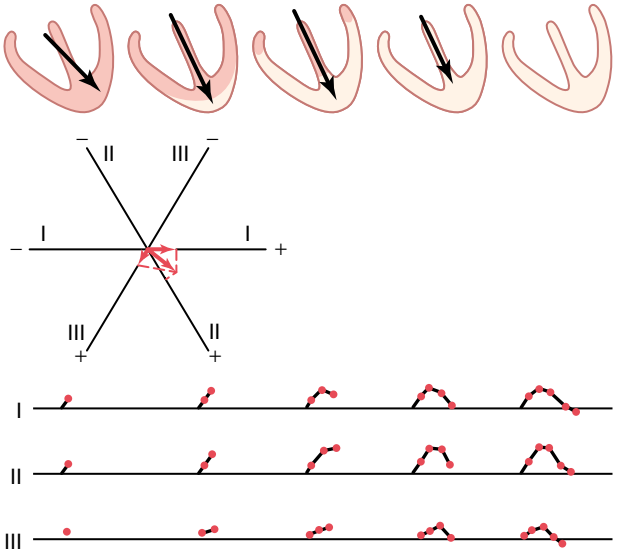


Figure 12-8 Generation of the T wave during repolarization of the ventricles, showing also vectorial analysis of the first stage of repolarization. The total time from the beginning of the T wave to its end is approximately 0.15 second.

Depolarization of the Atria—the P Wave

Depolarization of the atria begins in the sinus node and spreads in all directions over the atria. Therefore, the point of original electronegativity in the atria is about at the point of entry of the superior vena cava where the sinus node lies, and the direction of initial depolarization is denoted by the black vector in Figure 12-9. Furthermore, the vector remains generally in this direction throughout the process of normal atrial depolarization. Because this direction is generally in the positive directions of the axes of the three standard bipolar limb leads I, II, and III, the electrocardiograms recorded from the atria during depolarization are also usually positive in all three of these leads, as shown in Figure 12-9. This record of atrial depolarization is known as the atrial P wave.

Repolarization of the Atria—the Atrial T Wave. Spread of depolarization through the atrial muscle is *much slower than in the ventricles* because the atria have no Purkinje system for fast conduction of the depolarization signal. Therefore, the musculature around the sinus node becomes depolarized a long time before the musculature in distal parts of the atria. Because of this, *the area in the atria that also becomes repolarized first is the sinus nodal region, the area that had originally become depolarized first.* Thus, when repolarization begins, the region around the sinus node becomes positive with respect to the rest of the atria. Therefore, the atrial repolarization

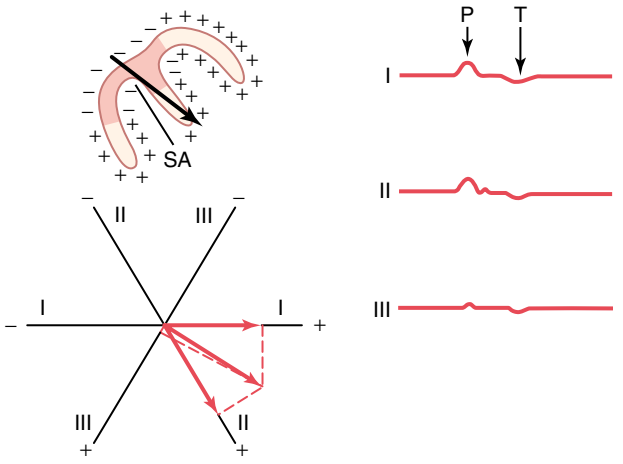


Figure 12-9 Depolarization of the atria and generation of the P wave, showing the maximum vector through the atria and the resultant vectors in the three standard leads. At the right are the atrial P and T waves. SA, sinoatrial node.

vector is *backward to the vector of depolarization.* (Note that this is opposite to the effect that occurs in the ventricles.) Therefore, as shown to the right in Figure 12-9, the so-called atrial T wave follows about 0.15 second after the atrial P wave, but this T wave is on the opposite side of the zero reference line from the P wave; that is, it is normally negative rather than positive in the three standard bipolar limb leads.

In the normal electrocardiogram, the *atrial* T wave appears at about the same time that the QRS complex of the ventricles appears. Therefore, it is almost always totally obscured by the large *ventricular* QRS complex, although in some very abnormal states, it does appear in the recorded electrocardiogram.

Vectorcardiogram

It has been noted in the discussion up to this point that the vector of current flow through the heart changes rapidly as the impulse spreads through the myocardium. It changes in two aspects: First, the vector increases and decreases in length because of increasing and decreasing voltage of the vector. Second, the vector changes direction because of changes in the average direction of the electrical potential from the heart. The so-called *vectorcardiogram* depicts these changes at different times during the cardiac cycle, as shown in Figure 12-10.

In the large vectorcardiogram of Figure 12-10, point 5 is the *zero reference point,* and this point is the negative end of all the successive vectors. While the heart muscle is polarized between heartbeats, the positive end of the vector remains at the zero point because there is no vectorial electrical potential. However, as soon as current begins to flow through the ventricles at the beginning of ventricular depolarization, the positive end of the vector leaves the zero reference point.

When the septum first becomes depolarized, the vector extends downward toward the apex of the ventricles, but it is relatively weak, thus generating the first portion of the ventricular vectorcardiogram, as shown by the positive end of vector 1. As more of the ventricular muscle becomes depolarized, the vector becomes stronger and stronger, usually swinging slightly to one side. Thus, vector 2 of Figure 12-10 represents the state of depolarization of the ventricles about 0.02 second after vector 1. After another 0.02 second, vector 3 represents the potential, and vector 4 occurs in another 0.01 second. Finally, the ventricles become totally depolarized, and the vector becomes zero once again, as shown at point 5.

The elliptical figure generated by the positive ends of the vectors is called the *QRS vectorcardiogram.* Vectorcardiograms can be recorded on an oscilloscope by connecting body surface electrodes from the neck and lower abdomen to the vertical plates of the oscilloscope and connecting chest surface electrodes from each side of the heart to the horizontal plates. When the vector changes, the spot of light on the oscilloscope follows the course of the positive end of the changing vector, thus inscribing the vectorcardiogram on the oscilloscopic screen.

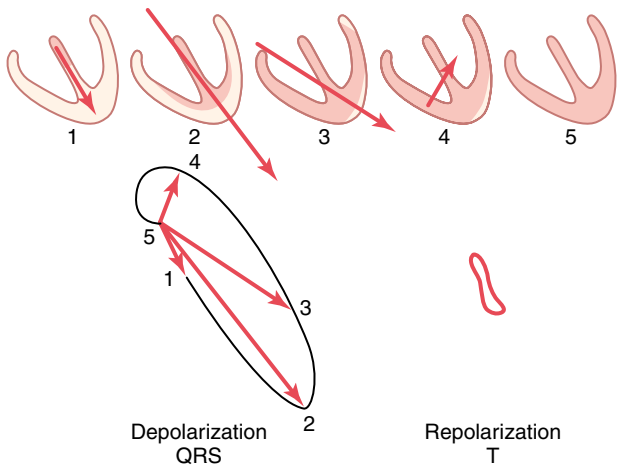


Figure 12-10 QRS and T vectorcardiograms.

Mean Electrical Axis of the Ventricular QRS—and Its Significance

The vectorcardiogram during ventricular depolarization (the QRS vectorcardiogram) shown in Figure 12-10 is that of a normal heart. Note from this vectorcardiogram that the preponderant direction of the vectors of the ventricles during depolarization is mainly toward the apex of the heart. That is, during most of the cycle of ventricular depolarization, the direction of the electrical potential (negative to positive) is from the base of the ventricles toward the apex. This preponderant direction of the potential during depolarization is called the *mean electrical axis of the ventricles.* The mean electrical axis of the normal ventricles is 59 degrees. In many pathological conditions of the heart, this direction changes markedly, sometimes even to opposite poles of the heart.

Determining the Electrical Axis from Standard Lead Electrocardiograms

Clinically, the electrical axis of the heart is usually estimated from the standard bipolar limb lead electrocardiograms rather than from the vectorcardiogram. Figure 12-11 shows a method for doing this. After recording the standard leads, one determines the net potential and polarity of the recordings in leads I and III. In lead I of Figure 12-11, the recording is positive, and in lead III, the recording is mainly positive but negative during part of the cycle. If any part of a recording is negative, *this negative potential is subtracted from the positive part of the potential* to determine the *net potential* for that lead, as shown by the arrow to the right of the QRS complex for lead III. Then each net potential for leads I and III is plotted on the axes of the respective leads, with the base of the potential at the point of intersection of the axes, as shown in Figure 12-11.

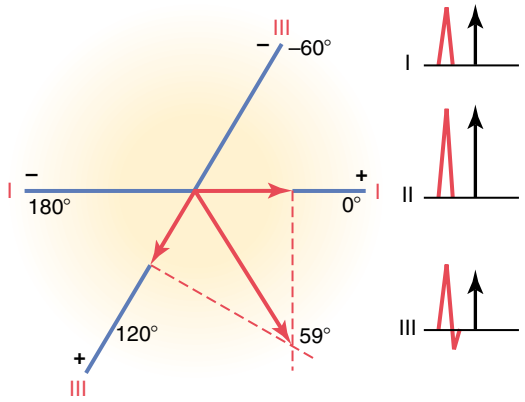


Figure 12-11 Plotting the mean electrical axis of the ventricles from two electrocardiographic leads (leads I and III).

If the net potential of lead I is positive, it is plotted in a positive direction along the line depicting lead I. Conversely, if this potential is negative, it is plotted in a negative direction. Also, for lead III, the net potential is placed with its base at the point of intersection, and, if positive, it is plotted in the positive direction along the line depicting lead III. If it is negative, it is plotted in the negative direction.

To determine the vector of the total QRS ventricular mean electrical potential, one draws perpendicular lines (the dashed lines in the figure) from the apices of leads I and III, respectively. The point of intersection of these two perpendicular lines represents, by vectorial analysis, the apex of the *mean* QRS vector in the ventricles, and the point of intersection of the lead I and lead III axes represents the negative end of the mean vector. Therefore, the *mean QRS vector* is drawn between these two points. The approximate average potential generated by the ventricles during depolarization is represented by the length of this mean QRS vector, and the mean electrical axis is represented by the direction of the mean vector. Thus, the orientation of the mean electrical axis of the normal ventricles, as determined in Figure 12-11, is 59 degrees positive (+59 degrees).

Abnormal Ventricular Conditions That Cause Axis Deviation

Although the mean electrical axis of the ventricles averages about 59 degrees, this axis can swing even in the normal heart from about 20 degrees to about 100 degrees. The causes of the normal variations are mainly anatomical differences in the Purkinje distribution system or in the musculature itself of different hearts. However, a number of abnormal conditions of the heart can cause axis deviation beyond the normal limits, as follows.

Change in the Position of the Heart in the Chest. If the heart itself is angulated to the left, the mean electrical axis of the heart also *shifts to the left*. Such shift occurs (1) at the end of deep expiration, (2) when a person lies down, because the abdominal contents press upward against the diaphragm, and (3) quite frequently in obese people whose diaphragms normally press upward against the heart all the time due to increased visceral adiposity.

Likewise, angulation of the heart to the right causes the mean electrical axis of the ventricles to *shift to the right.* This occurs (1) at the end of deep inspiration, (2) when a person stands up, and (3) normally in tall, lanky people whose hearts hang downward.

Hypertrophy of One Ventricle. When one ventricle greatly hypertrophies, *the axis of the heart shifts toward the hypertrophied ventricle* for two reasons. First, a far greater quantity of muscle exists on the hypertrophied side of the heart than on the other side, and this allows generation of greater electrical potential on that side. Second, more time is required for the depolarization wave to travel through the hypertrophied ventricle than through the normal ventricle. Consequently, the *normal* ventricle becomes depolarized considerably in advance of the *hypertrophied* ventricle, and this causes a strong vector from the normal side of the heart toward the hypertrophied side, which remains strongly positively charged. Thus, the axis deviates toward the hypertrophied ventricle.

Vectorial Analysis of Left Axis Deviation Resulting from Hypertrophy of the Left Ventricle. Figure 12-12 shows the three standard bipolar limb lead electrocardiograms. Vectorial analysis demonstrates left axis deviation with mean electrical axis pointing in the –15-degree direction. This is a typical electrocardiogram caused by increased muscle mass of the left ventricle. In this instance, the axis deviation was caused by *hypertension* (high arterial blood pressure), which caused the left ventricle to hypertrophy so that it could pump blood against elevated systemic arterial pressure. A similar picture of left axis deviation occurs when the

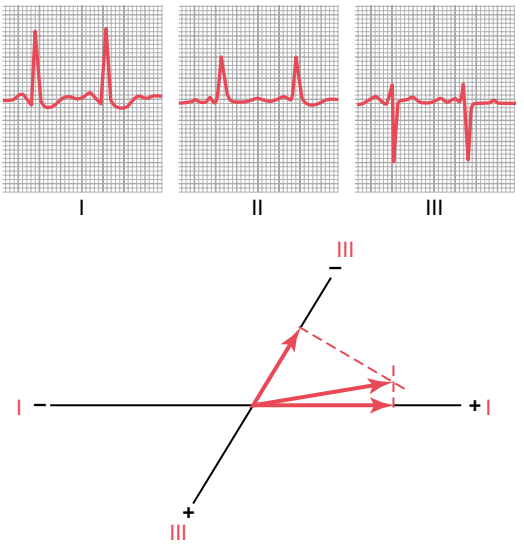


Figure 12-12 Left axis deviation in a ***hypertensive heart (hypertrophic left ventricle)***. Note the slightly prolonged QRS complex as well.

left ventricle hypertrophies as a result of *aortic valvular stenosis, aortic valvular regurgitation,* or any number of *congenital heart conditions* in which the left ventricle enlarges while the right ventricle remains relatively normal in size.

Vectorial Analysis of Right Axis Deviation Resulting from Hypertrophy of the Right Ventricle. The electrocardiogram of Figure 12-13 shows intense right axis deviation, to an electrical axis of 170 degrees, which is 111 degrees to the right of the normal 59-degree mean ventricular QRS axis. The right axis deviation demonstrated in this figure was caused by hypertrophy of the right ventricle as a result of *congenital pulmonary valve stenosis.* Right axis deviation also can occur in other congenital heart conditions that cause hypertrophy of the right ventricle, such as *tetralogy of Fallot* and *interventricular septal defect.*

Bundle Branch Block Causes Axis Deviation. Ordinarily, the lateral walls of the two ventricles depolarize at almost the same instant because both the left and the right bundle branches of the Purkinje system transmit the cardiac impulse to the two ventricular walls at almost the same instant. As a result, the potentials generated by the two ventricles (on the two opposite sides of the heart) almost neutralize each other. But if only one of the major bundle branches is blocked, the cardiac impulse spreads through the normal ventricle long before it spreads through the other. Therefore, depolarization of the two ventricles does not occur even nearly simultaneously, and the depolarization potentials do not neutralize each other. As a result, axis deviation occurs as follows.

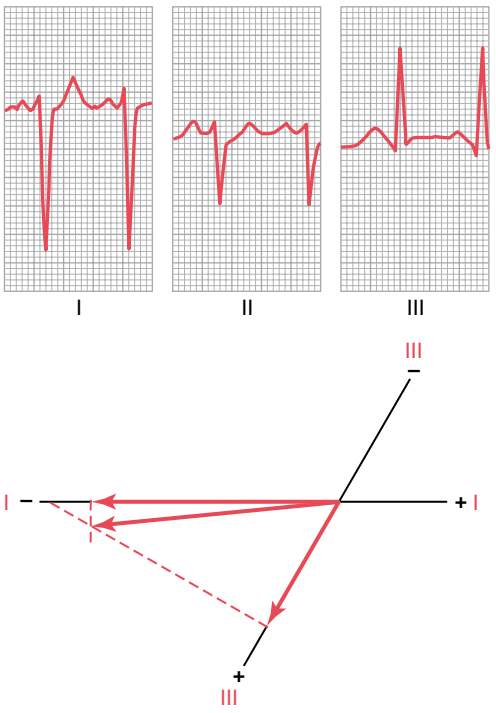


Figure 12-13 High-voltage electrocardiogram in *congenital pulmonary valve stenosis* ***with right ventricular hypertrophy.*** Intense right axis deviation and a slightly prolonged QRS complex also are seen.

Vectorial Analysis of Left Axis Deviation in Left Bundle Branch Block. When the left bundle branch is blocked, cardiac depolarization spreads through the right ventricle two to three times as rapidly as through the left ventricle. Consequently, much of the left ventricle remains polarized for as long as 0.1 second after the right ventricle has become totally depolarized. Thus, the right ventricle becomes electronegative, whereas the left ventricle remains electropositive during most of the depolarization process, and a strong vector projects from the right ventricle toward the left ventricle. In other words, there is intense left axis deviation of about –50 degrees because the positive end of the vector points toward the left ventricle. This is demonstrated in Figure 12-14, which shows typical left axis deviation resulting from left bundle branch block.

Because of slowness of impulse conduction when the Purkinje system is blocked, in addition to axis deviation, the duration of the QRS complex is greatly prolonged because of extreme slowness of depolarization in the affected side of the heart. One can see this by observing the excessive widths of the QRS waves in Figure 12-14. This is discussed in greater detail later in the chapter. This extremely prolonged QRS complex differentiates bundle branch block from axis deviation caused by hypertrophy.

