The Electrocardiogram 5.6

Learning Objectives

- Define what is meant by ECG and describe its utility in medicine and physiology
- Define Einthoven's Triangle and write the convention for leads I, II, and III
- Explain why Kirchhoff's law results in lead I + lead III = lead II
- Identify the main features of the ECG including the P, QRS complex, and T wave
- Be able to determine the heart's electric dipole from the value of leads I and III
- Identify the PR interval, PR segment, ST segment, and QT interval from an ECG trace
- Describe the events in the heart that produce the P wave
- Describe the events in the heart that produce the QRS complex
- Describe the events in the heart that produce the T wave
- Describe the general idea of augmented limb leads
- Define mean electrical axis, left axis deviation, and right axis deviation
- Describe why Einthoven's triangle is an idealized abstraction that is useful but not strictly valid

THE ECG IS THE PROJECTION OF CARDIAC ELECTRICAL ACTIVITY ONTO THE BODY SURFACE

The electrocardiogram (ECG) is well known as an integral part of the examination of patients, but its physical basis and the source of its signals are less well known. **The ECG is a record of the electrical activity of the heart that is projected onto the surface of the body where it is measured by surface electrodes**. Because this record represents the heart's electrical events, it aids diagnosis of electrical abnormalities. Its usefulness resides entirely in its clinical application. The cardiologist uses the ECG to understand the electrical character of the heart including (a) excitation of the pacemakers; (b) spread of activation from one region of the heart to another; (c) the pathways by which the wave of activation spreads; and (d) the basis of the action potential itself. The ECG will not illuminate the mechanical effectiveness of the heart: the ECG can be perfectly normal in mechanically ineffective hearts, whereas abnormal ECGs can be recorded in a mechanically sound heart. Clinical abnormalities in the ECG are legion, and many years of experience have produced systematic analyses of these abnormalities. Here we focus on how the normal ECG derives from the normal heart beat.

THE HEART MUSCLE FIBERS ACT AS *ELECTRIC DIPOLES*

As excitation of the cardiac muscle is conveyed through the myocardium, the electrical state of the muscle fibers differs from place to place. At some time during the conduction of the action potential, some parts of the muscle are depolarized and other parts are not. This produces the equivalent of an electrical dipole, as shown in Figure 5.6.1. The electric dipole is discussed in Chapter 1.4 (See Appendix 1.4.A1). The **dipole moment** is given as

$$\vec{p} = q_+ \vec{d} \quad [5.6.1]$$

where $\boldsymbol{d}$ is a vector pointing from q_- to q_+, and q_+ is the magnitude of charge separated by distance, d. A potential surrounds the dipole, which is just the sum of the potentials due to each of the charges. It is given (Appendix 1.4.A1) approximately as

$$V = \frac{p \cos\theta}{4\pi\varepsilon_0 r^2} \quad [5.6.2]$$

The electric dipole of the heart produces an electrical potential throughout the thorax and projected onto the skin. Under ideal conditions, this electric potential can be calculated or graphically constructed by the projection laws of dipole vectors, but these simple calculations or projections are valid only if the body is a spherical, homogeneous conductor, with the dipole lying at the center of the sphere. The body is not such an ideal spherical conductor.

EINTHOVEN IDEALIZED THE THORAX AS A TRIANGLE

Although not the first to record electrical activity from a heart, Einthoven (1860–1927) devised a string galvanometer to record the small potentials accurately, and he provided a simplified analysis of the ECG that is still used today. Einthoven was awarded the 1924 Nobel Prize in Medicine and Physiology for his contributions to the measurement and interpretation of the ECG.

Einthoven imagined that the electrical state of the heart at any time could be represented by a single vector, representing the electric dipole moment, located in the center of the thorax. Electrodes attached to the right

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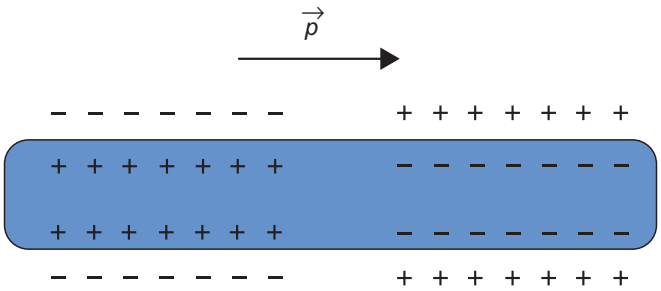

FIGURE 5.6.1 The equivalent dipole of the heart muscle during partial activation. The muscle at rest is polarized, negative inside, as shown to the right in the muscle. Activation of the muscle begins by its depolarization, with positive potential inside. This situation produces a dipole moment as shown. As activation proceeds, the dipole moment changes and so does the voltage projected to the surface of the skin.

arm (RA), left arm (LA), and left leg (LL) ideally measure voltages at the apices of an equilateral triangle that has the heart at its center (see Figure 5.6.2) The voltage differences between the different electrodes can be measured in three combinations:

$$\text{Left arm} - \text{right arm} = \text{LEAD I}$$
$$\text{Left leg} - \text{right arm} = \text{LEAD II}$$
$$\text{Left leg} - \text{left arm} = \text{LEAD III}$$

These leads are bipolar leads, consisting of the potential difference between two sites. The voltages recorded at these bipolar leads are not independent. Because of Kirchoff's voltage law, which states that the voltage drop around any closed circuit is zero, we have

