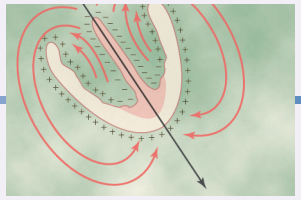

CHAPTER 12

UNIT III

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 (ECG). 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

To understand how cardiac abnormalities affect the contours of the ECG, one must first become familiar with the concept of *vectors* and *vectorial analysis* as applied to electrical potentials in and around the heart.

In Chapter 11 we 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. The shaded area and the minus signs in **Figure 12-1** show depolarization of the ventricular septum and of parts of the apical endocardial walls of the two ventricles. At the 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 the base toward the apex. Furthermore, because the summated current is considerable in quantity, the potential is large and the vector is long.

THE 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.

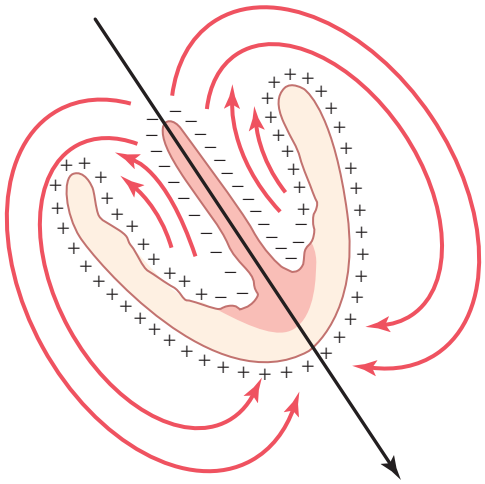

Figure 12-1. Mean vector through the partially depolarized ventricles.

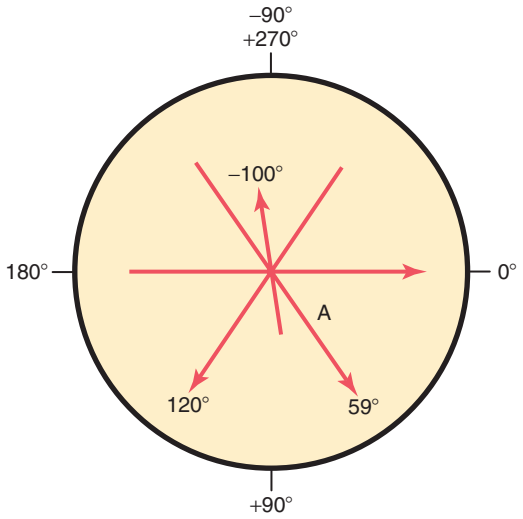

Figure 12-2. Vectors drawn to represent potentials for several different hearts, and the axis of the potential (expressed in degrees) for each heart.

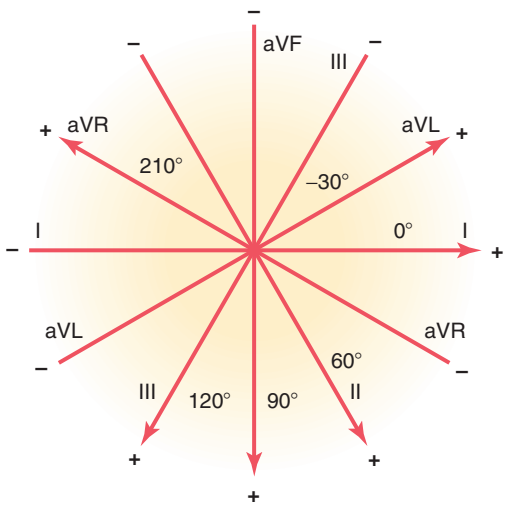

Figure 12-3. Axes of the three bipolar and three unipolar leads.

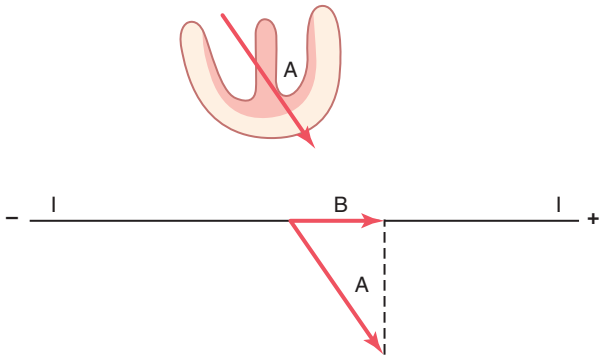

Figure 12-4. Determination of a projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