Vectorial Analysis of Right Axis Deviation in Right Bundle Branch Block. When the right bundle branch is blocked, the left ventricle depolarizes far more rapidly than the right ventricle, so the left side of the ventricles becomes electronegative as long as 0.1 second before the right. Therefore, a strong vector develops, with its negative end toward the left ventricle and its positive end toward the right ventricle. In other words, intense right axis deviation occurs. Right axis deviation caused by right bundle branch block is demonstrated, and its vector is

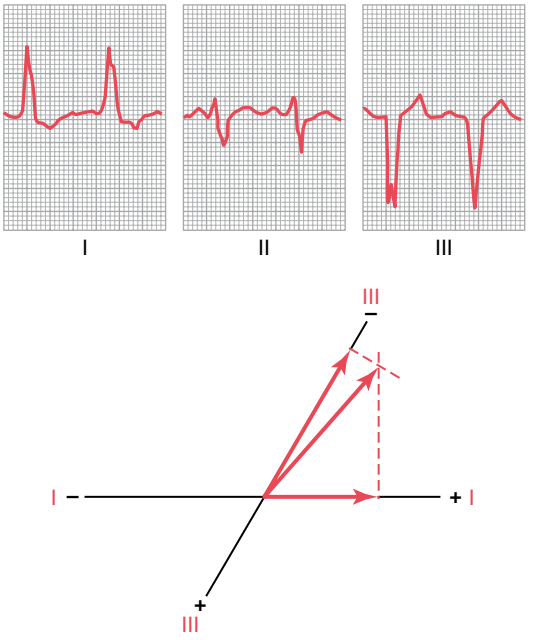


Figure 12-14 Left axis deviation caused by *left bundle branch block.* Note also the greatly prolonged QRS complex.

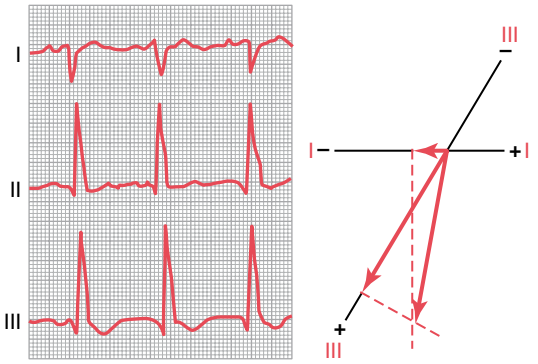


Figure 12-15 Right axis deviation caused by *right bundle branch block*. Note also the greatly prolonged QRS complex.

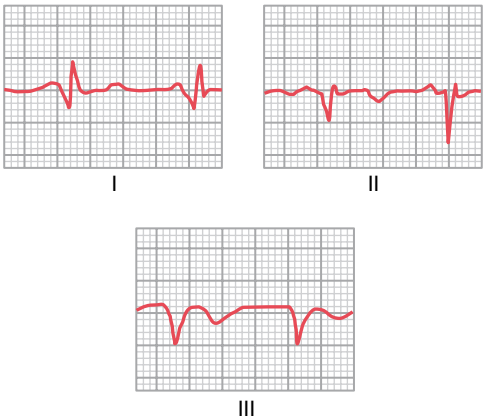


Figure 12-16 Low-voltage electrocardiogram following local damage throughout the ventricles caused by *previous myocardial infarction*.

analyzed, in Figure 12-15, which 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. 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 electrocardiogram.

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 causes generation of increased quantities of 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 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 electrocardiogram 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 electrocardiograms are decreased.

Pulmonary emphysema can decrease the electrocardiographic potentials, but for a different reason than that of pericardial effusion. In pulmonary emphysema, conduction of electrical current through the lungs is depressed considerably because of 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 normally. Therefore, the lungs act as an insulator to prevent spread of electrical voltage from the heart to the surface of the body, and this results in decreased electrocardiographic potentials in the various leads.

Prolonged and Bizarre Patterns of the QRS Complex

Prolonged QRS Complex as a Result of Cardiac Hypertrophy or Dilatation

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 dilatation of the left or right ventricle, the QRS complex may be prolonged to 0.09 to 0.12 second.

Prolonged QRS Complex Resulting from Purkinje System Blocks

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 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 pathological block somewhere in the ventricular conduction system, as shown by the electrocardiograms 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 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 itself, often cause part of the heart to remain partially or totally *depolarized all the time.* When this occurs, current flows between the pathologically depolarized and the normally polarized areas even between heartbeats. This 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 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 (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,* 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 electrocardiogram 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 electrocardiogram, 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—the Zero Reference Potential for Analyzing Current of Injury

One might think that the electrocardiograph machines for recording electrocardiograms could determine when no current is flowing around the heart. However, many

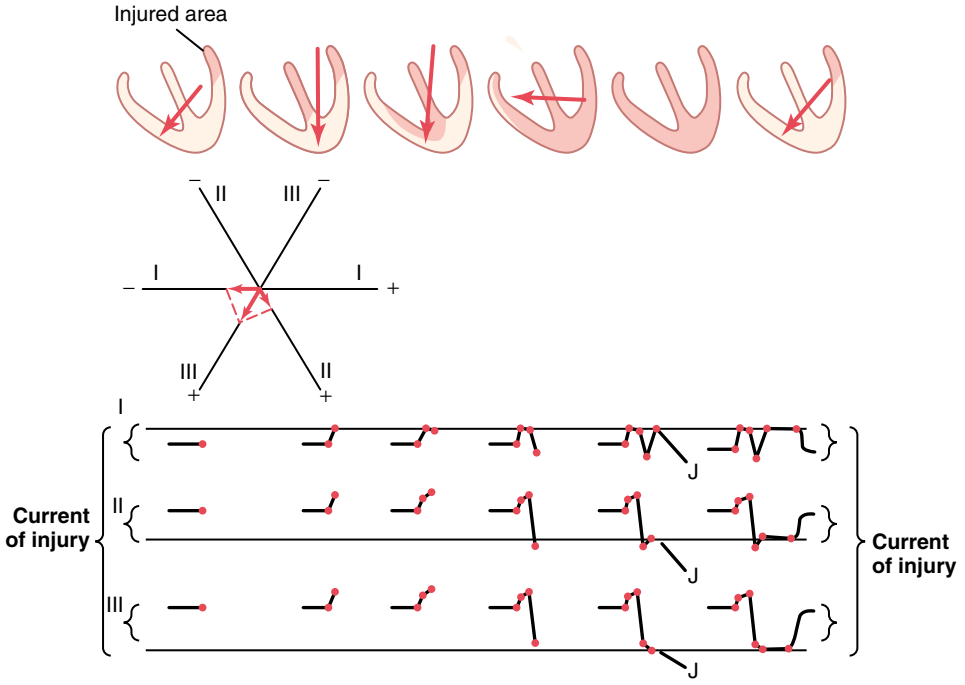


Figure 12-17 Effect of a current of injury on the electrocardiogram.

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 electrocardiogram.

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 electrocardiogram, as shown in Figure 12-18.

Then, for analysis of the electrical axis of the injury potential caused by a current of injury, a horizontal line is drawn in the electrocardiogram for each lead at the level of the J point. This horizontal line is then the *zero potential level* in the electrocardiogram 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 electrocardiograms (leads I and III) from an injured heart. Both records show injury potentials. In other words, the J point of each of these two electrocardiograms 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 electrocardiogram 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

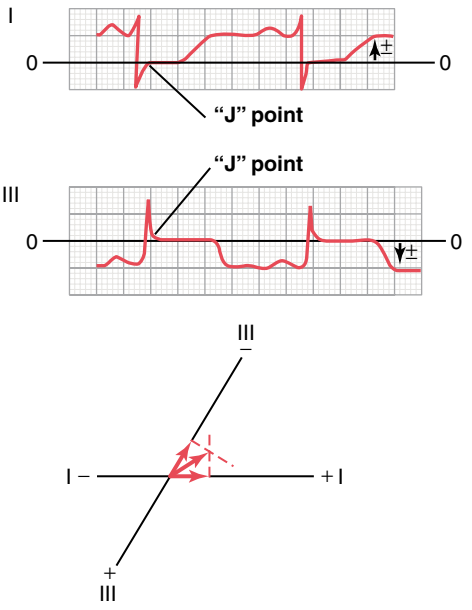


Figure 12-18 J point as the zero reference potential of the electrocardiograms for leads I and III. Also, the method for plotting the axis of the injury potential is shown by the lowermost panel.

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.

This analysis is obviously complex. However, it is essential that the student go over 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 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 electrocardiograms recorded after acute coronary thrombosis is the current of injury.

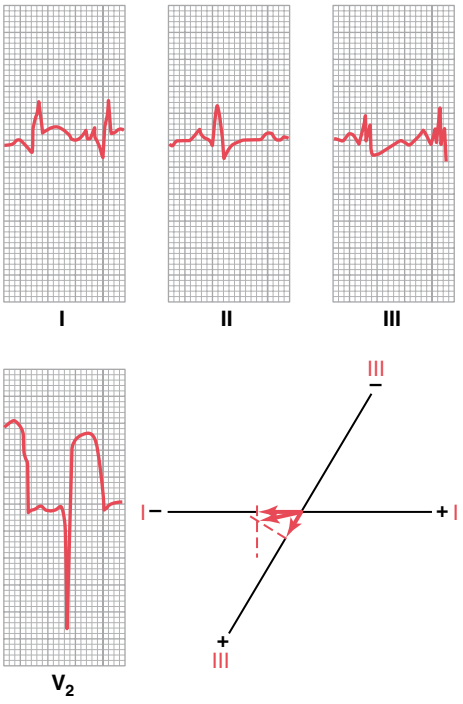


Figure 12-19 Current of injury in *acute anterior wall infarction*. Note the intense injury potential in lead V_2.

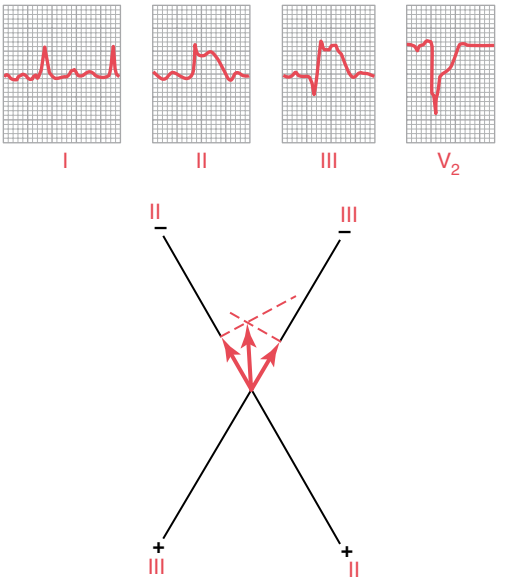


Figure 12-20 Injury potential in ***acute posterior wall, apical infarction.***

Acute Anterior Wall Infarction. Figure 12-19 shows the electrocardiogram 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 important diagnostic feature of this electrocardiogram is the intense injury potential in chest lead V_2. If one draws a zero horizontal potential line through the J point of this electrocardiogram, 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.*

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 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 particular electrocardiogram, 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 electrocardiogram 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 (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 electrocardiogram is the basis for diagnosing posterior wall infarction.

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. By 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 must 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 V_3 chest lead from a patient with acute posterior wall infarction, demonstrating changes in the electrocardiogram from the day of the attack to 1 week later, 3 weeks later, and finally 1 year later. From this electrocardiogram, one can see that the injury potential is strong immediately after the acute attack (T-P segment 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 electrocardiogram 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.

Conversely, in some patients with myocardial infarction, the infarcted area never redevelops adequate coronary blood supply. Often, some of the heart muscle

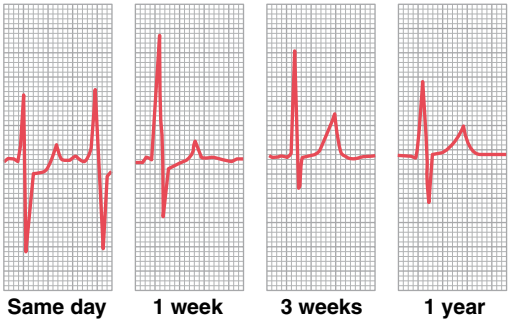


Figure 12-21 Recovery of the myocardium after ***moderate posterior wall infarction,*** demonstrating disappearance of the injury potential that is present on the first day after the infarction and still slightly present at 1 week.

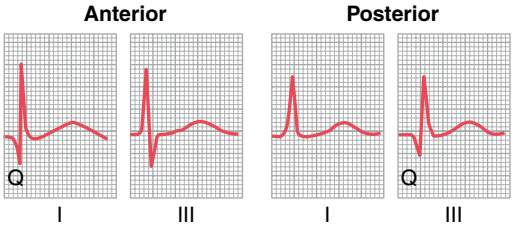


Figure 12-22 Electrocardiograms of anterior and posterior wall infarctions that occurred about 1 year previously, showing a Q wave in lead I in anterior wall infarction and a Q wave in lead III in ***posterior wall infarction.***

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 electrocardiogram 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, it was 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.

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 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; this 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 electrocardiogram. 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 as a Cause of 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.

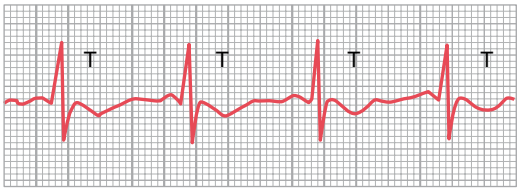


Figure 12-23 Inverted T wave resulting from mild *ischemia at the apex* of the ventricles.

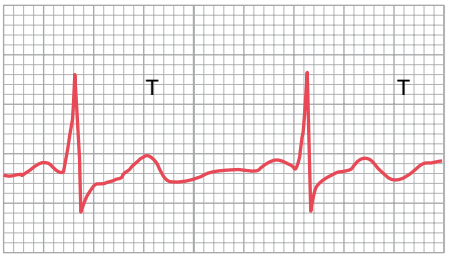


Figure 12-24 Biphasic T wave caused by *digitalis toxicity*.

Mild ischemia is by far the most common cause of shortening of depolarization of cardiac muscle because this 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 electrocardiogram, 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.

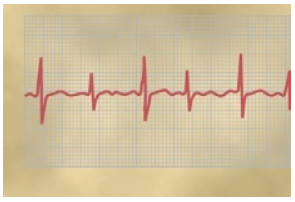
Effect of Digitalis on the T Wave. As discussed in Chapter 22, digitalis is a drug that can be used during coronary insufficiency to increase the strength of cardiac muscle contraction. But when overdosages of digitalis are given, depolarization duration in one part of the ventricles may be increased 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.

Bibliography

See bibliography for Chapter 13.

CHAPTER 13

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 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/min. 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.

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 increases about 10 beats/min for each degree of 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/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 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 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

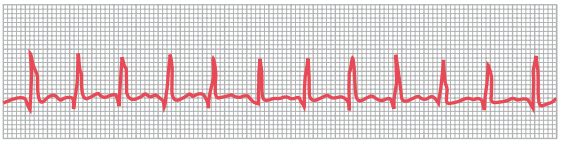


Figure 13-1 Sinus tachycardia (lead I).

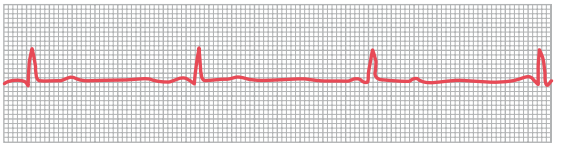


Figure 13-2 Sinus bradycardia (lead III).

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 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 percent 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 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. 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.

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

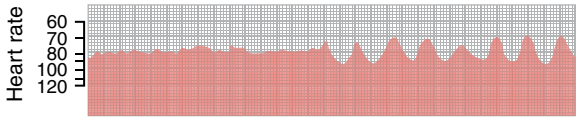


Figure 13-3 Sinus arrhythmia as recorded by a cardiotachometer. To the left is the record when the subject was breathing normally; to the right, when breathing deeply.

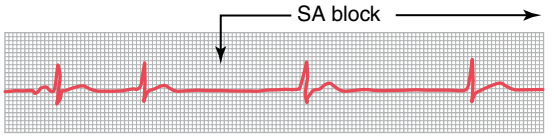


Figure 13-4 Sinoatrial nodal block, with A-V nodal rhythm during the block period (lead III).

sudden cessation of P waves, with resultant standstill of the atria. However, the ventricles pick up a new rhythm, 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 conductivity from the atria to the ventricles. Inflammation results frequently from 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

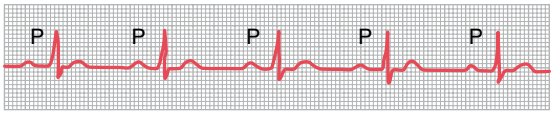


Figure 13-5 Prolonged P-R interval caused by first degree A-V heart block (lead II).

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 is sometimes strong enough to pass through the bundle into the ventricles and sometimes 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 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 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.

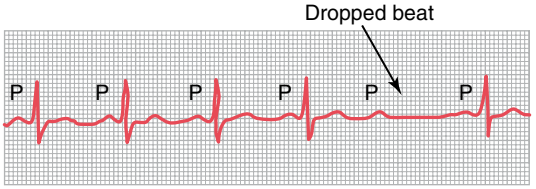


Figure 13-6 Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead V_3).