$$\text{I} + \text{III} = \text{II} \qquad [5.6.3]$$

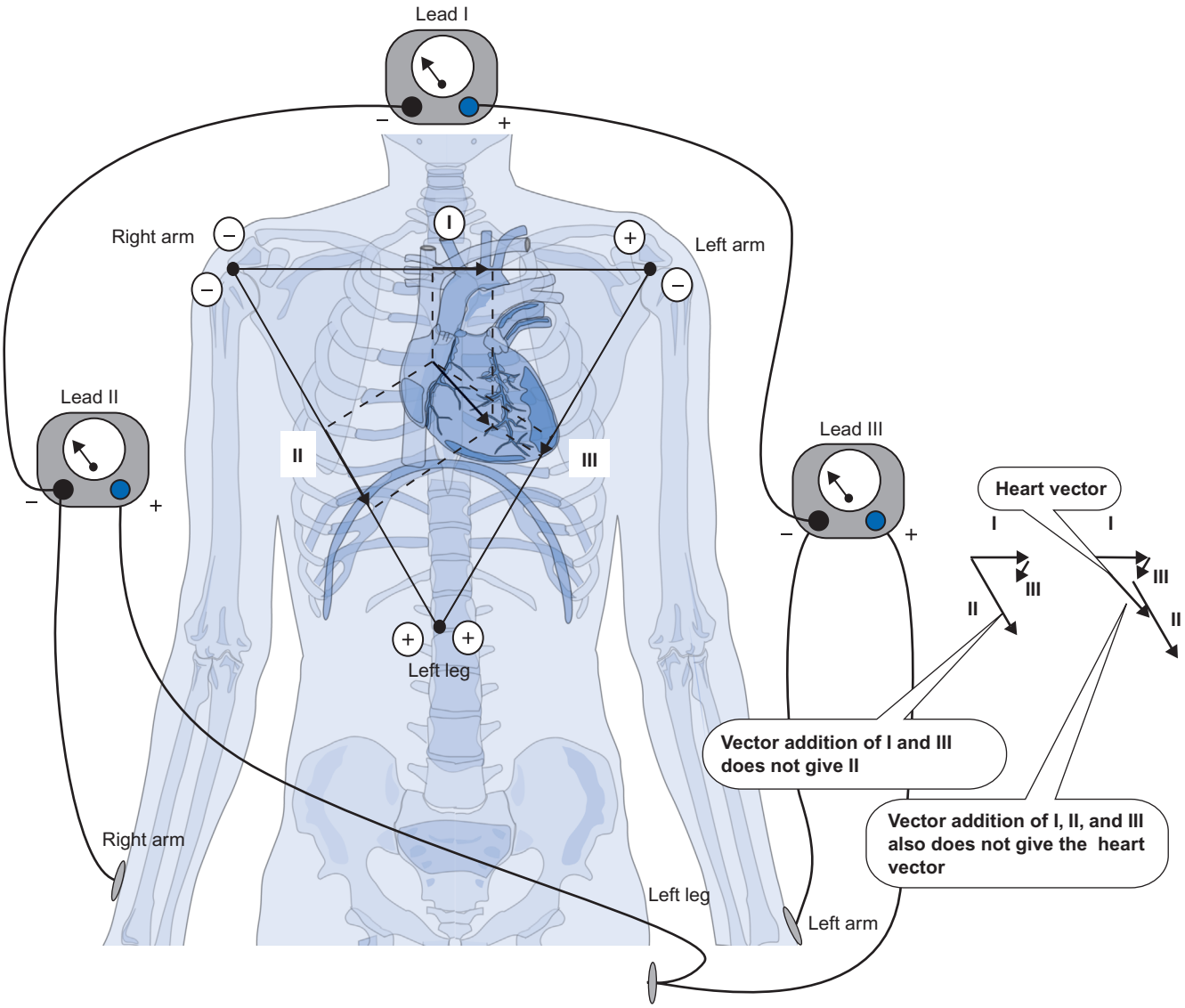

FIGURE 5.6.2 Einthoven's triangle. The electrodes are placed on the right arm, left arm, and left leg, which are considered to measure voltages as indicated on the torso. The three electrodes ideally form an equilateral triangle with the heart's electric dipole at its center. The voltage measured along any axis is the projection of the dipole moment onto that axis, as shown. The dipole vectors do not add like normal vectors, head to tail, because the triangular coordinate system is not orthogonal.

so that the voltage in the third bipolar lead can always be calculated from the other two. This peculiar form of Kirchoff's voltage law is due to the fact that lead II is reported in the counterclockwise direction in Figure 5.6.2 (left leg—right arm) rather than in the clockwise direction that is used for leads I and III. This convention causes the voltages to add as in Eqn [5.6.3]. Einthoven selected this convention because he wanted positive voltages corresponding to the cardiac impulse in all leads in normal individuals.

The voltages recorded in each of the bipolar leads at any instant is equal to the projection of the heart's electric dipole at that instant onto the bipolar lead. This projection is shown in Figure 5.6.2.

THE HEART'S ELECTRIC DIPOLE MOMENT VARIES WITH TIME—AND SO DOES ITS RECORDING ON LEADS I, II, AND III

At rest, the heart fibers are uniformly polarized and so there is no net dipole moment and therefore no voltage recorded on leads I, II, or III. The heart muscle depolarizes in a coordinated matter, with the atria depolarizing first, and the ventricles depolarizing later and eventually repolarizing. This sequence of the cardiac impulse within the muscle fibers causes the electric dipole of the entire heart to change magnitude and direction with time. The resulting projections of the electric dipole moment onto the limb leads produces a record of voltage that varies with time—the **scalar electrocardiogram**, or **ECG**. The ECG is typically produced at a chart speed of 25 mm s^{-1} on a scale of 1 mV cm^{-1}. A typical example of an ECG is shown in Figure 5.6.3.

THE VALUES OF LEADS I AND III CAN BE USED TO CALCULATE THE ELECTRIC DIPOLE MOMENT OF THE HEART

The description above indicates that the values recorded on leads I, II, and III at any time are the result of the electric dipole moment of the heart at that time. However, what we measure are the voltages on these leads while the electric dipole moment remains unknown. The direction and magnitude of the electric dipole moment can be approximately reconstructed from the measured voltages. Reconstruction of the heart's electrical axis cannot be done by ordinary vector addition, however. Figure 5.6.2 shows that addition of vectors I and III does not produce vector II, nor does addition of I, II, and III produce the electrical axis of the heart. The reason for this is that the vectors I, II, and III are projections of the heart's dipole moment onto axes that are not **orthogonal**.

In orthogonal coordinate systems, a vector parallel to one coordinate has no components in the direction of any other coordinate. In the triangular coordinate system, a vector along one axis *does* have components along both of the other axes. What this means is that the electric dipole can be reconstructed only by reversing the projection onto the axes, as described in the legend to Figure 5.6.4.