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.*

VECTORIAL ANALYSIS OF POTENTIALS RECORDED IN DIFFERENT LEADS

Figure 12-4 shows a partially depolarized heart, with vector *A* representing 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 millivolts. 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 ECG of lead I is positive. 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 side. 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 it is in the negative direction, indicating that at

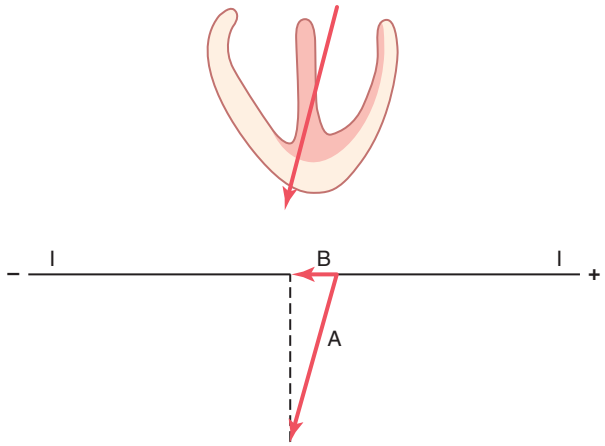

Figure 12-5. Determination of the projected vector B along the axis of lead I when vector A represents the instantaneous potential in the ventricles.

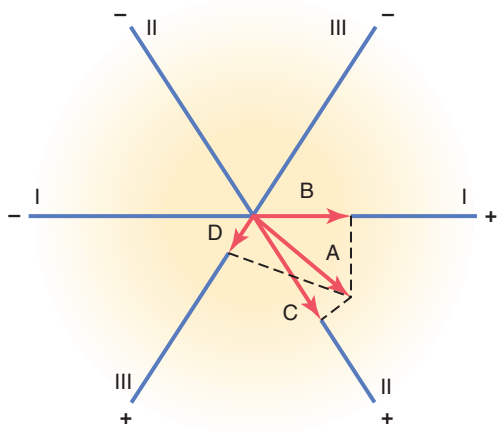

Figure 12-6. Determination of projected vectors in leads I, II, and III when vector A represents the instantaneous potential in the ventricles.

this particular instant, the recording in lead I will be negative (below the zero line in the ECG), 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 ECG 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 ECG 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 ECG 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.

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 ECG 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