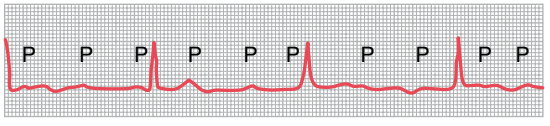


Figure 13-7 Complete A-V block (lead II).

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.

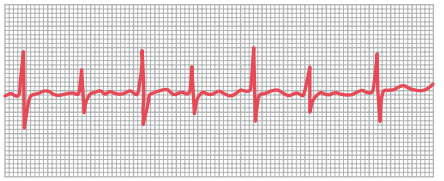


Figure 13-8 Partial intraventricular block—"electrical alternans" (lead III).

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.

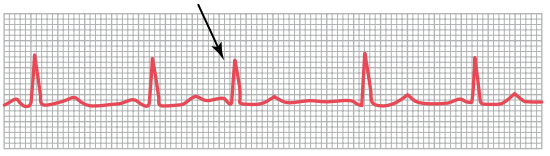


Figure 13-9 Atrial premature beat (lead I).

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 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

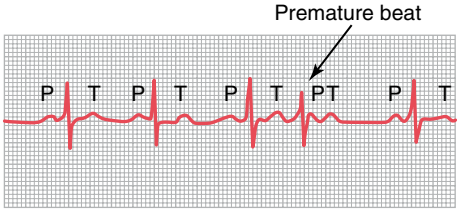


Figure 13-10 A-V nodal premature contraction (lead III).

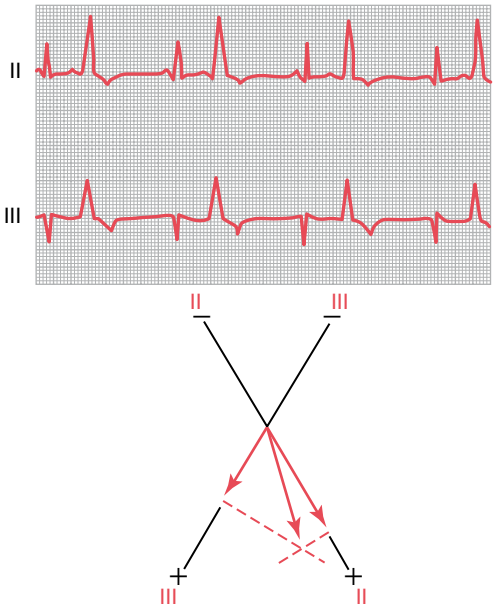


Figure 13-11 Premature ventricular contractions (PVCs) demonstrated by the large abnormal QRS-T complexes (leads II and III). Axis of the premature contractions is plotted in accordance with the principles of vectorial analysis explained in Chapter 12; this shows the origin of the PVC to be near the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

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; 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, 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. 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.

Disorders of Cardiac Repolarization—The Long QT Syndromes. Recall that the Q wave corresponds to ventricular depolarization while 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 following the action potential cause prolonged ventricular action potentials and therefore excessively long Q-T intervals on the electrocardiogram, a condition called *long QT syndrome* (LQTS).

The major reason that the long QT syndrome is of concern is that delayed repolarization of ventricular muscle increases a person's susceptibility to develop 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 LQTS are rare disorders caused by mutations of sodium or potassium channel genes. At least 10 different mutations of these genes that can cause 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), others exhibit fainting and ventricular arrhythmias that may be precipitated by physical exercise, intense emotions such as fright or anger, or when startled by a noise. The ventricular arrhythmias associated with LQTS can, in some cases, deteriorate into ventricular fibrillation and sudden death.

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

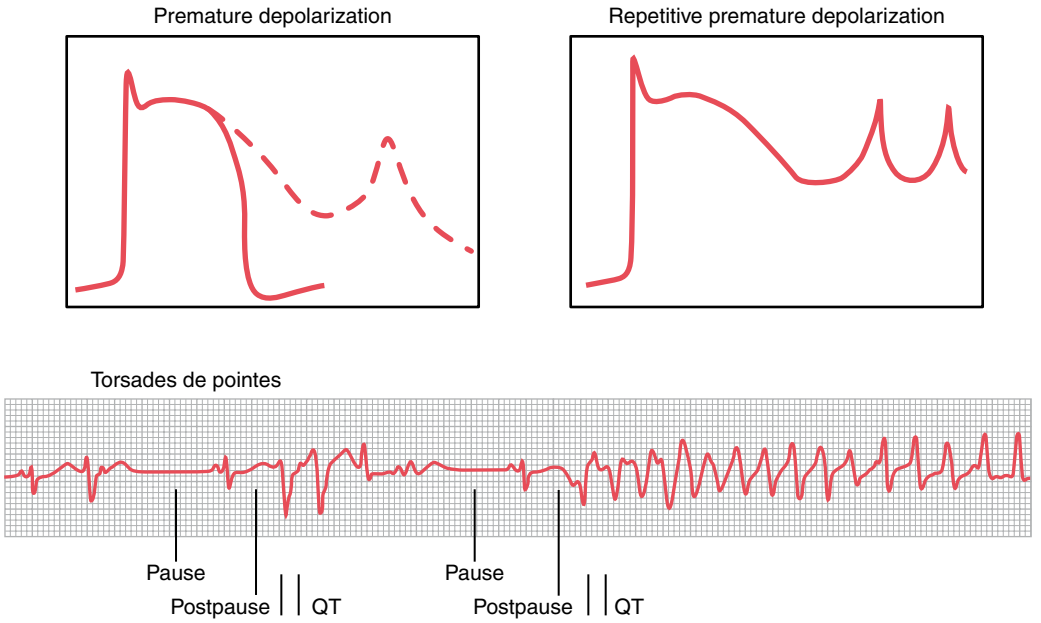


Figure 13-12 Development of arrhythmias in long QT syndrome (LQTS). When the ventricular muscle fiber action potential is prolonged as a result of delayed repolarization, a premature depolarization (***dashed line in top left figure***) may occur before complete repolarization. Repetitive premature depolarizations (***right top 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. (Redrawn from Murray KT, Roden DM: Disorders of cardiac repolarization: the long QT syndromes. In: Crawford MG, DiMarco JP [eds]: Cardiology. London: Mosby, 2001.)

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 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 13-13 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

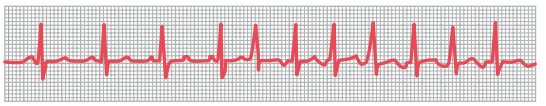


Figure 13-13 Atrial paroxysmal tachycardia—onset in middle of record (lead I).

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 13-14 shows a typical short paroxysm of ventricular tachycardia. The electrocardiogram of ventricular paroxysmal tachycardia has the appearance of a series of

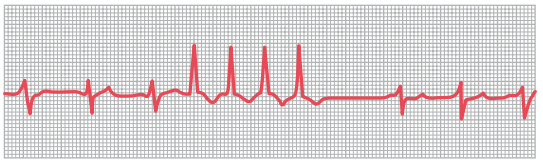


Figure 13-14 Ventricular paroxysmal tachycardia (lead III).

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 13-15 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. 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.

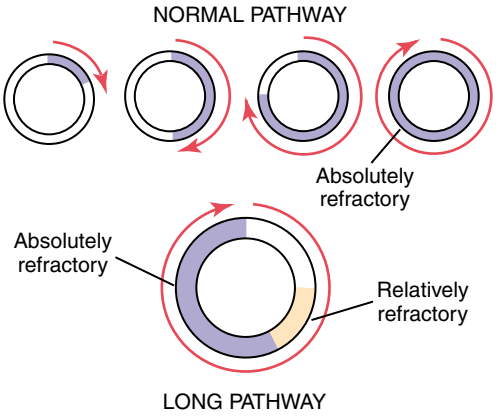


Figure 13-15 Circus movement, showing annihilation of the impulse in the short pathway and continued propagation of the impulse in the long pathway.

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 13-15. 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 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 13-16, 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 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.

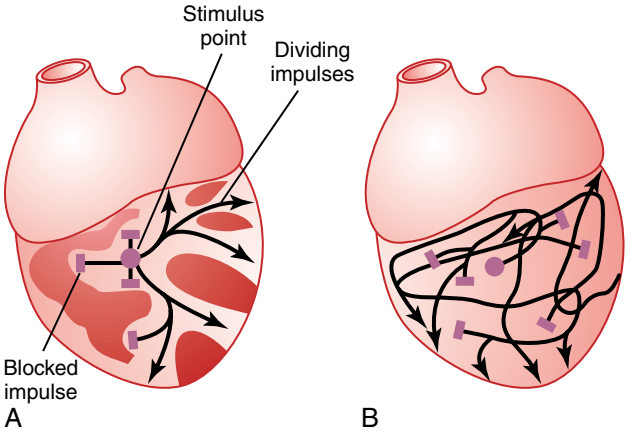


Figure 13-16 *A,* Initiation of fibrillation in a heart when patches of refractory musculature are present. *B,* Continued propagation of *fibrillatory impulses* in the fibrillating ventricle.

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 13-16 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 13-17) and ordinarily shows no tendency toward a regular rhythm of any type. During the first few seconds

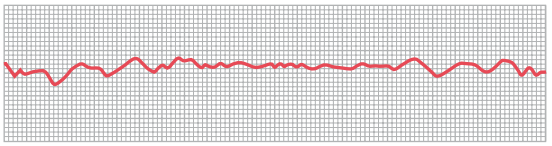


Figure 13-17 Ventricular fibrillation (lead II).

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.

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 13-18, the

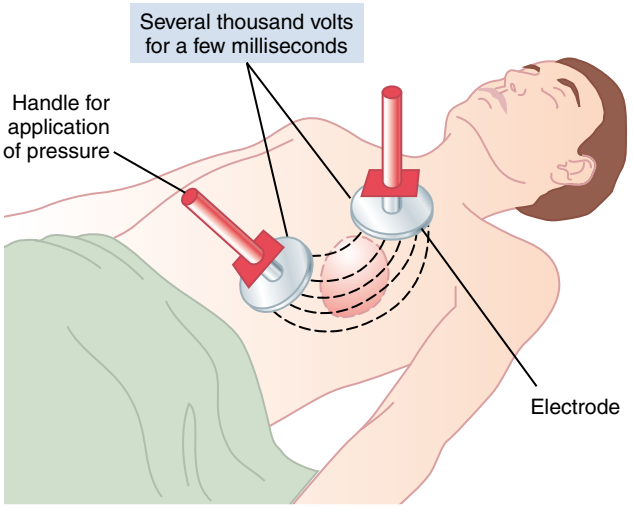


Figure 13-18 Application of electrical current to the chest to stop ventricular fibrillation.

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.

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.

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 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 percent. 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 13-19 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 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 13-19, 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.

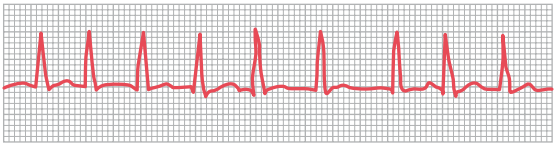


Figure 13-19 Atrial fibrillation (lead I). The waves that can be seen are ventricular QRS and T waves.

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 13-20. 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, there are usually two to three beats of the atria for every single beat of the ventricles.

Figure 13-21 shows a typical electrocardiogram in atrial flutter. The P waves are strong because of contraction of semicoordinate masses of muscle. However, note

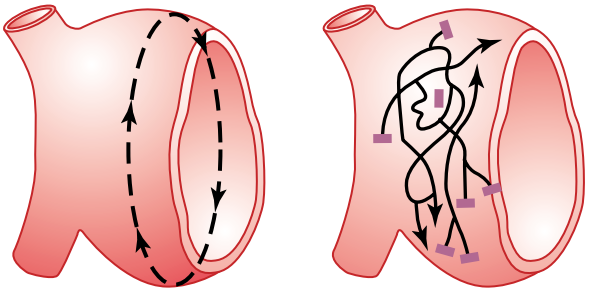


Figure 13-20 Pathways of impulses in atrial flutter and atrial fibrillation.

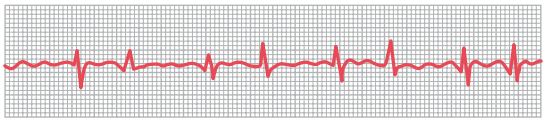


Figure 13-21 Atrial flutter—2:1 and 3:1 atrial to ventricle rhythm (lead I).

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.* This 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 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 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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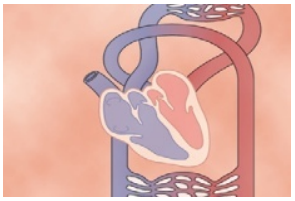
Overview of the Circulation; Biophysics of Pressure, Flow, and Resistance



The function of the circulation is to service the needs of the body tissues—to transport nutrients to the body tissues, to transport waste products away, to transport hormones from one part of the body to another, and, in general, to maintain an appropriate environment in all the tissue fluids of the body for optimal survival and function of the cells.

The rate of blood flow through many tissues is controlled mainly in response to tissue need for nutrients. In some organs, such as the kidneys, the circulation serves additional functions. Blood flow to the kidney, for example, is far in excess of its metabolic requirements and is related to its excretory function, which demands that a large volume of blood be filtered each minute.

The heart and blood vessels, in turn, are controlled to provide the necessary cardiac output and arterial pressure to cause the needed tissue blood flow. What are the mechanisms for controlling blood volume and blood flow, and how does this relate to all the other functions of the circulation? These are some of the topics and questions that we discuss in this section on the circulation.

Physical Characteristics of the Circulation

The circulation, shown in Figure 14-1, is divided into the *systemic circulation* and the *pulmonary circulation.* Because the systemic circulation supplies blood flow to all the tissues of the body except the lungs, it is also called the *greater circulation* or *peripheral circulation.*

Functional Parts of the Circulation. Before discussing the details of circulatory function, it is important to understand the role of each part of the circulation.

The function of the *arteries* is to transport blood *under high pressure* to the tissues. For this reason, the arteries have strong vascular walls, and blood flows at a high velocity in the arteries.

The *arterioles* are the last small branches of the arterial system; they act as *control conduits* through which blood is released into the capillaries. Arterioles have strong muscular walls that can close the arterioles completely or can, by relaxing, dilate the vessels severalfold, thus having the capability of vastly altering blood flow in each tissue in response to its needs.

The function of the *capillaries* is to exchange fluid, nutrients, electrolytes, hormones, and other substances between the blood and the interstitial fluid. To serve this role, the capillary walls are very thin and have numerous minute *capillary pores* permeable to water and other small molecular substances.

The *venules* collect blood from the capillaries and gradually coalesce into progressively larger veins.

The *veins* function as conduits for transport of blood from the venules back to the heart; equally important, they serve as a major reservoir of extra blood. Because the pressure in the venous system is very low, the venous walls are thin. Even so, they are muscular enough to contract or expand and thereby act as a controllable reservoir for the extra blood, either a small or a large amount, depending on the needs of the circulation.

Volumes of Blood in the Different Parts of the Circulation. Figure 14-1 gives an overview of the circulation and lists the percentage of the total blood volume in major segments of the circulation. For instance, about 84 percent of the entire blood volume of the body is in the systemic circulation and 16 percent is in the heart and lungs. Of the 84 percent in the systemic circulation, 64 percent is in the veins, 13 percent in the arteries, and 7 percent in the systemic arterioles and capillaries. The heart contains 7 percent of the blood, and the pulmonary vessels, 9 percent.

Most surprising is the low blood volume in the capillaries. It is here, however, that the most important function of the circulation occurs, diffusion of substances back and forth between the blood and the tissues. This function is discussed in detail in Chapter 16.

Cross-Sectional Areas and Velocities of Blood Flow. If all the *systemic vessels* of each type were put side by side, their approximate total cross-sectional areas for the average human being would be as follows:

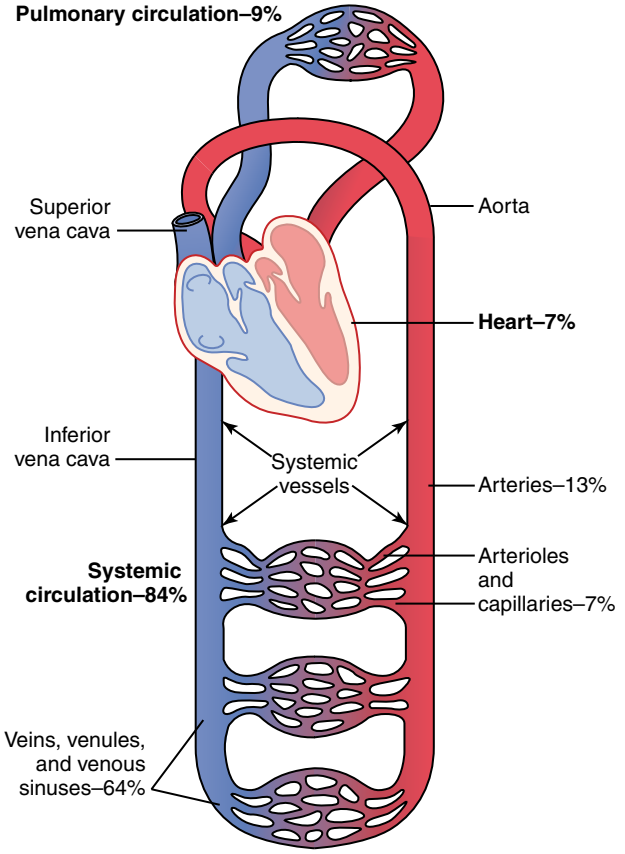


Figure 14-1 Distribution of blood (in percentage of total blood) in the different parts of the circulatory system.