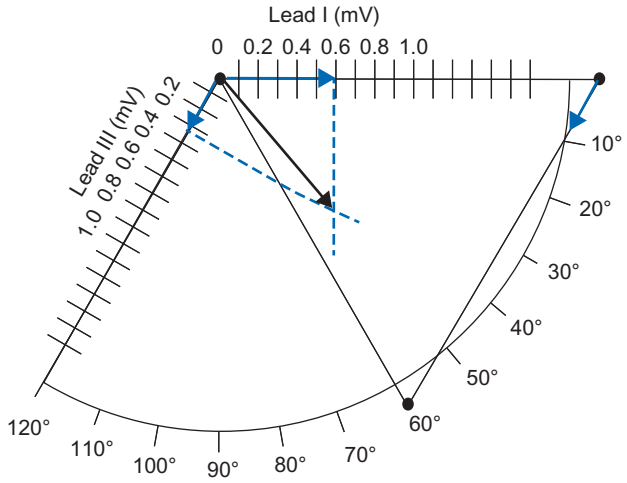

FIGURE 5.6.4 Reconstruction of the heart's electrical dipole from leads I and III. A vector whose magnitude is the voltage of lead I is drawn with its starting point at the origin and its end on the lead I axis. A second vector equal to the voltage of lead III is drawn the same way but oriented on an axis parallel to the lead III axis and starting at the origin. The electric dipole extends from the origin to the intersection of the two lines drawn perpendicular to the axes at the ends of the lead I and lead III vectors. The electric dipole of the heart obtained this way pertains to the time leads I and III were measured. This dipole has a magnitude (its length in the same units as for the two lead axes) and a direction. The direction is usually given in degrees using the convention shown.

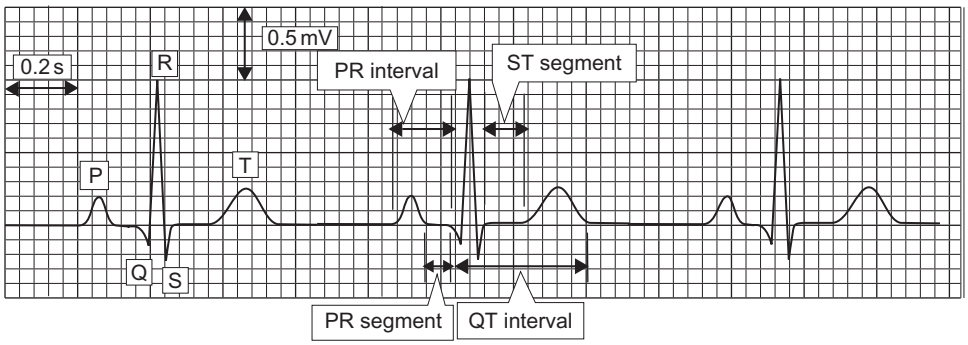

FIGURE 5.6.3 Typical appearance of the ECG on lead II ($V_{LL} - V_{RA}$). The ECG consists of several named electrical events, beginning with the P wave. The P wave is the first electrical event of the heart cycle and corresponds to the depolarization of the atria. The QRS complex corresponds to depolarization of the ventricles. Buried within it is the electrical signature of the repolarization of the atria. The T wave corresponds to repolarization of the ventricles. It is upright because of the sequence of repolarization of different parts of the ventricles, as described in the text.

ATRIAL DEPOLARIZATION CAUSES THE P WAVE

The size and magnitude of the cardiac electric dipole changes continuously as excitation spreads through the heart. The stepwise state of the heart and its corresponding electric dipole throughout the cardiac cycle is shown in Figures 5.6.5 and 5.6.6. The heart beat begins with SA node depolarization to threshold and initiation of the cardiac impulse in the SA node. This impulse travels outward through the right atrium and, via Bachmann's bundle, to the left atrium. This produces an electric dipole with positive contributions along the lead II axis, and so it is recorded as a positive voltage on this lead. When the atria are completely depolarized, there is no remaining dipole except over the small amount of tissue that makes up the AV node. Because of its small size, this tissue does not produce much voltage at the leads.

SEQUENTIAL DEPOLARIZATION OF THE VENTRICLES PRODUCES THE QRS COMPLEX

As described in Chapter 5.4, conduction of the cardiac impulse to the ventricles is delayed by the AV node. This allows the atria to contract and fully load the ventricles prior to their contraction and ejection of blood into the pulmonary and systemic circulations. This delay shows up as a lag between the P wave and the QRS complex, the part of the ECG that derives from ventricular depolarization. The nodal delay is typically about 0.1 s. Depolarization of the ventricles then occurs in a sequential fashion beginning with the left ventricular septum. At the very beginning of the QRS complex, Q is usually slightly negative. This is due to the fact that the cardiac dipole at the beginning of activation points slightly away from the lead II axis, and therefore its projection is in the opposite direction. Part of the Q wave in the QRS complex also results from atrial repolarization (see Figure 5.6.6).

THE SUBEPICARDIUM REPOLARIZES BEFORE THE SUBENDOCARDIUM, CAUSING AN UPRIGHT T WAVE

The **epicardium** is the layer of cells on the outer surface of the heart, facing the pericardial fluid. The **endocardium** is the layer of cells lining the inner surface of the heart, facing the blood. Cardiomyocytes adjacent to these layers are called the **subepicardium** and **subendocardium**, respectively. Cells in the middle layer of the ventricle form the **midmyocardium**. These cells are somewhat heterogeneous in that their action potentials have different durations. Cells in the subendocardium depolarize before cells in the subepicardium. However, these cells repolarize roughly in