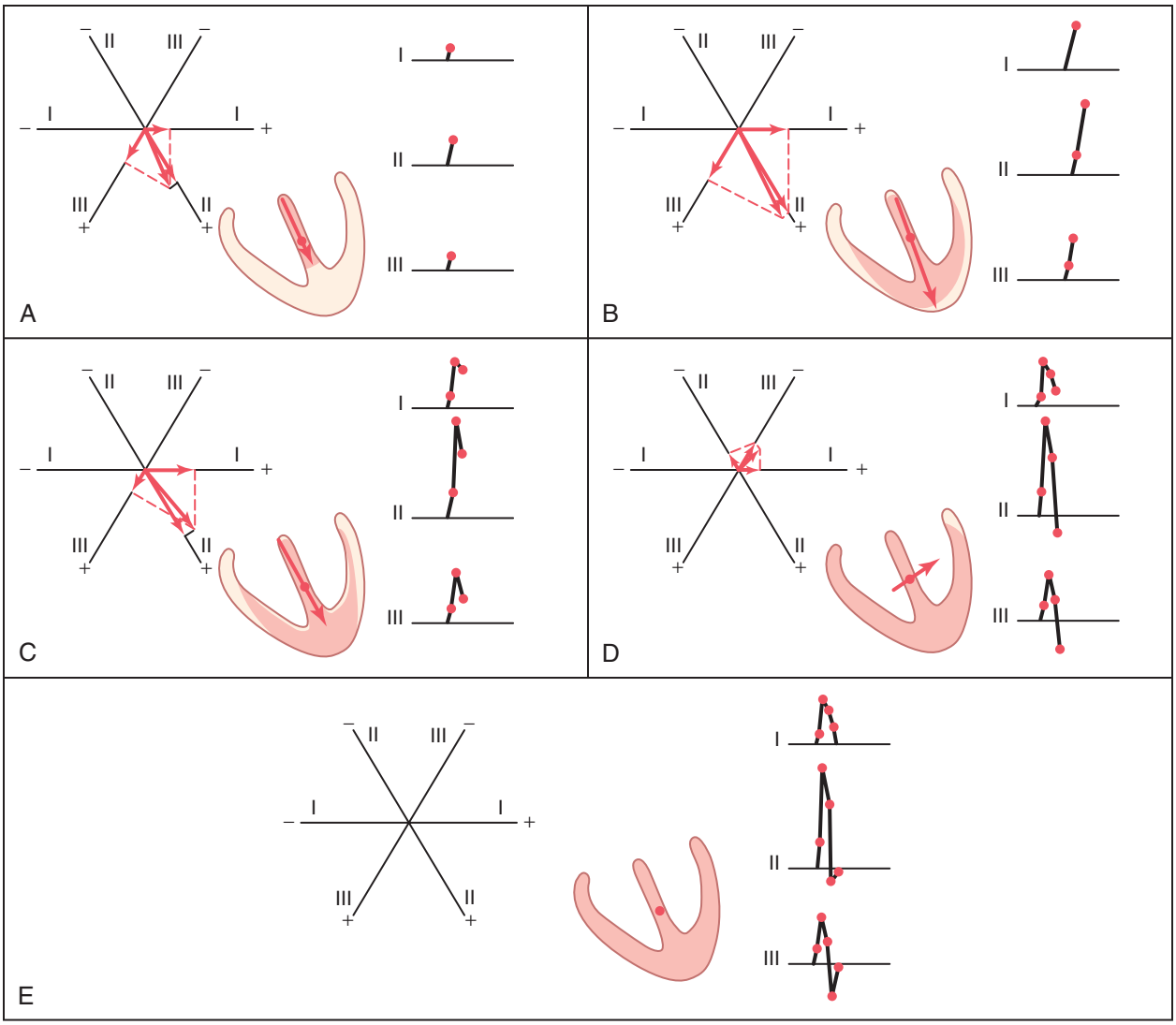

Figure 12-7. Shaded areas of the ventricles are depolarized (–); nonshaded areas are still polarized (+). The ventricular vectors and QRS complexes 0.01 second after onset of ventricular depolarization (**A**); 0.02 second after onset of depolarization (**B**); 0.035 second after onset of depolarization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

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.

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 is the right ventricle. 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 ECG.

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 do 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 ECGs 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 entire process to take place, the T wave of the ECG is generated.

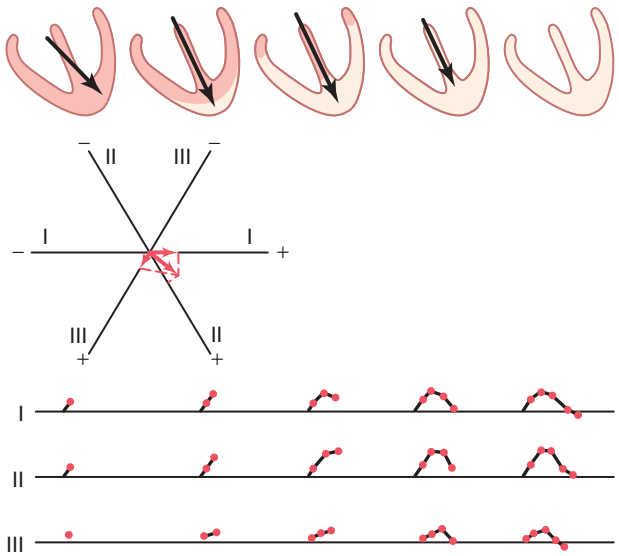

Figure 12-8. Generation of the T wave during repolarization of the ventricles, showing also vectorial analysis of the first stage of repolarization. The total time from the beginning of the T wave to its end is approximately 0.15 second.

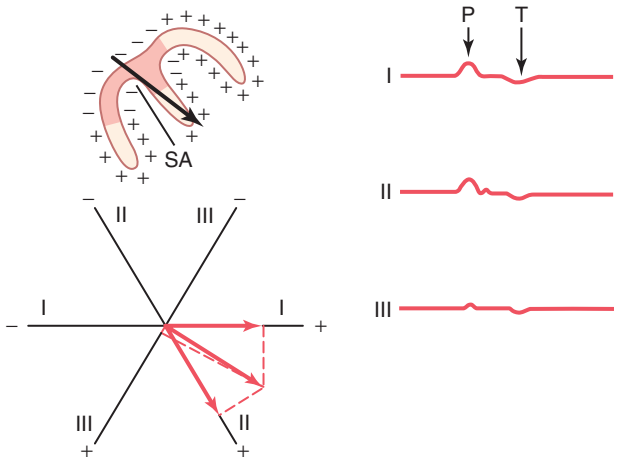

Figure 12-9. Depolarization of the atria and generation of the P wave, showing the maximum vector through the atria and the resultant vectors in the three standard leads. At the right are the atrial P and T waves. SA, sinoatrial node.