Vessel	Cross-Sectional Area (cm^2)
Aorta	2.5
Small arteries	20
Arterioles	40
Capillaries	2500
Venules	250
Small veins	80
Venae cavae	8

Note particularly the much larger cross-sectional areas of the veins than of the arteries, averaging about four times those of the corresponding arteries. This explains the large blood storage capacity of the venous system in comparison with the arterial system.

Because the same volume of blood flow (F) must pass through each segment of the circulation each minute, the velocity of blood flow (v) is inversely proportional to vascular cross-sectional area (A):

$$v = F/A$$

Thus, under resting conditions, the velocity averages about 33 cm/sec in the aorta but only 1/1000 as rapidly in the capillaries, about 0.3 mm/sec. However, because the capillaries have a typical length of only 0.3 to 1 millimeter, the blood remains in the capillaries for only 1 to 3 seconds. This short time is surprising because all diffusion of nutrient food substances and electrolytes that occurs through the capillary walls must do so in this short time.

Pressures in the Various Portions of the Circulation. Because the heart pumps blood continually into the aorta, the mean pressure in the aorta is high, averaging about 100 mm Hg. Also, because heart pumping is pulsatile, the arterial pressure alternates between a *systolic pressure level* of 120 mm Hg and a *diastolic pressure level* of 80 mm Hg, as shown on the left side of Figure 14-2.

As the blood flows through the *systemic circulation,* its mean pressure falls progressively to about 0 mm Hg by the time it reaches the termination of the venae cavae where they empty into the right atrium of the heart.

The pressure in the systemic capillaries varies from as high as 35 mm Hg near the arteriolar ends to as low as 10 mm Hg near the venous ends, but their average "functional" pressure in most vascular beds is about 17 mm Hg, a pressure low enough that little of the plasma leaks through the minute *pores* of the capillary walls, even though nutrients can *diffuse* easily through these same pores to the outlying tissue cells.

Note at the far right side of Figure 14-2 the respective pressures in the different parts of the *pulmonary circulation.* In the pulmonary arteries, the pressure is pulsatile, just as in the aorta, but the pressure is far less: *pulmonary artery systolic pressure* averages about 25 mm Hg and *diastolic pressure* 8 mm Hg, with a mean pulmonary arterial pressure of only 16 mm Hg. The mean pulmonary capillary pressure averages only 7 mm Hg. Yet the total blood flow through the lungs each minute is the same as through the systemic circulation. The low pressures of the pulmonary system are in accord with the needs of the lungs because all that is required is to expose the blood in the pulmonary capillaries to oxygen and other gases in the pulmonary alveoli.

Basic Principles of Circulatory Function

Although the details of circulatory function are complex, there are three basic principles that underlie all functions of the system.

1. **The rate of blood flow to each tissue of the body is almost always precisely controlled in relation to the tissue need.** When tissues are active, they need a greatly increased supply of nutrients and therefore much more blood flow than when at rest—occasionally as much as 20 to 30 times the resting level. Yet the heart normally cannot increase its cardiac output more than four to seven times greater than resting levels. Therefore, it is not possible simply to increase blood flow everywhere in the body when a particular tissue demands increased flow. Instead, the microvessels of each tissue continuously monitor tissue needs, such as the availability of oxygen and other nutrients

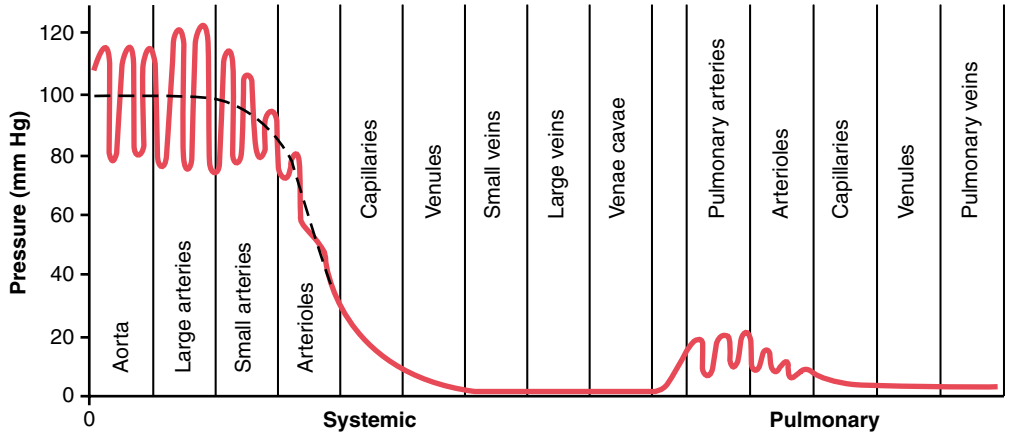


Figure 14-2 Normal blood pressures in the different portions of the circulatory system when a person is lying in the horizontal position.

and the accumulation of carbon dioxide and other tissue waste products, and these in turn act directly on the local blood vessels, dilating or constricting them, to control local blood flow precisely to that level required for the tissue activity. Also, nervous control of the circulation from the central nervous system and hormones provide additional help in controlling tissue blood flow.

2. **The cardiac output is controlled mainly by the sum of all the local tissue flows.** When blood flows through a tissue, it immediately returns by way of the veins to the heart. The heart responds automatically to this increased inflow of blood by pumping it immediately back into the arteries. Thus, the heart acts as an automaton, responding to the demands of the tissues. The heart, however, often needs help in the form of special nerve signals to make it pump the required amounts of blood flow.
3. **Arterial pressure regulation is generally independent of either local blood flow control or cardiac output control.** The circulatory system is provided with an extensive system for controlling the arterial blood pressure. For instance, if at any time the pressure falls significantly below the normal level of about 100 mm Hg, within seconds a barrage of nervous reflexes elicits a series of circulatory changes to raise the pressure back toward normal. The nervous signals especially (a) increase the force of heart pumping, (b) cause contraction of the large venous reservoirs to provide more blood to the heart, and (c) cause generalized constriction of most of the arterioles throughout the body so that more blood accumulates in the large arteries to increase the arterial pressure. Then, over more prolonged periods, hours and days, the kidneys play an additional major role in pressure control both by secreting pressure-controlling hormones and by regulating the blood volume.

Thus, in summary, the needs of the individual tissues are served specifically by the circulation. In the remainder of this chapter, we begin to discuss the basic details of the management of tissue blood flow and control of cardiac output and arterial pressure.

Interrelationships of Pressure, Flow, and Resistance

Blood flow through a blood vessel is determined by two factors: (1) *pressure difference* of the blood between the two ends of the vessel, also sometimes called "pressure gradient" along the vessel, which is the force that pushes the blood through the vessel, and (2) the impediment to blood flow through the vessel, which is called *vascular resistance*. Figure 14-3 demonstrates these relationships, showing a blood vessel segment located anywhere in the circulatory system.

P_1 represents the pressure at the origin of the vessel; at the other end, the pressure is P_2. Resistance occurs as a result of friction between the flowing blood and the intravascular endothelium all along the inside of the vessel. The flow through the vessel can be calculated by the following formula, which is called *Ohm's law* :

$$F = \frac{\Delta P}{R}$$

in which F is blood flow, ΔP is the pressure difference ($P_1 - P_2$) between the two ends of the vessel, and R is the resistance. This formula states that the blood flow is directly proportional to the pressure difference but inversely proportional to the resistance.

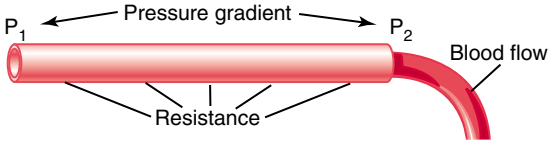


Figure 14-3 Interrelationships of pressure, resistance, and blood flow.

Note that it is the *difference* in pressure between the two ends of the vessel, not the absolute pressure in the vessel, that determines rate of flow. For example, if the pressure at both ends of a vessel is 100 mm Hg and yet no difference exists between the two ends, there will be no flow despite the presence of 100 mm Hg pressure.

Ohm's law, illustrated in Equation 1, expresses the most important of all the relations that the reader needs to understand to comprehend the hemodynamics of the circulation. Because of the extreme importance of this formula, the reader should also become familiar with its other algebraic forms:

$$\Delta P = F \times R$$

$$R = \frac{\Delta P}{F}$$

Blood Flow

Blood flow means the quantity of blood that passes a given point in the circulation in a given period of time. Ordinarily, blood flow is expressed in *milliliters per minute* or *liters per minute,* but it can be expressed in milliliters per second or in any other units of flow and time.

The overall blood flow in the total circulation of an adult person at rest is about 5000 ml/min. This is called the *cardiac output* because it is the amount of blood pumped into the aorta by the heart each minute.

Methods for Measuring Blood Flow. Many mechanical and mechanoelectrical devices can be inserted in series with a blood vessel or, in some instances, applied to the outside of the vessel to measure flow. They are called *flowmeters.*

Electromagnetic Flowmeter. One of the most important devices for measuring blood flow without opening the vessel is the electromagnetic flowmeter, the principles of which are illustrated in Figure 14-4. Figure 14-4*A* shows the generation of electromotive force (electrical voltage) in a wire that is moved rapidly in a cross-wise direction through a magnetic field. This is the well-known principle for production of electricity by the electric generator. Figure 14-4*B* shows that the same principle applies for generation of electromotive force in blood that is moving through a magnetic field. In this case, a blood vessel is placed between the poles of a strong magnet, and electrodes are placed on the two sides of the vessel perpendicular to the magnetic lines of force. When blood flows through the vessel, an electrical voltage proportional to the rate of blood flow is generated between the two electrodes, and this is recorded using an appropriate voltmeter or electronic recording apparatus. Figure 14-4*C* shows an actual "probe" that is placed on a large blood vessel to record its blood flow. The probe contains both the strong magnet and the electrodes.

A special advantage of the electromagnetic flowmeter is that it can record changes in flow in less than 1/100 of a second, allowing accurate recording of pulsatile changes in flow, as well as steady flow.

Ultrasonic Doppler Flowmeter. Another type of flowmeter that can be applied to the outside of the vessel and that has many of the same advantages as the electromagnetic flowmeter is the *ultrasonic Doppler flowmeter,* shown in Figure 14-5. A minute piezoelectric crystal is mounted at one end in the wall of the device. This crystal, when energized with an appropriate electronic apparatus, transmits ultrasound at a frequency of several hundred thousand cycles per second downstream along the flowing

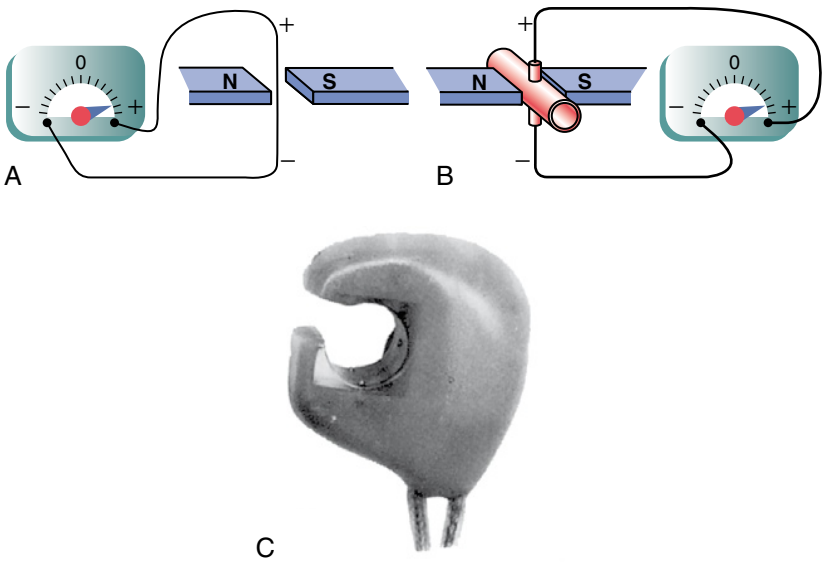


Figure 14-4 Flowmeter of the electromagnetic type, showing generation of an electrical voltage in a wire as it passes through an electromagnetic field (*A*); generation of an electrical voltage in electrodes on a blood vessel when the vessel is placed in a strong magnetic field and blood flows through the vessel (*B*); and a modern electromagnetic flowmeter probe for chronic implantation around blood vessels (*C*).

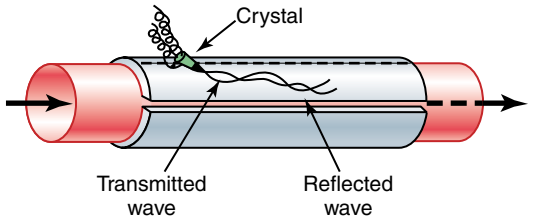


Figure 14-5 Ultrasonic Doppler flowmeter.

blood. A portion of the sound is reflected by the red blood cells in the flowing blood. The reflected ultrasound waves then travel backward from the blood cells toward the crystal. These reflected waves have a lower frequency than the transmitted wave because the red cells are moving away from the transmitter crystal. This is called the *Doppler effect.* (It is the same effect that one experiences when a train approaches and passes by while blowing its whistle. Once the whistle has passed by the person, the pitch of the sound from the whistle suddenly becomes much lower than when the train is approaching.)

For the flowmeter shown in Figure 14-5, the high-frequency ultrasound wave is intermittently cut off, and the reflected wave is received back onto the crystal and amplified greatly by the electronic apparatus. Another portion of the electronic apparatus determines the frequency difference between the transmitted wave and the reflected wave, thus determining the velocity of blood flow. As long as diameter of a blood vessel does not change, changes in blood flow in the vessel are directly related to changes in flow velocity.

Like the electromagnetic flowmeter, the ultrasonic Doppler flowmeter is capable of recording rapid, pulsatile changes in flow, as well as steady flow.

Laminar Flow of Blood in Vessels. When blood flows at a steady rate through a long, smooth blood vessel, it flows in *streamlines,* with each layer of blood remaining the same distance from the vessel wall. Also, the centralmost portion of the blood stays in the center of the vessel. This type of flow is called *laminar flow* or *streamline flow,* and it is the opposite of *turbulent flow,* which is blood flowing in all directions in the vessel and continually mixing within the vessel, as discussed subsequently.

Parabolic Velocity Profile during Laminar Flow. When laminar flow occurs, the velocity of flow in the center of the vessel is far greater than that toward the outer edges. This is demonstrated in Figure 14-6. In Figure 14-6*A*, a vessel contains two fluids, the one at the left colored by a dye and the one at the right a clear fluid, but there is no flow in the vessel. When the fluids are made to flow, a parabolic interface develops between them, as shown 1 second later in Figure 14-6*B*; the portion of fluid adjacent to the vessel wall has hardly moved, the portion slightly away from the wall has moved a small distance, and the portion in the center of the vessel has moved a long distance. This effect is called the "parabolic profile for velocity of blood flow."

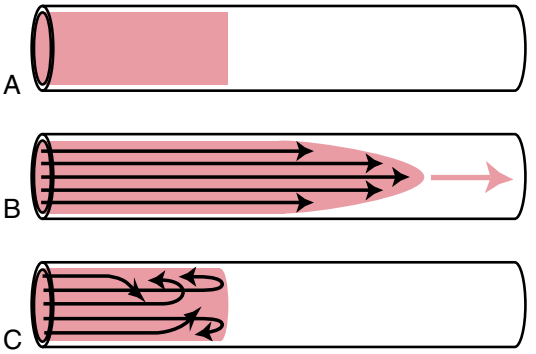


Figure 14-6 *A*, Two fluids (one dyed red, and the other clear) before flow begins; *B*, the same fluids 1 second after flow begins; *C*, turbulent flow, with elements of the fluid moving in a disorderly pattern.

The cause of the parabolic profile is the following: The fluid molecules touching the wall move slowly because of adherence to the vessel wall. The next layer of molecules slips over these, the third layer over the second, the fourth layer over the third, and so forth. Therefore, the fluid in the middle of the vessel can move rapidly because many layers of slipping molecules exist between the middle of the vessel and the vessel wall; thus, each layer toward the center flows progressively more rapidly than the outer layers.

Turbulent Flow of Blood under Some Conditions. When the rate of blood flow becomes too great, when it passes by an obstruction in a vessel, when it makes a sharp turn, or when it passes over a rough surface, the flow may then become *turbulent,* or disorderly, rather than streamlined (see Figure 14-6*C*). Turbulent flow means that the blood flows crosswise in the vessel and along the vessel, usually forming whorls in the blood, called *eddy currents.* These are similar to the whirlpools that one frequently sees in a rapidly flowing river at a point of obstruction.

When eddy currents are present, the blood flows with much greater resistance than when the flow is streamlined, because eddies add tremendously to the overall friction of flow in the vessel.

The tendency for turbulent flow increases in direct proportion to the velocity of blood flow, the diameter of the blood vessel, and the density of the blood and is inversely proportional to the viscosity of the blood, in accordance with the following equation:

$$Re = \frac{v \cdot d \cdot \rho}{\eta}$$

where Re is *Reynolds' number* and is the measure of the tendency for turbulence to occur, v is the mean velocity of blood flow (in centimeters/second), d is the vessel diameter (in centimeters), ρ is density, and η is the viscosity (in poise). The viscosity of blood is normally about ⅓₀ poise, and the density is only slightly greater than 1. When Reynolds' number rises above 200 to 400, turbulent

flow will occur at some branches of vessels but will die out along the smooth portions of the vessels. However, when Reynolds' number rises above approximately 2000, turbulence will usually occur even in a straight, smooth vessel.