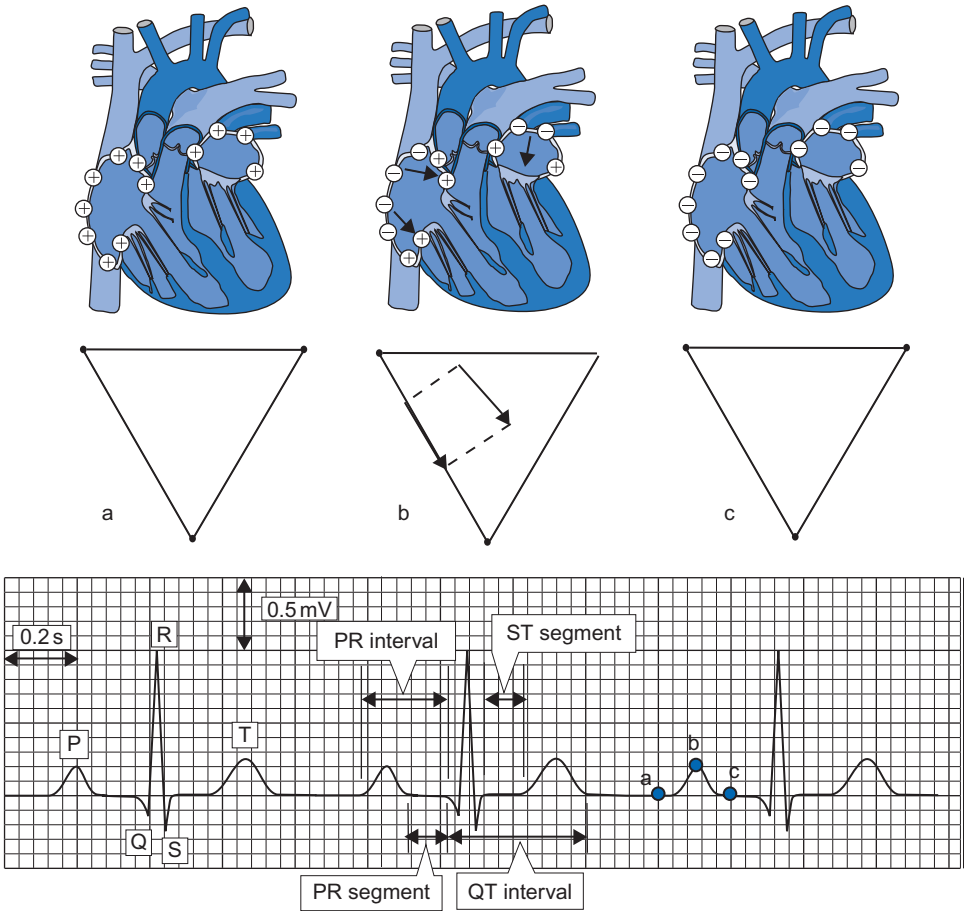

FIGURE 5.6.5 Origin of the P wave on the ECG. At rest all of the heart cells are polarized and there is no electric dipole. When the SA node depolarizes to threshold and initiates a cardiac impulse, the excitation is carried over the atria sequentially, with cells nearest the SA node depolarizing first. Bachmann's bundle simultaneously carries the excitation from the SA node to the left atrium, causing simultaneous activation of right and left atria. This pattern of depolarization creates an electric dipole directed more or less along the lead II axis and it is picked up by the ECG as the P wave. The magnitude of the lead II voltage at any time is the projection of the cardiac electric dipole moment onto the lead II axis, as shown in (b). This is always true, but the magnitude of the electric dipole is zero prior to activation (a) and after the atria are completely depolarized (c).

the inverse order of their depolarization: the last cells to depolarize (the subepicardium) are the first to repolarize. This is due to the length of their action potentials. Cells in the subepicardium have shorter action potentials than cells in the subendocardium. The consequence is that the cardiac dipole points along the lead II axis during repolarization, and the T wave, the electrical event corresponding to ventricular repolarization, is positive (see Figure 5.6.6).

THE CARDIAC DIPOLE TRACES A CLOSED CURVE DURING EACH HEART BEAT

Figure 5.6.6 d, e, and f show that the cardiac electric dipole moment changes its direction and magnitude through the QRS complex. A more detailed analysis shows that the cardiac dipole changes continuously so that the end of the vector traces a closed curve every time the heart beats. The entire cycle lasts the duration of the QRS complex, which is approximately 80 ms. An approximation of the dipole and its location at various times after the beginning of the QRS complex is shown in Figure 5.6.8.

THE LARGEST DEPOLARIZATION DEFINES THE *MEAN ELECTRICAL AXIS*

The cycle shown in Figure 5.6.8 is elongated toward the vector produced upon depolarization of the thick left ventricular wall. This elongation defines an **electrical axis** of the heart, which normally should relate to the heart's anatomical position within the thorax. The electrical axis is most easily defined as the cardiac electric dipole at the