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 ECGs 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. Consequently, *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 remainder of the atria. Therefore, the atrial repolarization 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 a normal ECG, 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 ECG.

Vectorcardiogram

As noted previously, 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 *vectorcardiogram* depicts these changes at different times during the cardiac cycle, as shown in Figure 12-10.

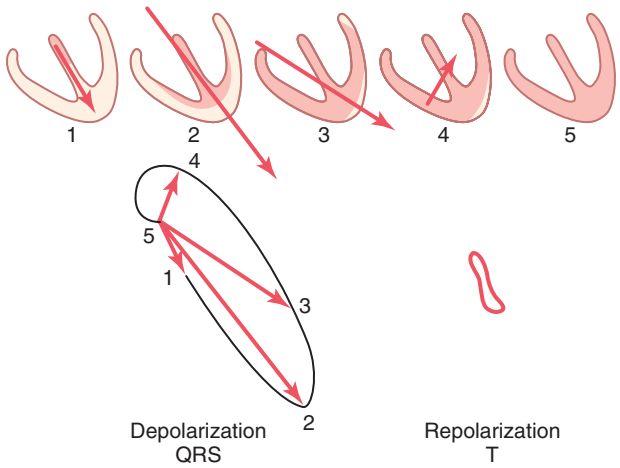

Figure 12-10. QRS and T vectorcardiograms.

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.

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 ECGs

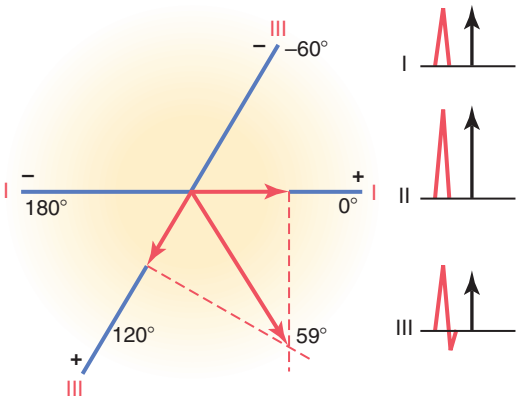

Figure 12-11. Plotting the mean electrical axis of the ventricles from two electrocardiographic leads (leads I and III).

rather than from the vectorcardiogram. **Figure 12-11** shows a method for performing this estimation. 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**.

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 a 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 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 as a result of 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 shift 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 greater quantity of muscle exists on the hypertrophied side of the heart than on the other side, which 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 situation 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 ECGs. Vectorial analysis demonstrates left axis deviation with mean electrical axis pointing in the –15-degree direction. This is a typical ECG 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 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

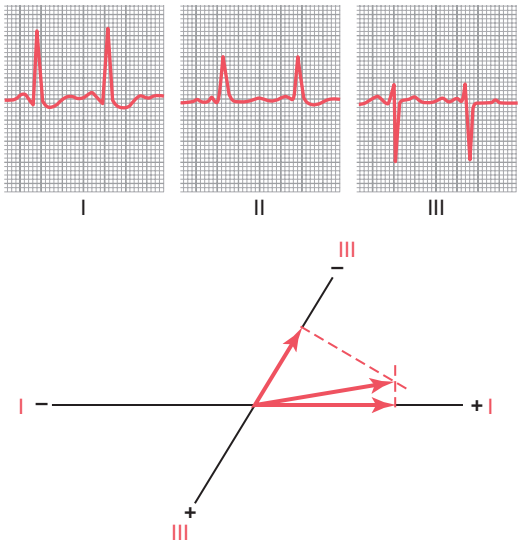

Figure 12-12. Left axis deviation in a *hypertensive heart (hypertrophic left ventricle).* Note the slightly prolonged QRS complex as well.