Reynolds' number for flow in the vascular system even normally rises to 200 to 400 in large arteries; as a result there is almost always some turbulence of flow at the branches of these vessels. In the proximal portions of the aorta and pulmonary artery, Reynolds' number can rise to several thousand during the rapid phase of ejection by the ventricles; this causes considerable turbulence in the proximal aorta and pulmonary artery where many conditions are appropriate for turbulence: (1) high velocity of blood flow, (2) pulsatile nature of the flow, (3) sudden change in vessel diameter, and (4) large vessel diameter. However, in small vessels, Reynolds' number is almost never high enough to cause turbulence.

Blood Pressure

Standard Units of Pressure. Blood pressure almost always is measured in millimeters of mercury (mm Hg) because the mercury manometer has been used as the standard reference for measuring pressure since its invention in 1846 by Poiseuille. Actually, blood pressure means the *force exerted by the blood against any unit area of the vessel wall*. When one says that the pressure in a vessel is 50 mm Hg, this means that the force exerted is sufficient to push a column of mercury against gravity up to a level 50 millimeters high. If the pressure is 100 mm Hg, it will push the column of mercury up to 100 millimeters.

Occasionally, pressure is measured in *centimeters of water (cm H_2O)*. A pressure of 10 cm H_2O means a pressure sufficient to raise a column of water against gravity to a height of 10 centimeters. *One millimeter of mercury pressure equals 1.36 cm water pressure* because the specific gravity of mercury is 13.6 times that of water, and 1 centimeter is 10 times as great as 1 millimeter.

High-Fidelity Methods for Measuring Blood Pressure. The mercury in a manometer has so much inertia that it cannot rise and fall rapidly. For this reason, the mercury manometer, although excellent for recording steady pressures, cannot respond to pressure changes that occur more rapidly than about one cycle every 2 to 3 seconds. Whenever it is desired to record rapidly changing pressures, some other type of pressure recorder is necessary. Figure 14-7 demonstrates the basic principles of three electronic pressure *transducers* commonly used for converting blood pressure and/or rapid changes in pressure into electrical signals and then recording the electrical signals on a high-speed electrical recorder. Each of these transducers uses a very thin, highly stretched metal membrane that forms one wall of the fluid chamber. The fluid chamber in turn is connected through a needle or catheter inserted into the blood vessel in which the pressure is to be measured. When the pressure is high, the membrane bulges slightly, and when it is low, it returns toward its resting position.

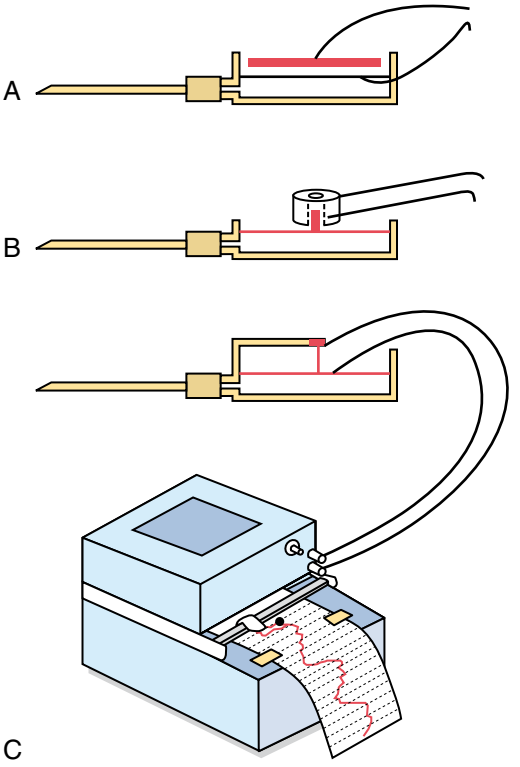


Figure 14-7 Principles of three types of electronic transducers for recording rapidly changing blood pressures (explained in the text).

In Figure 14-7*A*, a simple metal plate is placed a few hundredths of a centimeter above the membrane. When the membrane bulges, the membrane comes closer to the plate, which increases the *electrical capacitance* between these two, and this change in capacitance can be recorded using an appropriate electronic system.

In Figure 14-7*B*, a small iron slug rests on the membrane, and this can be displaced upward into a center space inside an electrical wire coil. Movement of the iron into the coil increases the *inductance* of the coil, and this, too, can be recorded electronically.

Finally, in Figure 14-7*C*, a very thin, stretched resistance wire is connected to the membrane. When this wire is stretched greatly, its resistance increases; when it is stretched less, its resistance decreases. These changes, too, can be recorded by an electronic system.

The electrical signals from the transducer are sent to an amplifier and then to an appropriate recording device. With some of these high-fidelity types of recording systems, pressure cycles up to 500 cycles per second have been recorded accurately. In common use are recorders capable of registering pressure changes that occur as rapidly as 20 to 100 cycles per second, in the manner shown on the recording paper in Figure 14-7*C*.

Resistance to Blood Flow

Units of Resistance. Resistance is the impediment to blood flow in a vessel, but it cannot be measured by any direct means. Instead, resistance must be calculated from measurements of blood flow and pressure

difference between two points in the vessel. If the pressure difference between two points is 1 mm Hg and the flow is 1 ml/sec, the resistance is said to be 1 *peripheral resistance unit,* usually abbreviated *PRU*.

Expression of Resistance in CGS Units. Occasionally, a basic physical unit called the CGS (centimeters, grams, seconds) unit is used to express resistance. This unit is dyne sec/cm^5. Resistance in these units can be calculated by the following formula:

$$R\left(\text{in}\ \frac{\text{dyne sec}}{\text{cm}^5}\right) = \frac{1333 \times \text{mm Hg}}{\text{ml/sec}}$$

Total Peripheral Vascular Resistance and Total Pulmonary Vascular Resistance. The rate of blood flow through the entire circulatory system is equal to the rate of blood pumping by the heart—that is, it is equal to the cardiac output. In the adult human being, this is approximately 100 ml/sec. The pressure difference from the systemic arteries to the systemic veins is about 100 mm Hg. Therefore, the resistance of the entire systemic circulation, called the *total peripheral resistance,* is about 100/100, or 1 peripheral resistance unit (PRU).

In conditions in which all the blood vessels throughout the body become strongly constricted, the total peripheral resistance occasionally rises to as high as 4 PRU. Conversely, when the vessels become greatly dilated, the resistance can fall to as little as 0.2 PRU.

In the pulmonary system, the mean pulmonary arterial pressure averages 16 mm Hg and the mean left atrial pressure averages 2 mm Hg, giving a net pressure difference of 14 mm. Therefore, when the cardiac output is normal at about 100 ml/sec, the *total pulmonary vascular resistance* calculates to be about 0.14 PRU (about one seventh that in the systemic circulation).

"Conductance" of Blood in a Vessel and Its Relation to Resistance. Conductance is a measure of the blood flow through a vessel for a given pressure difference. This is generally expressed in terms of milliliters per second per millimeter of mercury pressure, but it can also be expressed in terms of liters per second per millimeter of mercury or in any other units of blood flow and pressure.

It is evident that conductance is the exact reciprocal of resistance in accord with the following equation:

$$\text{Conductance} = \frac{1}{\text{Resistance}}$$

Very Slight Changes in Diameter of a Vessel Can Change Its Conductance Tremendously! Slight changes in the diameter of a vessel cause tremendous changes in the vessel's ability to conduct blood when the blood flow is streamlined. This is demonstrated by the experiment illustrated in Figure 14-8*A*, which shows three vessels with relative diameters of 1, 2, and 4 but with the same pressure difference of 100 mm Hg between the two ends of the vessels. Although the diameters of these vessels increase only fourfold, the respective flows are 1, 16, and 256 ml/min, which is a 256-fold increase in flow. Thus, the conductance of the vessel increases in proportion to the *fourth power of the diameter,* in accordance with the following formula:

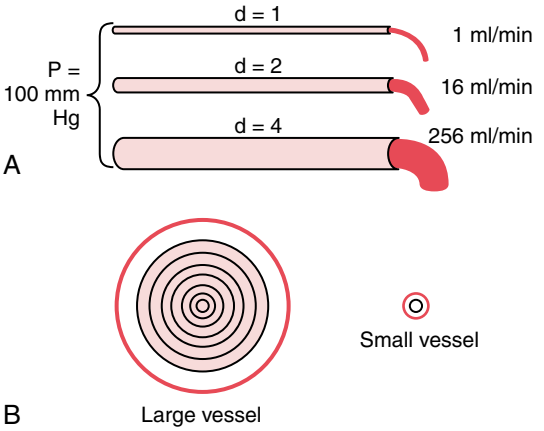


Figure 14-8 *A*, Demonstration of the effect of vessel diameter on blood flow. *B*, Concentric rings of blood flowing at different velocities; the farther away from the vessel wall, the faster the flow.

$$\text{Conductance} \propto \text{Diameter}^4$$

Poiseuille's Law. The cause of this great increase in conductance when the diameter increases can be explained by referring to Figure 14-8*B*, which shows cross sections of a large and a small vessel. The concentric rings inside the vessels indicate that the velocity of flow in each ring is different from that in the adjacent rings because of *laminar* flow, which was discussed earlier in the chapter. That is, the blood in the ring touching the wall of the vessel is barely flowing because of its adherence to the vascular endothelium. The next ring of blood toward the center of the vessel slips past the first ring and, therefore, flows more rapidly. The third, fourth, fifth, and sixth rings likewise flow at progressively increasing velocities. Thus, the blood that is near the wall of the vessel flows slowly, whereas that in the middle of the vessel flows much more rapidly.

In the small vessel, essentially all the blood is near the wall, so the extremely rapidly flowing central stream of blood simply does not exist. By integrating the velocities of all the concentric rings of flowing blood and multiplying them by the areas of the rings, one can derive the following formula, known as Poiseuille's law:

$$F = \frac{\pi \Delta P r^4}{8 \eta l}$$

in which F is the rate of blood flow, ΔP is the pressure difference between the ends of the vessel, r is the radius of the vessel, l is length of the vessel, and η is viscosity of the blood.

Note particularly in this equation that the rate of blood flow is directly proportional to the *fourth power of the radius* of the vessel, which demonstrates once again that the diameter of a blood vessel (which is equal to twice the radius) plays by far the greatest role of all factors in determining the rate of blood flow through a vessel.

Importance of the Vessel Diameter "Fourth Power Law" in Determining Arteriolar Resistance. In the systemic circulation, about two thirds of the total systemic resistance to blood flow is arteriolar resistance in the small arterioles. The internal diameters of the arterioles range from as little as 4 micrometers to as great as 25 micrometers. However, their strong vascular walls allow the internal diameters to change tremendously, often as much as fourfold. From the fourth power law discussed earlier that relates blood flow to diameter of the vessel, one can see that a fourfold increase in vessel diameter can increase the flow as much as 256-fold. Thus, this fourth power law makes it possible for the arterioles, responding with only small changes in diameter to nervous signals or local tissue chemical signals, either to turn off almost completely the blood flow to the tissue or at the other extreme to cause a vast increase in flow. Indeed, ranges of blood flow of more than 100-fold in separate tissue areas have been recorded between the limits of maximum arteriolar constriction and maximum arteriolar dilatation.

Resistance to Blood Flow in Series and Parallel Vascular Circuits. Blood pumped by the heart flows from the high-pressure part of the systemic circulation (i.e., aorta) to the low-pressure side (i.e., vena cava) through many miles of blood vessels arranged in series and in parallel. The arteries, arterioles, capillaries, venules, and veins are collectively arranged in series. When blood vessels are arranged in series, flow through each blood vessel is the same and the total resistance to blood flow (R_{total}) is equal to the sum of the resistances of each vessel:

$$R_{total} = R_1 + R_2 + R_3 + R_4 \ldots$$

The total peripheral vascular resistance is therefore equal to the sum of resistances of the arteries, arterioles, capillaries, venules, and veins. In the example shown in Figure 14-9*A*, the total vascular resistance is equal to the sum of R_1 and R_2.

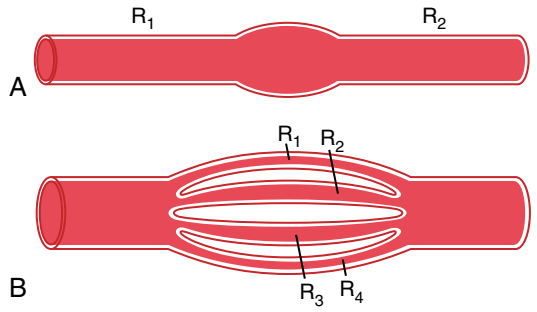


Figure 14-9 Vascular resistances: *A*, in series and *B*, in parallel.

Blood vessels branch extensively to form parallel circuits that supply blood to the many organs and tissues of the body. This parallel arrangement permits each tissue to regulate its own blood flow, to a great extent, independently of flow to other tissues.

For blood vessels arranged in parallel (Figure 14-9*B*), the total resistance to blood flow is expressed as:

$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \frac{1}{R_4} \ldots$$

It is obvious that for a given pressure gradient, far greater amounts of blood will flow through this parallel system than through any of the individual blood vessels. Therefore, the total resistance is far less than the resistance of any single blood vessel. Flow through each of the parallel vessels in Figure 14-9*B* is determined by the pressure gradient and its own resistance, not the resistance of the other parallel blood vessels. However, increasing the resistance of any of the blood vessels increases the total vascular resistance.

It may seem paradoxical that adding more blood vessels to a circuit reduces the total vascular resistance. Many parallel blood vessels, however, make it easier for blood to flow through the circuit because each parallel vessel provides another pathway, or *conductance,* for blood flow. The total conductance (C_{total}) for blood flow is the sum of the conductance of each parallel pathway:

$$C_{total} = C_1 + C_2 + C_3 + C_4 \ldots$$

For example, brain, kidney, muscle, gastrointestinal, skin, and coronary circulations are arranged in parallel, and each tissue contributes to the overall conductance of the systemic circulation. Blood flow through each tissue is a fraction of the total blood flow (cardiac output) and is determined by the resistance (the reciprocal of conductance) for blood flow in the tissue, as well as the pressure gradient. Therefore, amputation of a limb or surgical removal of a kidney also removes a parallel circuit and reduces the total vascular conductance and total blood flow (i.e., cardiac output) while increasing total peripheral vascular resistance.

Effect of Blood Hematocrit and Blood Viscosity on Vascular Resistance and Blood Flow

Note especially that another of the important factors in Poiseuille's equation is the viscosity of the blood. The greater the viscosity, the less the flow in a vessel if all other factors are constant. Furthermore, *the viscosity of normal blood is about three times as great as the viscosity of water.*

But what makes the blood so viscous? It is mainly the large numbers of suspended red cells in the blood, each of which exerts frictional drag against adjacent cells and against the wall of the blood vessel.

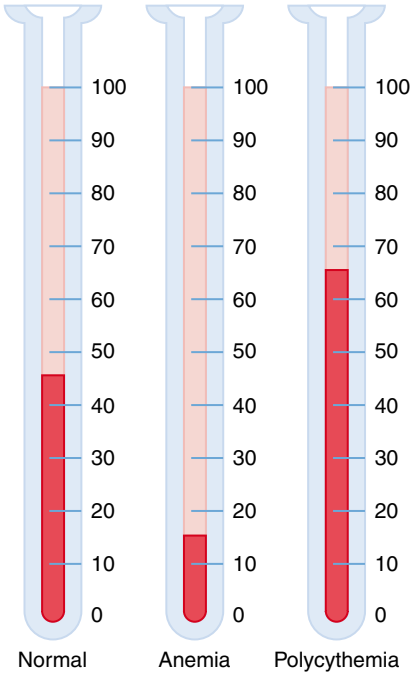


Figure 14-10 Hematocrits in a healthy (normal) person and in patients with anemia and polycythemia.

Hematocrit. The proportion of the blood that is red blood cells is called the *hematocrit.* Thus, if a person has a hematocrit of 40, this means that 40 percent of the blood volume is cells and the remainder is plasma. The hematocrit of adult men averages about 42, while that of women averages about 38. These values vary tremendously, depending on whether the person has anemia, on the degree of bodily activity, and on the altitude at which the person resides. These changes in hematocrit are discussed in relation to the red blood cells and their oxygen transport function in Chapter 32.

Hematocrit is determined by centrifuging blood in a calibrated tube, as shown in Figure 14-10. The calibration allows direct reading of the percentage of cells.

Effect of Hematocrit on Blood Viscosity. The viscosity of blood increases drastically as the hematocrit increases, as shown in Figure 14-11. The viscosity of whole blood at normal hematocrit is about 3; this means that three times as much pressure is required to force whole blood as to force water through the same blood vessel. When the hematocrit rises to 60 or 70, which it often does in *polycythemia,* the blood viscosity can become as great as 10 times that of water, and its flow through blood vessels is greatly retarded.