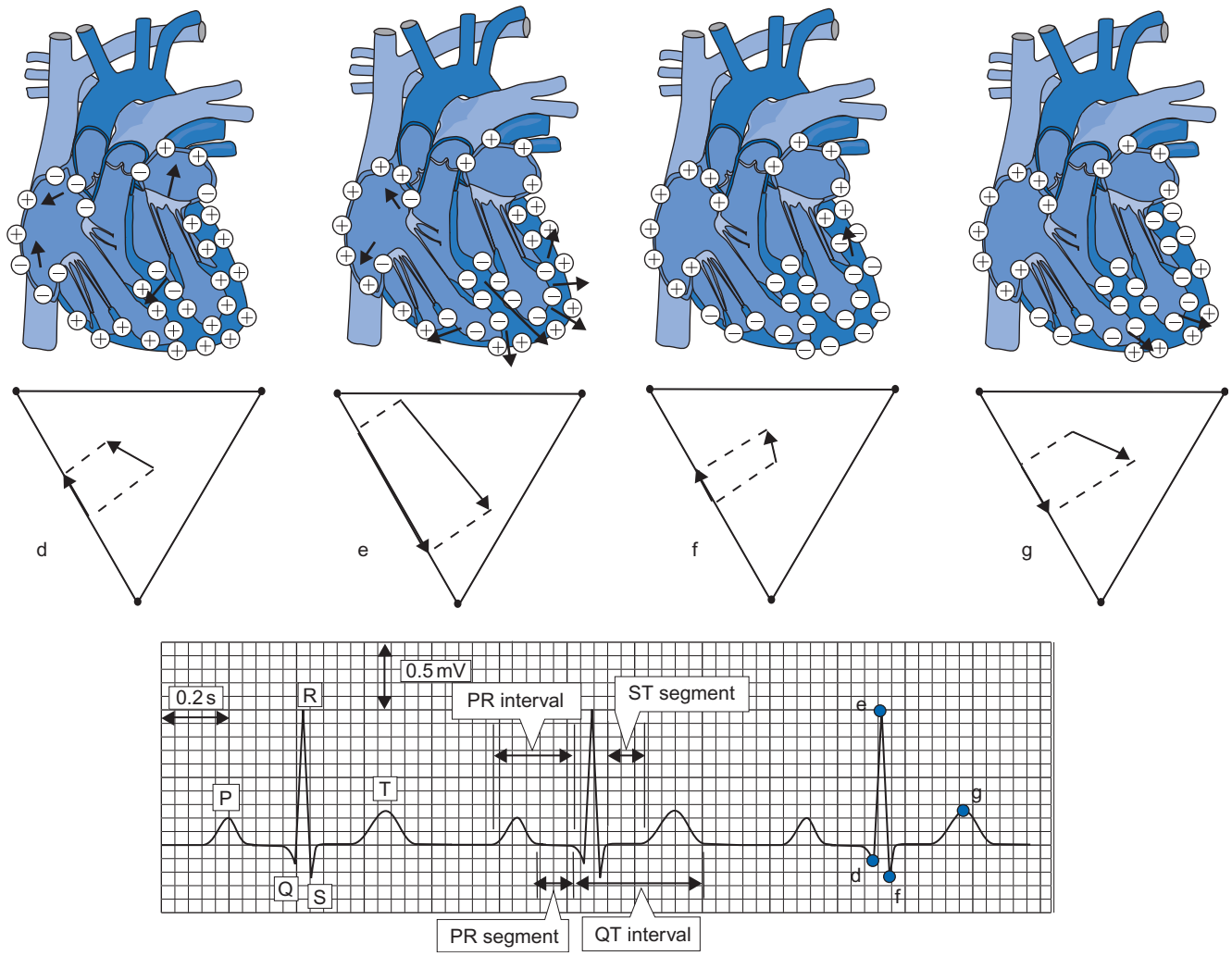

FIGURE 5.6.6 Origin of the QRS complex and T wave of the lead II scalar ECG. The spread of activation during ventricular activation creates a cardiac dipole that varies in magnitude and direction because of the sequence of activation produced by the left and right bundle branches and the Purkinje fibers. The Q wave is negative because the depolarization of the ventricles begins with the septum and the resulting dipole points away from the lead II axis (d). The largest vector arises from depolarization of the inner (subendocardium) apex of the heart, while the outer (subepicardium) apex remains polarized. This forms the R wave (e). The S wave is also negative and arises from the last depolarization of the free left ventricular wall near the left atrium. At this time the dipole vector points away from the lead II axis (f). Repolarization of the atria is buried in the QRS complex. Repolarization of the T wave is upright because the subepicardium has a shorter action potential and repolarizes before the subendocardium, creating a dipole pointing along the lead II axis (g).

EXAMPLE 5.6.1 Calculate the Heart Vector

At the top of the R wave, lead I reads 0.35 mV and lead III reads 0.75 mV. Calculate the heart vector at this time.

Refer to Figure 5.6.7. What we desire is the length (magnitude) of vector V_H and the angle it makes with respect to the lead I (horizontal) axis.

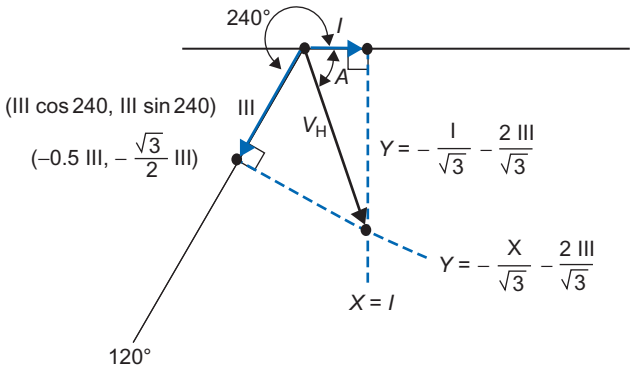

FIGURE 5.6.7 Calculation of the heart vector from lead I and lead III. The vector is the line from the origin to the point of intersection of two perpendiculars drawn from the end point of lead I and lead III. It is given as the angle with respect to lead I and its magnitude, in mV.

This can be calculated in various ways. Using analytical geometry, we can write the equation for the two lines perpendicular to the lead I and lead III axes at the end of the lead I and lead III vectors. The line perpendicular to the lead I is just $X = I$, where I is the magnitude of lead I. The line perpendicular to lead III can be calculated by knowing that its slope is $-1/\tan 240 = -1/\sqrt{3}$ and it passes through the point (X,Y) at the end of the vector, which is given as (III cos 240, III sin 240) = (−0.5 III, $-\sqrt{3}$/2 III), where III is the magnitude of lead III. This equation is given in the point-slope form as

$(Y + \sqrt{3}/2\ III)/(X + 0.5\ III) = -1/\sqrt{3}$ which can be rearranged to

$Y = -X/\sqrt{3} - 0.5\ III/\sqrt{3} - \sqrt{3}\ III/2$ or $[-X\ \ 2\sqrt{3} - III\ \ 4\sqrt{3}]/6$

The intersection of the two lines is given as $(I, [-I\ \ 2\sqrt{3} - III\ \ 4\sqrt{3}]/6)$

$$\text{Angle } A = \tan^{-1}(Y/I) = \tan^{-1}(-1/\sqrt{3} - 2/\sqrt{3}\ \ III/I)$$

For I = 0.35 and III = 0.75, this is $A = \tan^{-1}(-0.5774 - 2.4744) = \tan^{-1}(-3.05176) = -71.86°$. Convention takes this as positive **71.86°**.

The magnitude of the heart vector can then be calculated as $V_H = I/\cos A = 0.35/\cos(-71.86) =$ **1.12 mV**.

peak of the curve traced out in Figure 5.6.8. Because of its tilt, however, the projection of the vector tracing this curve peaks at different times on the lead I and lead III axes. The electrical axis can be determined from lead I and lead III ECGs using the voltage of the R wave and determining the resulting cardiac dipole as described in Figure 5.6.4.