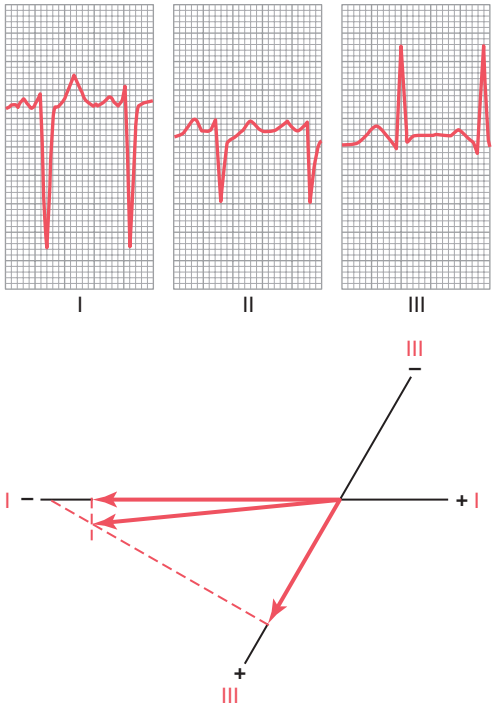

Figure 12-13. A high-voltage electrocardiogram for a person with *congenital pulmonary valve stenosis with right ventricular hypertrophy.* Intense right axis deviation and a slightly prolonged QRS complex also are seen.

enlarges while the right ventricle remains relatively normal in size.

Vectorial Analysis of Right Axis Deviation Resulting from Hypertrophy of the Right Ventricle. The ECG 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. However, 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 ventricle. Therefore, depolarization of the two ventricles does not occur even nearly at the same time, and the depolarization potentials do not neutralize each other. As a result, axis deviation occurs as follows.

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, intense left axis deviation of about –50 degrees occurs because the positive end of the vector points toward the left ventricle. This situation 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 as a result of extreme slowness of depolarization in the affected side of the heart. One can see this effect by observing the excessive widths of the QRS waves in **Figure 12-14**. This topic 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 does the right ventricle, and thus 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

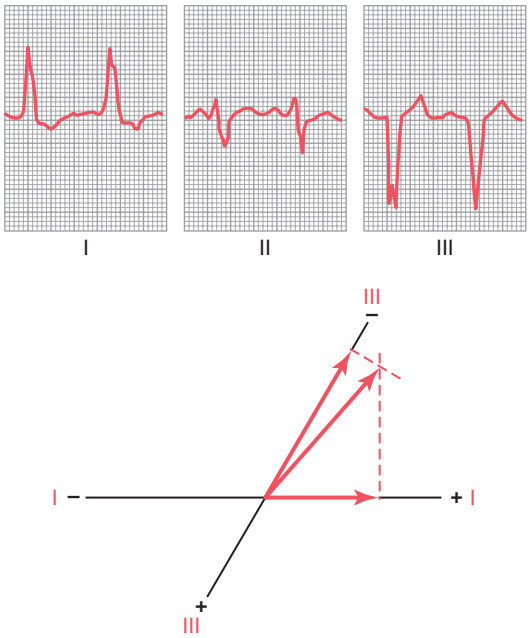

Figure 12-14. Left axis deviation caused by *left bundle branch block*. Note also the greatly prolonged QRS complex.

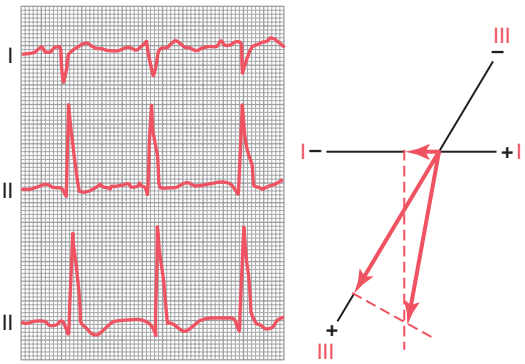

Figure 12-15. Right axis deviation caused by *right bundle branch block*. Note also the greatly prolonged QRS complex.

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

CONDITIONS THAT CAUSE ABNORMAL VOLTAGES OF THE QRS COMPLEX

INCREASED VOLTAGE IN THE STANDARD BIPOLAR LIMB LEADS

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

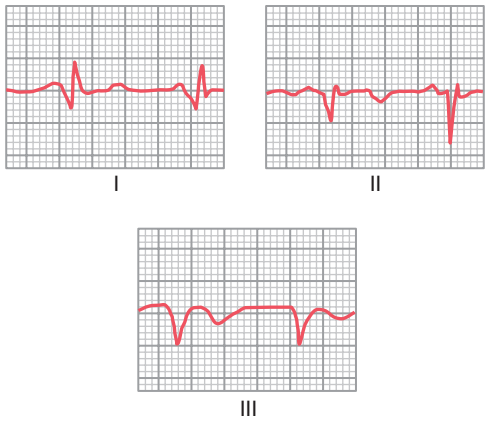

Figure 12-16. A low-voltage electrocardiogram following local damage throughout the ventricles caused by *previous myocardial infarction*.

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

DECREASED VOLTAGE OF THE ELECTROCARDIOGRAM

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

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