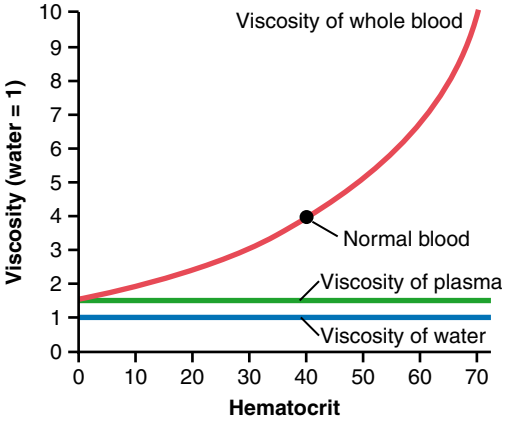


Figure 14-11 Effect of hematocrit on blood viscosity. (Water viscosity = 1.)

Other factors that affect blood viscosity are the plasma protein concentration and types of proteins in the plasma, but these effects are so much less than the effect of hematocrit that they are not significant considerations in most hemodynamic studies. The viscosity of blood plasma is about 1.5 times that of water.

Effects of Pressure on Vascular Resistance and Tissue Blood Flow

"Autoregulation" Attenuates the Effect of Arterial Pressure on Tissue Blood Flow. From the discussion thus far, one might expect an increase in arterial pressure to cause a proportionate increase in blood flow through the various tissues of the body. However, the effect of arterial pressure on blood flow in many tissues is usually far less than one would expect, as shown in Figure 14-12. The reason for this is that an increase in arterial pressure not only increases the force that pushes blood through the vessels but it also initiates compensatory increases in vascular resistance within a few seconds through activation of the local control mechanisms discussed in Chapter 17. Conversely, with reductions in arterial pressure most vascular resistance is promptly reduced in most tissues and blood flow is maintained relatively constant. The ability of each tissue to adjust its vascular resistance and to maintain normal blood flow during changes in arterial pressure between approximately 70 and 175 mm Hg is called *blood flow autoregulation.*

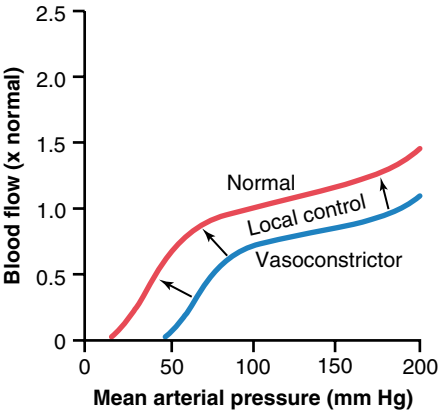


Figure 14-12 Effect of changes in arterial pressure over a period of several minutes on blood flow in a tissue such as skeletal muscle. Note that between pressure of 70 and 175 mm Hg blood flow is "autoregulated." The ***blue line*** shows the effect of sympathetic nerve stimulation or vasoconstriction by hormones such as norepinephrine, angiotensin II, vasopressin, or endothelin on this relationship. Reduced tissue blood flow is rarely maintained for more than a few hours due to activation of local autoregulatory mechanisms that eventually return blood flow toward normal.

Note in Figure 14-12 that changes in blood flow can be caused by strong sympathetic stimulation, which *constricts* the blood vessels. Likewise, hormonal vasoconstrictors, such as *norepinephrine, angiotensin II, vasopressin,* or *endothelin,* can also reduce blood flow, at least transiently.

Changes in tissue blood flow rarely last for more than a few hours even when increases in arterial pressure or increased levels of vasoconstrictors are sustained. The reason for the relative constancy of blood flow is that each tissue's local autoregulatory mechanisms eventually override most of the effects of vasoconstrictors in order to provide a blood flow that is appropriate for the needs of the tissue.

Pressure-Flow Relationship in Passive Vascular Beds. In isolated blood vessels or in tissues that do not exhibit autoregulation, changes in arterial pressure may have important effects on blood flow. In fact, the effect of pressure on blood flow may be greater than predicted by Poiseuille's equation, as shown by the upward curving lines in Figure 14-13. The reason for this is that increased arterial pressure not only increases the force that pushes blood through the vessels but it also distends the elastic vessels, actually *decreasing* vascular resistance. Conversely, decreased arterial pressure in passive blood vessels increases resistance as the elastic vessels gradually collapse due to reduced distending pressure. When pressure falls below a critical level, called the *critical closing pressure,* flow ceases as the blood vessels are completely collapsed.

Sympathetic stimulation and other vasoconstrictors can alter the passive pressure-flow relationship shown in Figure 14-13. Thus, *inhibition of* sympathetic activity

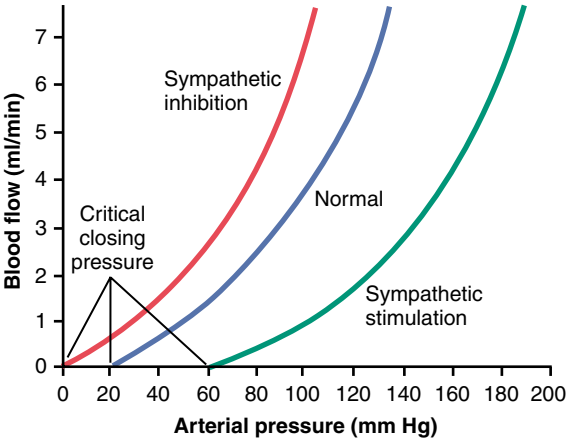


Figure 14-13 Effect of arterial pressure on blood flow through a *passive* blood vessel at different degrees of vascular tone caused by increased or decreased sympathetic stimulation of the vessel.

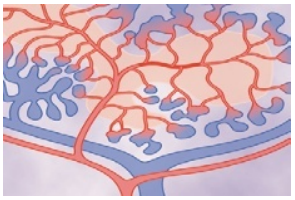
greatly dilates the vessels and can increase the blood flow twofold or more. Conversely, very strong sympathetic stimulation *can constrict* the vessels so much that blood flow occasionally decreases to as low as zero for a few seconds despite high arterial pressure.

In reality, there are few physiological conditions in which tissues display the passive pressure-flow relationship shown in Figure 14-13. Even in tissues that do not effectively autoregulate blood flow during acute changes in arterial pressure, blood flow is regulated according to the needs of the tissue when the pressure changes are sustained, as discussed in Chapter 17.

Bibliography

See bibliography for Chapter 15.

Vascular Distensibility and Functions of the Arterial and Venous Systems



Vascular Distensibility

A valuable characteristic of the vascular system is that all blood vessels are *distensible.* The distensible nature of the arteries allows them to accommodate the pulsatile output of the heart and to average out the pressure pulsations. This provides smooth, continuous flow of blood through the very small blood vessels of the tissues.

The most distensible by far of all the vessels are the veins. Even slight increases in venous pressure cause the veins to store 0.5 to 1.0 liter of extra blood. Therefore, the veins provide a *reservoir function* for storing large quantities of extra blood that can be called into use whenever required elsewhere in the circulation.

Units of Vascular Distensibility. Vascular distensibility normally is expressed as the fractional increase in volume for each millimeter of mercury rise in pressure, in accordance with the following formula:

$$\text{Vascular distensibility} = \frac{\text{Increase in volume}}{\text{Increase in pressure} \times \text{Original volume}}$$

That is, if 1 mm Hg causes a vessel that originally contained 10 millimeters of blood to increase its volume by 1 milliliter, the distensibility would be 0.1 per mm Hg, or 10 percent per mm Hg.

Difference in Distensibility of the Arteries and the Veins. Anatomically, the walls of the arteries are far stronger than those of the veins. Consequently, the veins, on average, are about eight times more distensible than the arteries. That is, a given increase in pressure causes about eight times as much increase in blood in a vein as in an artery of comparable size.

In the pulmonary circulation, the pulmonary vein distensibilities are similar to those of the systemic circulation. But the pulmonary arteries normally operate under pressures about one sixth of those in the systemic arterial system, and their distensibilities are correspondingly greater, about six times the distensibility of systemic arteries.

Vascular Compliance (or Vascular Capacitance)

In hemodynamic studies, it usually is much more important to know the *total quantity of blood* that can be stored in a given portion of the circulation for each mm Hg pressure rise than to know the distensibilities of the individual vessels. This value is called the *compliance* or *capacitance* of the respective vascular bed; that is,

$$\text{Vascular compliance} = \frac{\text{Increase in volume}}{\text{Increase in pressure}}$$

Compliance and distensibility are quite different. A highly distensible vessel that has a slight volume may have far less compliance than a much less distensible vessel that has a large volume because *compliance is equal to distensibility times volume.*

The compliance of a systemic vein is about 24 times that of its corresponding artery because it is about 8 times as distensible and it has a volume about 3 times as great ($8 \times 3 = 24$).

Volume-Pressure Curves of the Arterial and Venous Circulations

A convenient method for expressing the relation of pressure to volume in a vessel or in any portion of the circulation is to use the so-called *volume-pressure curve.* The red and blue solid curves in Figure 15-1 represent, respectively, the volume-pressure curves of the normal systemic arterial system and venous system, showing that when the arterial system of the average adult person (including all the large arteries, small arteries, and arterioles) is filled with about 700 milliliters of blood, the mean arterial pressure is 100 mm Hg, but when it is filled with only 400 milliliters of blood, the pressure falls to zero.

In the entire systemic venous system, the volume normally ranges from 2000 to 3500 milliliters, and a change of several hundred millimeters in this volume is required to change the venous pressure only 3 to 5 mm Hg. This mainly explains why as much as one half liter of blood can be

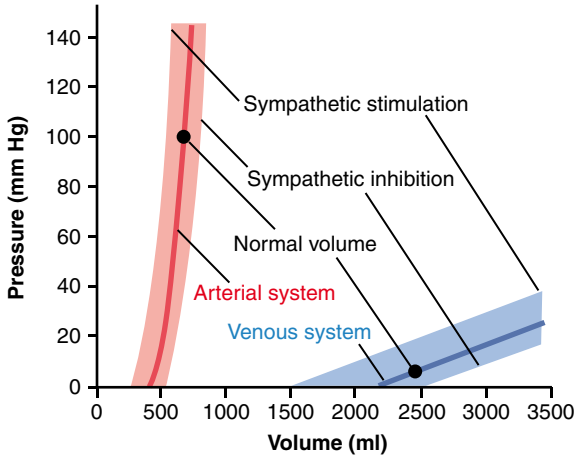


Figure 15-1 "Volume-pressure curves" of the systemic arterial and venous systems, showing the effects of stimulation or inhibition of the sympathetic nerves to the circulatory system.

transfused into a healthy person in only a few minutes without greatly altering function of the circulation.

Effect of Sympathetic Stimulation or Sympathetic Inhibition on the Volume-Pressure Relations of the Arterial and Venous Systems. Also shown in Figure 15-1 are the effects of exciting or inhibiting the vascular sympathetic nerves on the volume-pressure curves. It is evident that increase in vascular smooth muscle tone caused by sympathetic stimulation increases the pressure at each volume of the arteries or veins, whereas sympathetic inhibition decreases the pressure at each volume. Control of the vessels in this manner by the sympathetics is a valuable means for diminishing the dimensions of one segment of the circulation, thus transferring blood to other segments. For instance, an increase in vascular tone throughout the systemic circulation often causes large volumes of blood to shift into the heart, which is one of the principal methods that the body uses to increase heart pumping.

Sympathetic control of vascular capacitance is also highly important during hemorrhage. Enhancement of sympathetic tone, especially to the veins, reduces the vessel sizes enough that the circulation continues to operate almost normally even when as much as 25 percent of the total blood volume has been lost.

Delayed Compliance (Stress-Relaxation) of Vessels

The term "delayed compliance" means that a vessel exposed to increased volume at first exhibits a large increase in pressure, but progressive delayed stretching of smooth muscle in the vessel wall allows the pressure to return back toward normal over a period of minutes to hours. This effect is shown in Figure 15-2. In this figure, the pressure is recorded in a small segment of a vein that is occluded at both ends. An extra volume of blood is suddenly injected until the pressure rises from 5 to 12 mm Hg. Even though none of the blood is removed after it is injected, the pressure begins to decrease immediately and approaches about 9 mm Hg after several minutes. In other words, the volume of blood injected causes immediate

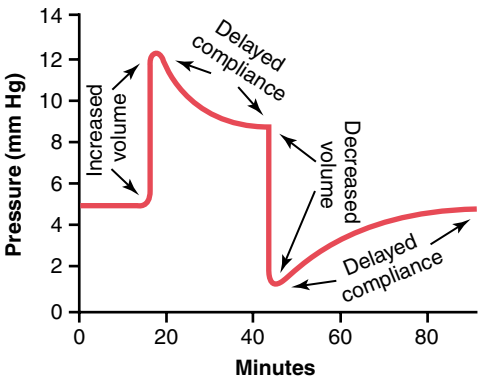


Figure 15-2 Effect on the intravascular pressure of injecting a volume of blood into a venous segment and later removing the excess blood, demonstrating the principle of delayed compliance.

elastic distention of the vein, but then the smooth muscle fibers of the vein begin to "creep" to longer lengths, and their tensions correspondingly decrease. This effect is a characteristic of all smooth muscle tissue and is called *stress-relaxation,* which was explained in Chapter 8.

Delayed compliance is a valuable mechanism by which the circulation can accommodate extra blood when necessary, such as after too large a transfusion. Delayed compliance in the reverse direction is one of the ways in which the circulation automatically adjusts itself over a period of minutes or hours to diminished blood volume after serious hemorrhage.

Arterial Pressure Pulsations

With each beat of the heart a new surge of blood fills the arteries. Were it not for distensibility of the arterial system, all of this new blood would have to flow through the peripheral blood vessels almost instantaneously, only during cardiac systole, and no flow would occur during diastole. However, the compliance of the arterial tree normally reduces the pressure pulsations to almost no pulsations by the time the blood reaches the capillaries; therefore, tissue blood flow is mainly continuous with very little pulsation.

A typical record of the *pressure pulsations* at the root of the aorta is shown in Figure 15-3. In the healthy young adult, the pressure at the top of each pulse, called the *systolic pressure,* is about 120 mm Hg. At the lowest point of each pulse, called the *diastolic pressure,* it is about 80 mm Hg. The difference between these two pressures, about 40 mm Hg, is called the *pulse pressure.*

Two major factors affect the pulse pressure: (1) the *stroke volume output* of the heart and (2) the *compliance (total distensibility)* of the arterial tree. A third, less important factor, is the character of ejection from the heart during systole.

In general, the greater the stroke volume output, the greater the amount of blood that must be accommodated in the arterial tree with each heartbeat, and, therefore, the greater the pressure rise and fall during systole and diastole, thus causing a greater pulse pressure. Conversely, the less the compliance of the arterial system, the greater the

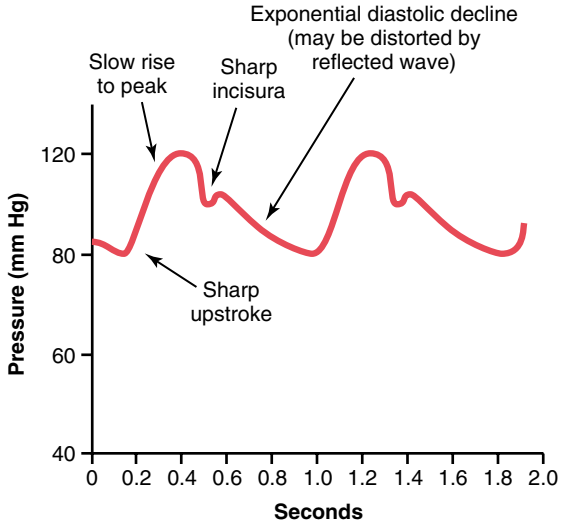


Figure 15-3 Pressure pulse contour in the ascending aorta.

rise in pressure for a given stroke volume of blood pumped into the arteries. For instance, as demonstrated by the middle top curves in Figure 15-4, the pulse pressure in old age sometimes rises to as much as twice normal, because the arteries have become hardened with *arteriosclerosis* and therefore are relatively noncompliant.

In effect, pulse pressure is determined approximately by the *ratio of stroke volume output to compliance of the arterial tree.* Any condition of the circulation that affects either of these two factors also affects the pulse pressure:

Pulse Pressure ≈ stroke volume/arterial compliance

Abnormal Pressure Pulse Contours

Some conditions of the circulation also cause *abnormal contours of the pressure pulse wave* in addition to altering the pulse pressure. Especially distinctive among these are aortic stenosis, patent ductus arteriosus, and aortic regurgitation, each of which is shown in Figure 15-4.

In *aortic valve stenosis,* the diameter of the aortic valve opening is reduced significantly, and the aortic pressure pulse is decreased significantly because of diminished blood flow outward through the stenotic valve.

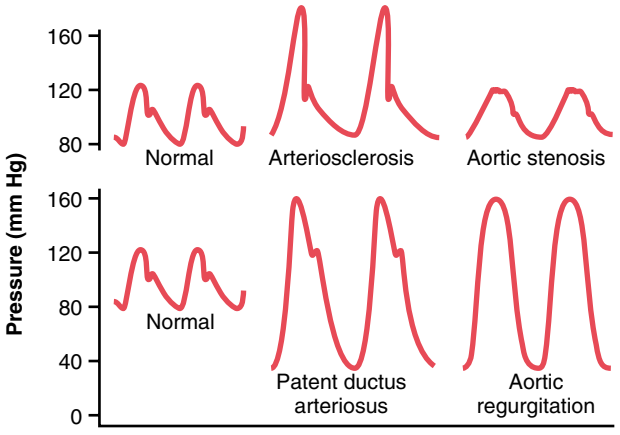


Figure 15-4 Aortic pressure pulse contours in arteriosclerosis, aortic stenosis, patent ductus arteriosus, and aortic regurgitation.