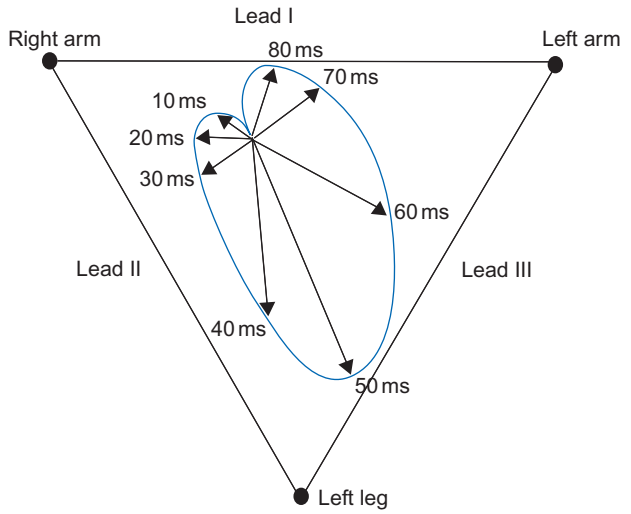

FIGURE 5.6.8 Approximate changes in the direction and magnitude of the cardiac dipole during ventricular excitation. The projection of the dipole on the different leads produces the ECG and accounts for different shapes of the QRS complex recorded in leads I, II, and III.

Typically the electrical axis is oriented about 60° below the horizontal, but it varies widely from −30° to 110°. The electrical axis depends in part on the anatomical orientation of the heart. A tall, thin person generally would have a more vertically oriented electrical axis compared to a short, stout person. As described in Chapter 5.4, the heart lies within a tough fibrous sac, the pericardium, which in turn is fused to the diaphragm. During inspiration, the diaphragm moves downward to expand the thoracic cavity. This movement pulls the heart into a more vertical orientation. Thus, the electrical axis normally becomes more vertical during inspiration and more horizontal during expiration.

The electrical axis also depends on the relative contributions of the right and left ventricles to the ECG. Hypertrophy of the left ventricle shifts the axis, causing **left axis deviation**. Hypertrophy of the right ventricle occurs in response to pulmonary hypertension and is associated with **right axis deviation**.

UNIPOLAR LEADS RECORD THE DIFFERENCE BETWEEN AN ELECTRODE AND A ZERO ELECTRODE

Every system for recording voltages consists of two electrodes whose potential difference is fed into the input of an amplifier. If the two electrodes are placed at two different places in an electric field, the system records $V_1 - V_2$. The two electrodes are called **bipolar electrodes**. If one of the electrodes is

placed in a "zero" area of the field, the potential difference recorded between the zero, or "indifferent" electrode and the "different" electrode is called a **unipolar potential**. The zero area surrounding an electric dipole lies along the line perpendicular to the dipole moment, as shown in Figure 1.4.A1.2. Using unipolar electrodes for recording ECGs requires knowing where to place the zero electrode. In practice, we never know the area of zero potential, and its location changes as the heart's electric dipole changes during the cardiac cycle. Using greatly simplified assumptions, we can construct a zero electrode by combining the three Einthoven leads through resistances $>5\ \text{k}\Omega$, as shown in Figure 5.6.9. The connection produces a **central terminal (CT)**. Its definition in this manner is equivalent to Kirchhoff's voltage law. By adding a large resistance, the effect of small differences in the output resistances for each of the three leads is minimized.

AUGMENTED UNIPOLAR LIMB LEADS USE COMBINATION OF ONLY TWO ELECTRODES FOR THE INDIFFERENT ELECTRODE

The three standard limb leads have magnitudes defined by:

$$\begin{aligned} \text{I} &= V_{\text{LA}} - V_{\text{RA}} \\ \text{II} &= V_{\text{LL}} - V_{\text{RA}} \\ \text{III} &= V_{\text{LL}} - V_{\text{LA}} \end{aligned} \qquad [5.6.4]$$

where V is the voltage at an electrode, LL signifies the left leg electrode, RA is the right arm, and LA is the left arm. The **augmented limb leads** use each of the three Einthoven electrode positions but combine each with the average of the opposite two. Their magnitude is given as:

$$\begin{aligned} aVR &= V_{\text{RA}} - \frac{[V_{\text{LA}} + V_{\text{LL}}]}{2} \\ aVL &= V_{\text{LA}} - \frac{[V_{\text{RA}} + V_{\text{LL}}]}{2} \\ aVF &= V_{\text{LL}} - \frac{[V_{\text{RA}} + V_{\text{LA}}]}{2} \end{aligned} \qquad [5.6.5]$$

where aVR stands for the augmented voltage for the right arm. In an ideal field where the heart sits at the center of an equilateral triangle, the average of two electrodes is the voltage found midway between them. Thus, voltages measured using these augmented limb leads correspond to the projection of the cardiac dipole onto the lead axes shown in Figure 5.6.10, oriented at 30°, 90°, and 150°.

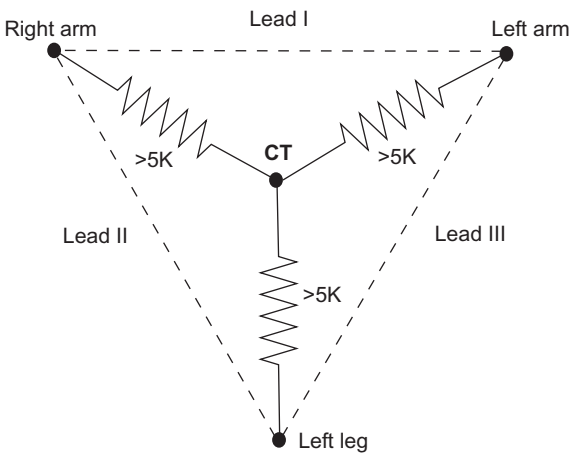

FIGURE 5.6.9 Construction of a zero terminal. Under simplified assumptions, the voltage drop around a loop is zero. Under this assumption, connection of all three leads to a CT should produce a zero reference point. The 5 kΩ resistances are added to balance small output resistances at each electrode.

THE EINTHOVEN TRIANGLE IS ONLY APPROXIMATELY VALID

Einthoven's triangle involves several assumptions. These are as follows:

1. The heart's electrical activity can be represented as a single electric dipole.
2. The heart is small compared to the field so that the heart can be considered to be located as a point in its center.
3. The thorax is a homogeneous conductor.
4. The thorax is a sphere.