In *patent ductus arteriosus,* one half or more of the blood pumped into the aorta by the left ventricle flows immediately backward through the wide-open ductus into the pulmonary artery and lung blood vessels, thus allowing the diastolic pressure to fall very low before the next heartbeat.

In *aortic regurgitation,* the aortic valve is absent or will not close completely. Therefore, after each heartbeat, the blood that has just been pumped into the aorta flows immediately backward into the left ventricle. As a result, the aortic pressure can fall all the way to zero between heartbeats. Also, there is no incisura in the aortic pulse contour because there is no aortic valve to close.

Transmission of Pressure Pulses to the Peripheral Arteries

When the heart ejects blood into the aorta during systole, at first only the proximal portion of the aorta becomes distended because the inertia of the blood prevents sudden blood movement all the way to the periphery. However, the rising pressure in the proximal aorta rapidly overcomes this inertia, and the wave front of distention spreads farther and farther along the aorta, as shown in Figure 15-5. This is called *transmission of the pressure pulse* in the arteries.

The velocity of pressure pulse transmission in the normal aorta is 3 to 5 m/sec; in the large arterial branches, 7 to 10 m/sec; and in the small arteries, 15 to 35 m/sec. In general, the greater the compliance of each vascular segment, the slower the velocity, which explains the slow transmission in the aorta and the much faster transmission in the much less compliant small distal arteries. In the aorta, the velocity of transmission of the pressure pulse is 15 or more times the velocity of blood flow because the

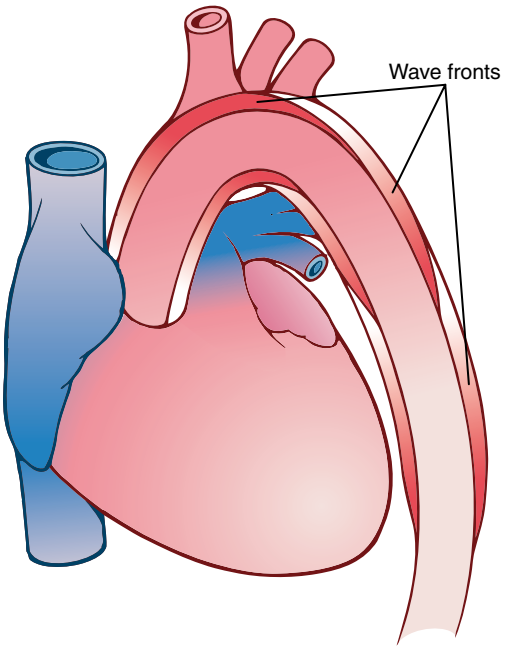


Figure 15-5 Progressive stages in transmission of the pressure pulse along the aorta.

pressure pulse is simply a moving wave of *pressure* that involves little forward total movement of blood volume.

Damping of the Pressure Pulses in the Smaller Arteries, Arterioles, and Capillaries. Figure 15-6 shows typical changes in the contours of the pressure pulse as the pulse travels into the peripheral vessels. Note especially in the three lower curves that the intensity of pulsation becomes progressively less in the smaller arteries, the arterioles, and, especially, the capillaries. In fact, only when the aortic pulsations are extremely large or the arterioles are greatly dilated can pulsations be observed in the capillaries.

This progressive diminution of the pulsations in the periphery is called *damping* of the pressure pulses. The cause of this is twofold: (1) resistance to blood movement in the vessels and (2) compliance of the vessels. The resistance damps the pulsations because a small amount of blood must flow forward at the pulse wave front to distend the next segment of the vessel; the greater the resistance, the more difficult it is for this to occur. The compliance damps the pulsations because the more compliant a vessel, the greater the quantity of blood required at the pulse wave front to cause an increase in pressure. Therefore, *the degree of damping is almost directly proportional to the product of resistance times compliance.*

Clinical Methods for Measuring Systolic and Diastolic Pressures

It is not reasonable to use pressure recorders that require needle insertion into an artery for making routine arterial pressure measurements in human patients, although these are used on occasion when special studies are necessary. Instead, the clinician determines systolic and diastolic pressures by indirect means, usually by the *auscultatory method*.

Auscultatory Method. Figure 15-7 shows the auscultatory method for determining systolic and diastolic arterial pressures. A stethoscope is placed over the antecubital artery and a blood pressure cuff is inflated around the upper arm. As long as the cuff continues to compress the arm with too little pressure to close the brachial artery, no sounds are heard from the antecubital artery with the stethoscope. However, when the cuff pressure is great enough to close the artery during part of the arterial pressure cycle, a sound then is heard with each pulsation. These sounds are called *Korotkoff sounds,*

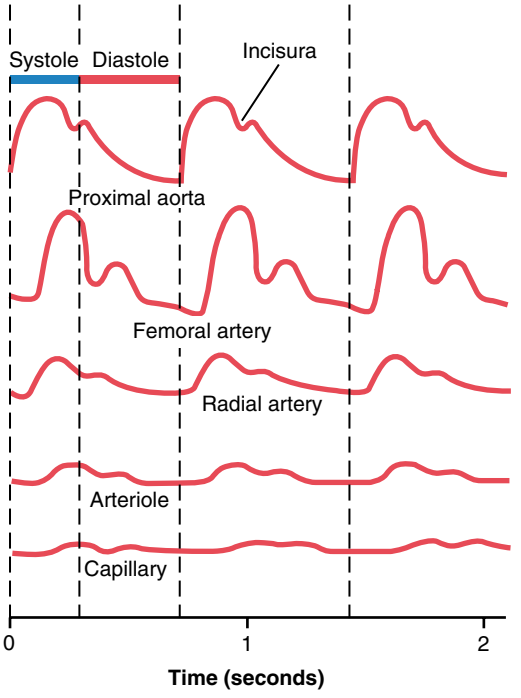


Figure 15-6 Changes in the pulse pressure contour as the pulse wave travels toward the smaller vessels.

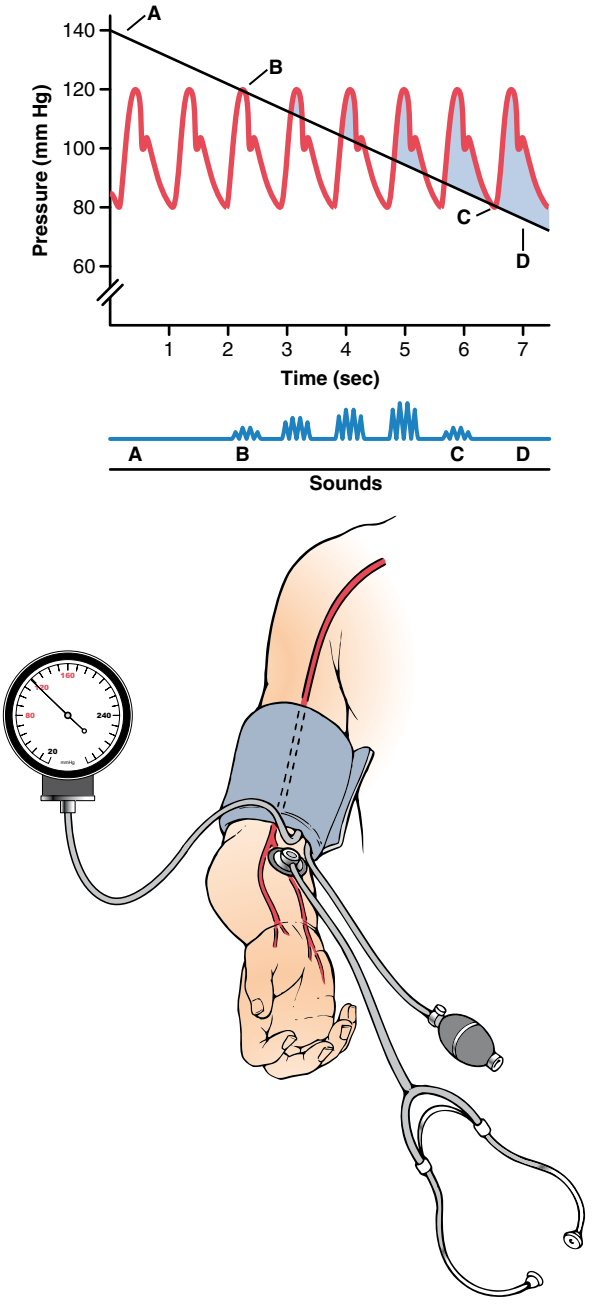


Figure 15-7 Auscultatory method for measuring systolic and diastolic arterial pressures.

named after *Nikolai Korotkoff,* a Russian physician who described them in 1905.

The Korotkoff sounds are believed to be caused mainly by blood jetting through the partly occluded vessel and by vibrations of the vessel wall. The jet causes turbulence in the vessel beyond the cuff, and this sets up the vibrations heard through the stethoscope.

In determining blood pressure by the auscultatory method, the pressure in the cuff is first elevated well above arterial systolic pressure. As long as this cuff pressure is higher than systolic pressure, the brachial artery remains collapsed so that no blood jets into the lower artery during any part of the pressure cycle. Therefore, no Korotkoff sounds are heard in the lower artery. But then the cuff pressure gradually is reduced. Just as soon as the pressure in the cuff falls below systolic pressure (point B, Figure 15-7), blood begins to slip through the artery beneath the cuff during the peak of systolic pressure, and one begins to hear *tapping* sounds from the antecubital artery in synchrony with the heartbeat. As soon as these sounds begin to be heard, the pressure level indicated by the manometer connected to the cuff is about equal to the systolic pressure.

As the pressure in the cuff is lowered still more, the Korotkoff sounds change in quality, having less of the tapping quality and more of a rhythmical and harsher quality. Then, finally, when the pressure in the cuff falls near diastolic pressure, the sounds suddenly change to a muffled quality (point C, Figure 15-7). One notes the manometer pressure when the Korotkoff sounds change to the muffled quality and this pressure is about equal to the diastolic pressure, although it slightly overestimates the diastolic pressure determined by direct intra-arterial catheter. As the cuff pressure falls a few mm Hg further, the artery no longer closes during diastole, which means that the basic factor causing the sounds (the jetting of blood through a squeezed artery) is no longer present. Therefore, the sounds disappear entirely. Many clinicians believe that the pressure at which the Korotkoff sounds completely disappear should be used as the diastolic pressure, except in situations in which the disappearance of sounds cannot reliably be determined because sounds are audible even after complete deflation of the cuff. For example, in patients with arteriovenous fistulas for hemodialysis or with aortic insufficiency, Korotkoff sounds may be heard after complete deflation of the cuff.

The auscultatory method for determining systolic and diastolic pressures is not entirely accurate, but it usually gives values within 10 percent of those determined by direct catheter measurement from inside the arteries.

Normal Arterial Pressures as Measured by the Auscultatory Method. Figure 15-8 shows the approximate normal systolic and diastolic arterial pressures at different ages. The progressive increase in pressure with age results from the effects of aging on the blood pressure control mechanisms. We shall see in Chapter 19 that the kidneys are primarily responsible for this long-term regulation of arterial pressure; and it is well known that the kidneys exhibit definitive changes with age, especially after the age of 50 years.

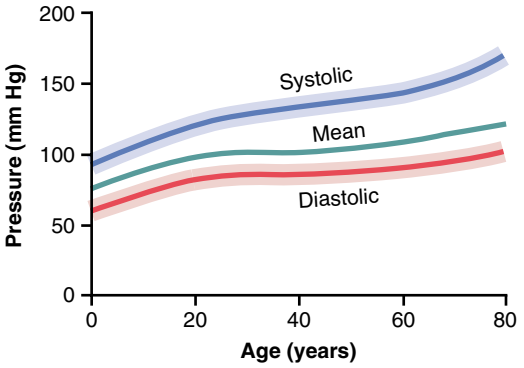


Figure 15-8 Changes in systolic, diastolic, and mean arterial pressures with age. The shaded areas show the approximate normal ranges.

A slight extra increase in *systolic* pressure usually occurs beyond the age of 60 years. This results from decreasing distensibility, or "hardening," of the arteries, which is often a result of *atherosclerosis.* The final effect is a higher systolic pressure with considerable increase in pulse pressure, as previously explained.

Mean Arterial Pressure. The mean arterial pressure is the average of the arterial pressures measured millisecond by millisecond over a period of time. It is not equal to the average of systolic and diastolic pressure because at normal heart rates, a greater fraction of the cardiac cycle is spent in diastole than is systole; thus, the arterial pressure remains nearer to diastolic pressure than to systolic pressure during the greater part of the cardiac cycle. The mean arterial pressure is therefore determined about 60 percent by the diastolic pressure and 40 percent by the systolic pressure. Note in Figure 15-8 that the mean pressure (solid green line) at all ages is nearer to the diastolic pressure than to the systolic pressure. However, at very high heart rates diastole comprises a smaller fraction of the cardiac cycle and the mean arterial pressure is more closely approximated as the average of systolic and diastolic pressures.

Veins and Their Functions

For years, the veins were considered to be nothing more than passageways for flow of blood to the heart, but it is now apparent that they perform other special functions that are necessary for operation of the circulation. Especially important, they are capable of constricting and enlarging and thereby storing either small or large quantities of blood and making this blood available when it is required by the remainder of the circulation. The peripheral veins can also propel blood forward by means of a so-called *venous pump,* and they even help to regulate cardiac output, an exceedingly important function that is described in detail in Chapter 20.

Venous Pressures—Right Atrial Pressure (Central Venous Pressure) and Peripheral Venous Pressures

To understand the various functions of the veins, it is first necessary to know something about pressure in the veins and what determines the pressure.

Blood from all the systemic veins flows into the right atrium of the heart; therefore, the pressure in the right atrium is called the *central venous pressure.*

Right atrial pressure is regulated by a balance between (1) the ability of the heart to pump blood out of the right atrium and ventricle into the lungs and *(2) the tendency for blood to flow from the peripheral veins into the right atrium.* If the right heart is pumping strongly, the right atrial pressure decreases. Conversely, weakness of the heart elevates the right atrial pressure. Also, any effect that causes rapid inflow of blood into the right atrium from the peripheral veins elevates the right atrial pressure. Some of the factors that can increase this venous return (and thereby increase the right atrial pressure) are (1) increased blood volume, (2) increased large vessel tone throughout the body with resultant increased peripheral venous pressures, and (3) dilatation of the arterioles, which decreases the peripheral resistance and allows rapid flow of blood from the arteries into the veins.

The same factors that regulate right atrial pressure also contribute to regulation of cardiac output because the amount of blood pumped by the heart depends on both the ability of the heart to pump and the tendency for blood to flow into the heart from the peripheral vessels. Therefore, we will discuss regulation of right atrial pressure in much more depth in Chapter 20 in connection with regulation of cardiac output.

The *normal right atrial pressure* is about 0 mm Hg, which is equal to the atmospheric pressure around the body. It can increase to 20 to 30 mm Hg under very abnormal conditions, such as (1) serious heart failure or (2) after massive transfusion of blood, which greatly increases the total blood volume and causes excessive quantities of blood to attempt to flow into the heart from the peripheral vessels.

The lower limit to the right atrial pressure is usually about −3 to −5 mm Hg below atmospheric pressure. This is also the pressure in the chest cavity that surrounds the heart. The right atrial pressure approaches these low values when the heart pumps with exceptional vigor or when blood flow into the heart from the peripheral vessels is greatly depressed, such as after severe hemorrhage.

Venous Resistance and Peripheral Venous Pressure

Large veins have so little resistance to blood flow *when they are distended* that the resistance then is almost zero and is of almost no importance. However, as shown in Figure 15-9, most of the large veins that enter the thorax are compressed at many points by the surrounding tissues so that blood flow is impeded at these points. For instance, the veins from the arms are compressed by their sharp angulations over the first rib. Also, the pressure in the neck veins often falls so low that the atmospheric pressure on the outside of the neck causes these veins to collapse. Finally, veins coursing through the abdomen are often compressed by different organs and by the intra-abdominal pressure, so they usually are at least partially collapsed to an ovoid or slitlike state. For these reasons, the *large veins do usually offer some resistance to blood flow,* and because of this, the pressure in the more peripheral small veins in a person lying down is usually +4 to +6 mm Hg greater than the right atrial pressure.

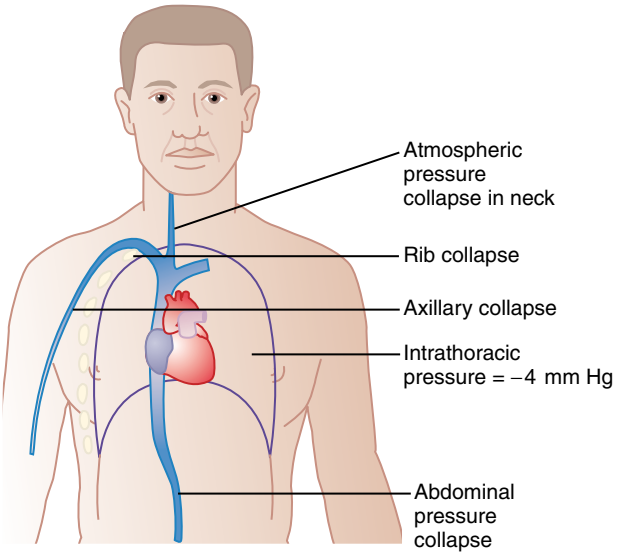


Figure 15-9 Compression points that tend to collapse the veins entering the thorax.

Effect of High Right Atrial Pressure on Peripheral Venous Pressure. When the right atrial pressure rises above its normal value of 0 mm Hg, blood begins to back up in the large veins. This enlarges the veins, and even the collapse points in the veins open up when the right atrial pressure rises above +4 to +6 mm Hg. Then, as the right atrial pressure rises still further, the additional increase causes a corresponding rise in peripheral venous pressure in the limbs and elsewhere. Because the heart must be weakened to cause a rise in right atrial pressure as high as +4 to +6 mm Hg, one often finds that the peripheral venous pressure is not noticeably elevated even in the early stages of heart failure.

Effect of Intra-abdominal Pressure on Venous Pressures of the Leg. The pressure in the abdominal cavity of a recumbent person normally averages about +6 mm Hg, but it can rise to +15 to +30 mm Hg as a result of pregnancy, large tumors, abdominal obesity, or excessive fluid (called "ascites") in the abdominal cavity. When the intra-abdominal pressure does rise, the pressure in the veins of the legs must rise *above* the abdominal pressure before the abdominal veins will open and allow the blood to flow from the legs to the heart. Thus, if the intra-abdominal pressure is +20 mm Hg, the lowest possible pressure in the femoral veins is also about +20 mm Hg.

Effect of Gravitational Pressure on Venous Pressure

In any body of water that is exposed to air, the pressure at the surface of the water is equal to atmospheric pressure, but the pressure rises 1 mm Hg for each 13.6 millimeters

of distance below the surface. This pressure results from the weight of the water and therefore is called *gravitational pressure* or *hydrostatic pressure.*

Gravitational pressure also occurs in the vascular system of the human being because of weight of the blood in the vessels, as shown in Figure 15-10. When a person is standing, the pressure in the right atrium remains about 0 mm Hg because the heart pumps into the arteries any excess blood that attempts to accumulate at this point. However, in an adult *who is standing absolutely still,* the pressure in the veins of the feet is about +90 mm Hg simply because of the gravitational weight of the blood in the veins between the heart and the feet. The venous pressures at other levels of the body are proportionately between 0 and 90 mm Hg.

In the arm veins, the pressure at the level of the top rib is usually about +6 mm Hg because of compression of the subclavian vein as it passes over this rib. The gravitational pressure down the length of the arm then is determined by the distance below the level of this rib. Thus, if the gravitational difference between the level of the rib and the hand is +29 mm Hg, this gravitational pressure is added to the +6 mm Hg pressure caused by compression of the vein as it crosses the rib, making a total of +35 mm Hg pressure in the veins of the hand.

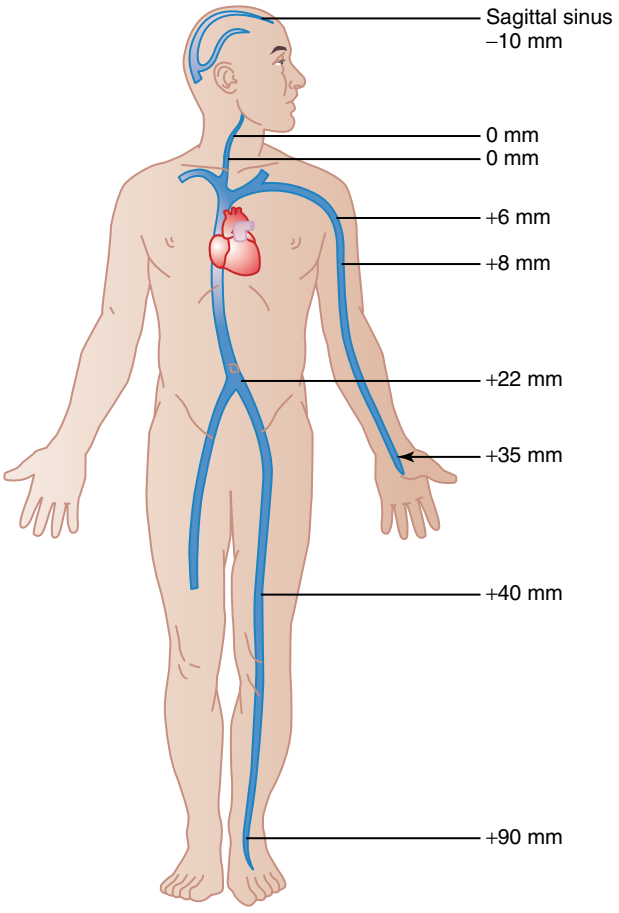


Figure 15-10 Effect of gravitational pressure on the venous pressures throughout the body in the standing person.

The neck veins of a person standing upright collapse almost completely all the way to the skull because of atmospheric pressure on the outside of the neck. This collapse causes the pressure in these veins to remain at zero along their entire extent. The reason for this is that any tendency for the pressure to rise above this level opens the veins and allows the pressure to fall back to zero because of flow of the blood. Conversely, any tendency for the neck vein pressure to fall below zero collapses the veins still more, which further increases their resistance and again returns the pressure back to zero.

The veins inside the skull, on the other hand, are in a noncollapsible chamber (the skull cavity) so that they cannot collapse. Consequently, *negative pressure can exist in the dural sinuses of the head;* in the standing position, the venous pressure in the sagittal sinus at the top of the brain is about –10 mm Hg because of the hydrostatic "suction" between the top of the skull and the base of the skull. Therefore, if the sagittal sinus is opened during surgery, air can be sucked immediately into the venous system; the air may even pass downward to cause air embolism in the heart, and death can ensue.

Effect of the Gravitational Factor on Arterial and Other Pressures. The gravitational factor also affects pressures in the peripheral arteries and capillaries, in addition to its effects in the veins. For instance, a standing person who has a mean arterial pressure of 100 mm Hg at the level of the heart has an arterial pressure in the feet of about 190 mm Hg. Therefore, when one states that the arterial pressure is 100 mm Hg, this generally means that this is the pressure at the gravitational level of the heart but not necessarily elsewhere in the arterial vessels.

Venous Valves and the "Venous Pump": Their Effects on Venous Pressure

Were it not for valves in the veins, the gravitational pressure effect would cause the venous pressure in the feet always to be about +90 mm Hg in a standing adult. However, every time one moves the legs, one tightens the muscles and compresses the veins in or adjacent to the muscles, and this squeezes the blood out of the veins. But the valves in the veins, shown in Figure 15-11, are arranged so that the direction of venous blood flow can be only toward the heart. Consequently, every time a person moves the legs or even tenses the leg muscles, a certain amount of venous blood is propelled toward the heart. This pumping system is known as the "venous pump" or "muscle pump," and it is efficient enough that under ordinary circumstances, the venous pressure in the feet of a walking adult remains less than +20 mm Hg.

If a person stands perfectly still, the venous pump does not work, and the venous pressures in the lower legs increase to the full gravitational value of 90 mm Hg in about 30 seconds. The pressures in the capillaries also increase greatly, causing fluid to leak from the circulatory system into the tissue spaces. As a result, the legs swell and the blood volume diminishes. Indeed, 10 to 20 percent of the blood volume can be lost from the circulatory

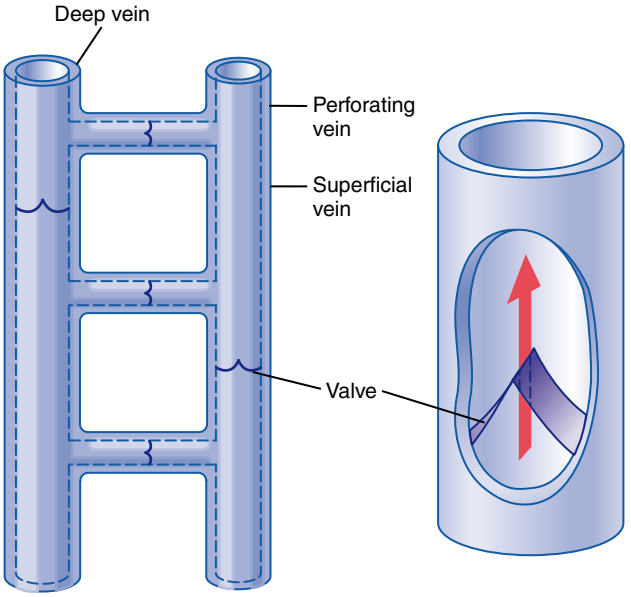


Figure 15-11 Venous valves of the leg.

system within the 15 to 30 minutes of standing absolutely still, as often occurs when a soldier is made to stand at rigid attention.

Venous Valve Incompetence Causes "Varicose" Veins. The valves of the venous system frequently become "incompetent" or sometimes even are destroyed. This is especially true when the veins have been overstretched by excess venous pressure lasting weeks or months, as occurs in pregnancy or when one stands most of the time. Stretching the veins increases their cross-sectional areas, but the leaflets of the valves do not increase in size. Therefore, the leaflets of the valves no longer close completely. When this develops, the pressure in the veins of the legs increases greatly because of failure of the venous pump; this further increases the sizes of the veins and finally destroys the function of the valves entirely. Thus, the person develops "varicose veins," which are characterized by large, bulbous protrusions of the veins beneath the skin of the entire leg, particularly the lower leg.

Whenever people with varicose veins stand for more than a few minutes, the venous and capillary pressures become very high and leakage of fluid from the capillaries causes constant edema in the legs. The edema in turn prevents adequate diffusion of nutritional materials from the capillaries to the muscle and skin cells, so the muscles become painful and weak and the skin frequently becomes gangrenous and ulcerates. The best treatment for such a condition is continual elevation of the legs to a level at least as high as the heart. Tight binders on the legs also can be of considerable assistance in preventing the edema and its sequelae.

Clinical Estimation of Venous Pressure. The venous pressure often can be estimated by simply observing the degree of distention of the peripheral veins—especially of the neck veins. For instance, in the sitting position, the neck veins are never distended in the normal quietly resting person. However, when the right atrial pressure becomes increased to as much as +10 mm Hg, the lower veins of the neck begin to protrude; and at +15 mm Hg atrial pressure essentially all the veins in the neck become distended.

Direct Measurement of Venous Pressure and Right Atrial Pressure

Venous pressure can also be measured with ease by inserting a needle directly into a vein and connecting it to a pressure recorder. The only means by which *right atrial pressure* can be measured accurately is by inserting a catheter through the peripheral veins and into the right atrium. Pressures measured through such *central venous catheters* are used almost routinely in some types of hospitalized cardiac patients to provide constant assessment of heart pumping ability.

Pressure Reference Level for Measuring Venous and Other Circulatory Pressures

In discussions up to this point, we often have spoken of right atrial pressure as being 0 mm Hg and arterial pressure as being 100 mm Hg, but we have not stated the gravitational level in the circulatory system to which this pressure is referred. There is one point in the circulatory system at which gravitational pressure factors caused by changes in body position of a healthy person usually do not affect the pressure measurement by more than 1 to 2 mm Hg. This is at or near the level of the tricuspid valve, as shown by the crossed axes in Figure 15-12. Therefore, all circulatory pressure measurements discussed in this text are referred to this level, which is called the *reference level for pressure measurement.*

The reason for lack of gravitational effects at the tricuspid valve is that the heart automatically prevents significant gravitational changes in pressure at this point in the following way:

If the pressure at the tricuspid valve rises slightly above normal, the right ventricle fills to a greater extent than usual, causing the heart to pump blood more rapidly and therefore to decrease the pressure at the tricuspid valve back toward the normal mean value. Conversely, if the pressure falls, the right ventricle fails to fill adequately, its pumping decreases, and blood dams up in the venous system until

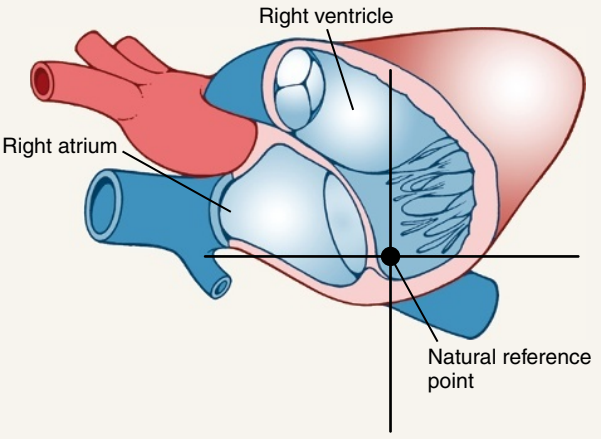


Figure 15-12 Reference point for circulatory pressure measurement (located near the tricuspid valve).

the pressure at the tricuspid level again rises to the normal value. In other words, *the heart acts as a feedback regulator of pressure* at the tricuspid valve.

When a person is lying on his or her back, the tricuspid valve is located at almost exactly 60 percent of the chest thickness in front of the back. This is the *zero pressure reference level* for a person lying down.

Blood Reservoir Function of the Veins

As pointed out in Chapter 14, more than 60 percent of all the blood in the circulatory system is usually in the veins. For this reason and also because the veins are so compliant, it is said that the venous system serves as a *blood reservoir* for the circulation.

When blood is lost from the body and the arterial pressure begins to fall, nervous signals are elicited from the carotid sinuses and other pressure-sensitive areas of the circulation, as discussed in Chapter 18. These in turn elicit nerve signals from the brain and spinal cord mainly through sympathetic nerves to the veins, causing them to constrict. This takes up much of the slack in the circulatory system caused by the lost blood. Indeed, even after as much as 20 percent of the total blood volume has been lost, the circulatory system often functions almost normally because of this variable reservoir function of the veins.

Specific Blood Reservoirs. Certain portions of the circulatory system are so extensive and/or so compliant that they are called "specific blood reservoirs." These include (1) the *spleen,* which sometimes can decrease in size sufficiently to release as much as 100 milliliters of blood into other areas of the circulation; (2) the *liver,* the sinuses of which can release several hundred milliliters of blood into the remainder of the circulation; (3) the *large abdominal veins,* which can contribute as much as 300 milliliters; and (4) the *venous plexus beneath the skin,* which also can contribute several hundred milliliters. The *heart* and the *lungs,* although not parts of the systemic venous reservoir system, must also be considered blood reservoirs. The heart, for instance, shrinks during sympathetic stimulation and in this way can contribute some 50 to 100 milliliters of blood; the lungs can contribute another 100 to 200 milliliters when the pulmonary pressures decrease to low values.

The Spleen as a Reservoir for Storing Red Blood Cells. Figure 15-13 shows that the spleen has two separate areas for storing blood: the *venous sinuses* and the *pulp.* The sinuses can swell the same as any other part of the venous system and store whole blood.

In the splenic pulp, the capillaries are so permeable that whole blood, including the red blood cells, oozes through the capillary walls into a trabecular mesh, forming the *red pulp.* The red cells are trapped by the trabeculae, while the plasma flows on into the venous sinuses and then into the general circulation. As a consequence, the red pulp of the spleen is a *special reservoir that contains large quantities of concentrated red blood cells.* These can then be expelled into the general circulation whenever the sympathetic nervous system becomes excited and causes the spleen and its vessels to contract. As much as 50 milliliters of concentrated red blood cells can be released into the circulation, raising the hematocrit 1 to 2 percent.

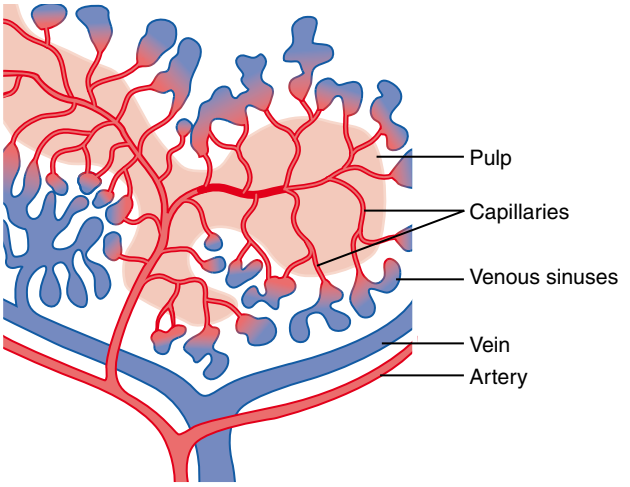


Figure 15-13 Functional structures of the spleen. (Courtesy Dr. Don W. Fawcett, Montana.)

In other areas of the splenic pulp are islands of white blood cells, which collectively are called the *white pulp.* Here lymphoid cells are manufactured similar to those manufactured in the lymph nodes. They are part of the body's immune system, described in Chapter 34.

Blood-Cleansing Function of the Spleen—Removal of Old Cells

Blood cells passing through the splenic pulp before entering the sinuses undergo thorough squeezing. Therefore, it is to be expected that fragile red blood cells would not withstand the trauma. For this reason, many of the red blood cells destroyed in the body have their final demise in the spleen. After the cells rupture, the released hemoglobin and the cell stroma are digested by the reticuloendothelial cells of the spleen, and the products of digestion are mainly reused by the body as nutrients, often for making new blood cells.

Reticuloendothelial Cells of the Spleen

The pulp of the spleen contains many large phagocytic reticuloendothelial cells, and the venous sinuses are lined with similar cells. These cells function as part of a cleansing system for the blood, acting in concert with a similar system of reticuloendothelial cells in the venous sinuses of the liver. When the blood is invaded by infectious agents, the reticuloendothelial cells of the spleen rapidly remove debris, bacteria, parasites, and so forth. Also, in many chronic infectious processes, the spleen enlarges in the same manner that lymph nodes enlarge and then performs its cleansing function even more avidly.

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