The individual heart cells are tiny and may be regarded as an electric dipole. It is not obvious that the superpositioning of all simultaneously generated fields produced by the many muscle fibers can be regarded as being produced by a single dipole. This assumption is

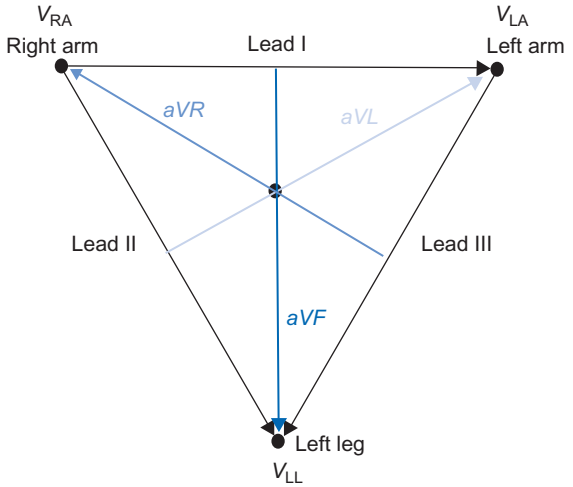

FIGURE 5.6.10 Orientation of the augmented limb leads. The voltage is measured between one of the Einthoven electrodes (right arm for aVR, left arm for aVL, and left leg for aVF) and the average of the two opposite leads. This average approximates the voltage at a position midway between the two electrodes. Thus, the voltages on the augmented limb leads are equivalent to the projection of the cardiac dipole onto the three lead axes as shown.

closely related to the second that the heart is small compared to the field so that it can be considered to be a point located in its center.

There is no question that the leads are not truly equidistant and that the thorax is not a homogeneous conductor. Each individual has somewhat different distances and different distributions of conductive media. A strict analysis of the projection would need to account for the exact shape and conductivity within the thorax. If the distribution of conductivity were known in detail, it is theoretically possible to predict the surface voltage for every electrical dipole of the heart. However, such a distribution is not known for any individual.

Despite the fact that Einthoven's triangle is strictly invalid, a large base of clinically relevant information has been assembled using its assumptions and diagnosis is possible within that frame of experience. In a sense, then, correcting for the errors in the concept amounts to a redefinition of the normal and doesn't produce clinical benefits worth the effort. A number of other electrode placement systems have been tried and evaluated.

THE CARDIAC CYCLE, REVISITED

Figure 5.4.7 shows the mechanical events that occur during the cardiac cycle, but it does not include the electrical events. Figure 5.6.11 shows these mechanical events along with the electrical events, as recorded by the ECG. Note in this figure that the electrical events precede the mechanical events. The mechanical events are caused by contraction of the heart muscle, and the electrical activation of the muscle triggers this contraction. The electrical event is not the same as contraction in the same sense that the electrical activation of a skeletal muscle is not the same as its contraction. The events that connect electrical excitation to contraction comprise excitation–contraction coupling. This topic will be covered in the next chapter.

Clinical Applications: Arrhythmias

Irregular rhythms of the heart vary from the benign and normal to the pathological but harmless palpitation to serious and life-threatening disorders. They can be diagnosed through the use of the ECG and are broadly classified as follows.

- **Sinus arrhythmia:** the SA node is the pacemaker and increased firing frequency produces **sinus tachycardia**, whereas decreased firing frequency causes **sinus bradycardia**. Normal heart rate varies with respiration, increasing during inspiration, and decreasing during expiration.
- **AV conduction blocks:** the excitation initiated by the SA is passed on to the AV node, which then relays it via the bundle of His and then the left and right bundle branches. A variety of ailments can interfere with the conduction here. **First degree heart block** shows a lengthening of the PR interval due to slowing of conduction from the AV node. A PR interval greater than 0.2 s is abnormal. **Second degree heart block** occurs when P waves intermittently fail to pass from the atria to the ventricles. Often the ratio between the number of P waves and QRS complexes is the ratio of two small integers, such as 2:1, 3:1, or 3:2. **Third degree heart block** occurs when no P waves are conducted through the AV node. In this case the ventricles initiate their own rhythm, typically at much slower rates. Thus, in first degree block the PR interval is extended; in second degree block conduction sometimes fails; in third degree block it fails all the time.
- **Premature depolarization:** abnormal myocytes occasionally can spontaneously reach threshold and begin an action potential before the normal excitation arrives from the SA node. Such an event can trigger a premature beat called an **ectopic beat** or **extrasystole**. The region that originates the premature beat is called an **ectopic focus**. An ectopic focus in the ventricles typically causes a broad QRS complex and a poorly coordinated contraction that fails to eject blood because the excitation is not properly distributed through the His–Purkinje conducting fibers. When the normal excitation resulting from the SA node appears after an extrasystole, the ventricles are refractory and no contraction results. This results in a long delay between the extrasystole and the next heart beat. The delay is called the **compensatory pause**.
- **Reentry or circus arrhythmia:** abnormal conduction pathways can develop that cause excitation to spread in a circuit. Cardiomyocytes that have just contracted and emerge from their refractory state are stimulated once again by the return of the excitation. This process is called re-entry. Re-entry in the atria or AV node can lead to tachycardia. This often begins and ends abruptly and is called **paroxysmal supraventricular tachycardia**, or **PSVT**, meaning that it is episodic (paroxysmal), originates from tissues above the ventricles (supraventricular), and causes a rapid heart rate (tachycardia). The QRS complexes are normal because excitation in the ventricles follows the normal pathways.
- **Fibrillation:** Fibrillation is an uncoordinated, repetitive excitation of the heart muscle that causes a writhing motion of the muscle but no effective ejection of blood. It probably results from multiple reentry circuits. **Atrial fibrillation** is not immediately life-threatening because atrial contraction is not essential to ventricular filling. However, regions of stagnant blood can form in the atria, predisposing to thrombus formation. The clot can dislodge and travel on to clog up part of the pulmonary or systemic vascular beds. **Ventricular fibrillation** is usually fatal unless the rhythm reverts to normal, which rarely occurs unaided. It causes loss of consciousness within seconds due to loss of cerebral perfusion and death ensues within minutes unless resuscitation efforts maintain perfusion.

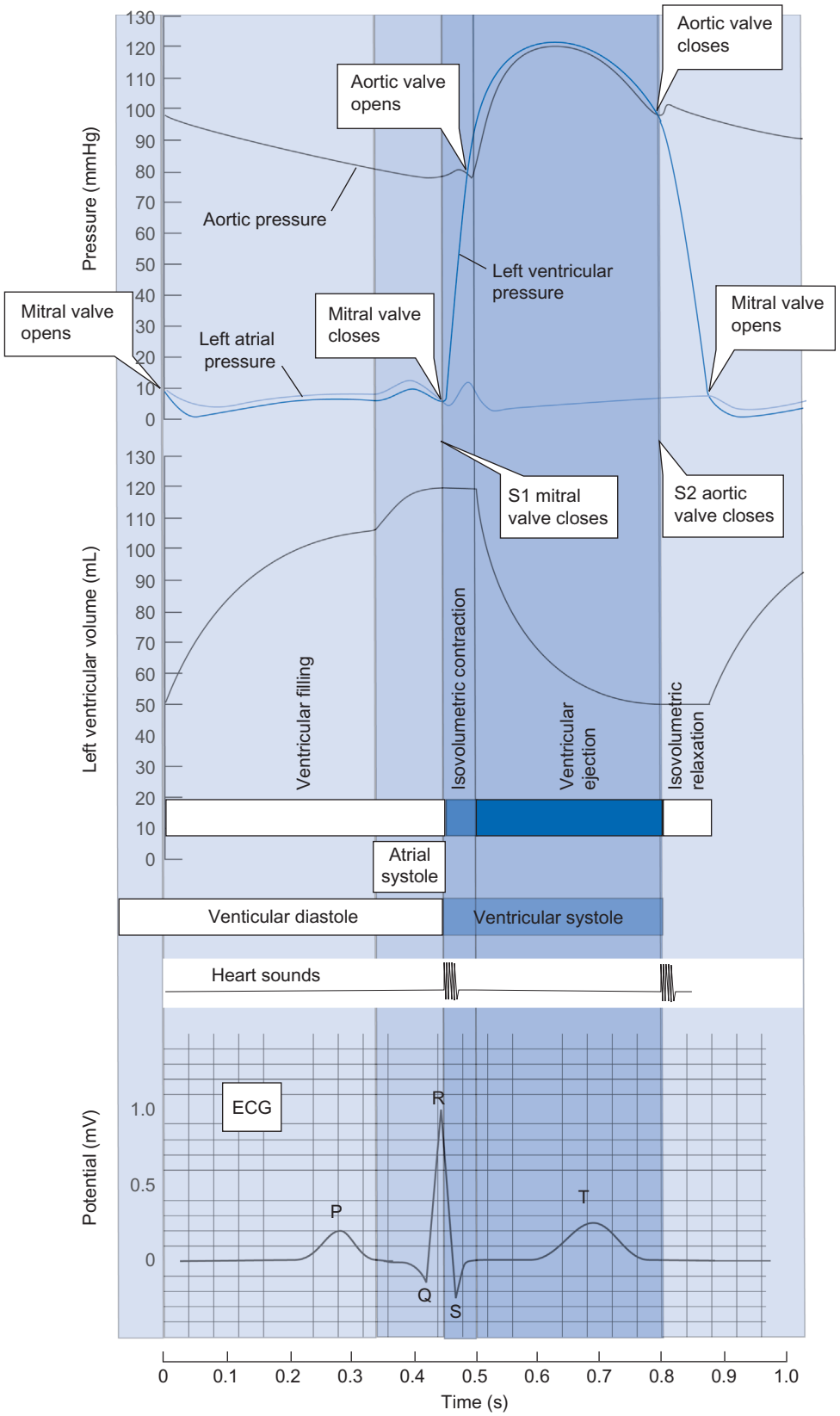

FIGURE 5.6.11 Mechanical and electrical events in the cardiac cycle.

SUMMARY

During diastole, all of the heart cells are polarized. Cells in the SA node spontaneously depolarize first and spread their depolarization through the atria. During the spread of excitation, part of the atria is depolarized and part is not, creating a cardiac electric dipole. This dipole can be detected by surface electrodes and its magnitude is equal to the projection of the heart's dipole moment, a vector, onto the axis connecting the bipolar electrodes. Einthoven invented a recording system that uses three electrodes: one on the right arm, one on the left arm, and a third on the left leg. The three leads are *defined* as: lead $\mathrm{I} = V_{\mathrm{LA}} - V_{\mathrm{RA}}$; lead $\mathrm{III} = V_{\mathrm{LL}} - V_{\mathrm{LA}}$; lead $\mathrm{II} = V_{\mathrm{LL}} - V_{\mathrm{RA}}$. The analysis is simplified by assuming that the three bipolar leads from these three electrodes form an equilateral triangle. At any time the cardiac dipole projects differently onto the three leads. Because the leads are not orthogonal, the voltages in the three leads are not independent. The sequence and the mass of tissue being depolarized cause the cardiac electric dipole to change magnitude and direction throughout activation. This causes the voltages on the three leads to vary with time. The recording of this voltage is the scalar electrocardiogram, or ECG.

The normal ECG has defined parts. Atrial depolarization produces the first part, the P wave, which precedes atrial contraction or systole. The AV node conducts the excitation through the annulus fibrosus but delays it so that the atria have enough time to fully fill the ventricles. This delay appears as a short, flat region of the ECG called the PR segment. The depolarization of the ventricles produces the QRS complex, which lasts about 80 ms. The Q wave may be negative, depending on the recording lead, because the cardiac dipole moment at that time points up and toward the right arm. The R wave has the largest amplitude and corresponds to ventricular depolarization. During the S wave the main part of the ventricles has already depolarized and only the base of the heart undergoes depolarization. During the ST segment the ventricles are depolarized fully and the cardiac dipole disappears. It reappears during ventricular repolarization to form the T wave. The T wave is upright because the last cells to depolarize, in the subepicardium, are the first to repolarize. Thus the cardiac dipole during repolarization has the same direction as that during depolarization. Atrial repolarization occurs during the QRS wave.

The angle of the largest dipole is called the electrical axis of the heart.

REVIEW QUESTIONS

1. In general terms, define the ECG. What is an electric dipole?
2. What are the three primary limb leads? How are they connected? What is the relationship betwen leads I and III and II?
3. Why do the three main leads not add as vectors? How can you reconstruct the heart vector from I and III?
4. What is the P wave? What electrical event in the heart does it correspond to? What is the PR interval? What electrical event does it correspond to?
5. What is the QRS complex? What electrical event in the heart does it correspond to? Where is atrial repolarization in the ECG? What is the ST segment? What is the T wave? Why is it upright?
6. What is the mean electrical axis of the heart? How does it change standing up vs lying down?
7. How would you create a zero terminal?