The technology and the clinical usefulness of the electrocardiogram (ECG) have continuously advanced over the past two centuries. Early demonstrations of the heart’s electrical activity during the last half of the 19th century were closely followed by direct recordings of cardiac potentials by Waller in 1887. Invention of the string galvanometer by Einthoven in 1901 provided a direct method for registering electrical activity of the heart. By 1910, use of the string galvanometer had emerged from the research laboratory into the clinic. Subsequently, the ECG became the first and most common bioelectric signal to be computer-processed and the most commonly used cardiac diagnostic signal. By 1910, use of the string galvanometer had emerged from the research laboratory into the clinic. Subsequently, the ECG became the first and most common bioelectric signal to be computer-processed and the most commonly used cardiac diagnostic signal.

Recent advances have extended the importance of the ECG. It is a vital test for determining the presence and severity of acute myocardial ischemia, localizing sites of origin and pathways of tachyarrhythmias, assessing therapeutic options for patients with heart failure, and identifying and evaluating patients with genetic diseases who are prone to arrhythmias. Achievements in physiology and technology, as discussed later in this chapter, have expanded the possibilities of extracting more information about the heart’s electrical activity from the ECG that will extend these clinical applications further.

It is the goal of this chapter to review the physiologic bases for electrocardiographic patterns in health and in disease, outline the criteria for the most common electrocardiographic diagnoses in adults, describe critical aspects of the clinical application of the ECG, and suggest future opportunities for the clinical practice of electrocardiography.

**Fundamental Principles**

The ECG is the final outcome of a complex series of physiologic and technologic processes. First, transmembrane ionic currents are generated by ion fluxes across cell membranes and between adjacent cells. These currents are synchronized by cardiac activation and recovery sequences to generate a cardiac electrical field in and around the heart that varies with time during the cardiac cycle. This electrical field passes through numerous other structures, including the lungs, blood, and skeletal muscle, that perturb the cardiac electrical field.

The currents reaching the skin are then detected by electrodes placed in specific locations on the extremities and torso that are configured to produce leads. The outputs of these leads are amplified, filtered, and displayed by a variety of devices to produce an electrocardiographic recording. In computerized systems, these signals are digitized, stored, and processed by pattern recognition software. Diagnostic criteria are then applied, either manually or with the aid of a computer, to produce an interpretation.

**Genesis of Cardiac Electrical Fields**

*Ionic Currents and Cardiac Electrical Field Generation During Activation.* Transmembrane ionic currents (see Chap. 35) are ultimately responsible for the potentials that are recorded as an ECG. Currents may be modeled as being carried by positively charged or negatively charged ions. A positive current moving in one direction is equivalent to a negative current of equal strength moving in the opposite direction. Through a purely arbitrary choice, electrophysiological currents are considered to be the movement of positive charge.

The process of generating the cardiac electrical field during activation is illustrated in Figure 13-1. A single cardiac fiber, 20 mm in length, is activated by a stimulus applied to its left-most margin (see Fig. 13-1A). Transmembrane potentials (V_m) are recorded as the difference between intracellular and extracellular potentials (Φ_in and Φ_out, respectively). Figure 13-1B plots V_m along the length of the fiber at the instant (t_0) at which activation has reached the point designated as X_0. As each site is activated, it undergoes depolarization, and the polarity of the transmembrane potential converts from negative to positive, as represented in the typical cardiac action potential. Thus, sites to the left of the point X_0 that have already undergone excitation have positive transmembrane potentials (i.e., the inside of the cell is positive relative to the outside of the cell), whereas those to the right of X_0 that remain in a resting state have negative transmembrane potentials. Near the site undergoing activation (site X_0), the potentials reverse polarity over a short distance.

Figure 13-1C displays the direction and magnitude of transmembrane currents (I_m) along the fiber at the instant (t_0) at which excitation has reached site X_0. Current flow is inwardly directed in fiber regions that have just undergone activation (i.e., to the left of point X_0) and outwardly directed in neighboring zones still at rest (i.e., to the right of X_0). Sites of outward current flow are current sources and those with inward current flow are current sinks. As depicted in the figure, current flow is most intense in each direction near the site of activation, X_0.

Because the border between inwardly and outwardly directed currents is relatively sharp, these currents may be visualized as if they were limited to the sites of maximal current flow, as depicted in Figure 13-1D, and separated by a distance, d, that is usually 1.0 mm or less. As activation proceeds along the fiber, the source-sink pair moves to the right at the speed of propagation in the fiber.

**The Cardiac Dipole.** Two point sources of equal strength but of opposite polarity located very near each other, such as the current source and current sink illustrated in Figure 13-1D, can be represented as a current dipole. Thus, activation of a fiber can be modeled as a current dipole that moves in the direction of activation.

Such a dipole is fully characterized by three parameters—strength or dipole moment, location, and orientation. In this case, the location of the dipole is the site undergoing activation (point X_0), and its orientation is in the direction of activation (i.e., from left to right along the fiber in Fig. 13-1). Dipole moment is proportional to the rate of change of intracellular potential—that is, action potential shape.

A current dipole produces a characteristic potential field with positive potentials projected ahead of it and negative potentials projected behind it. The actual potential recorded at any site within this field is directly proportional to the dipole moment, inversely proportional to the square of the distance from the dipole to the recording site, and directly proportional to the cosine of the angle between the axis of the dipole and a line drawn from the dipole to the recording site.
**SOLID ANGLE THEOREM.** One important and common method of estimating the potentials projected to some point away from an activation front is an application of the solid angle theorem. A solid angle is a geometric measure of the size of a region when viewed from a distant site. It equals the area on the surface of a sphere of unit radius constructed around an electrode that is cut by lines drawn from the recording electrode to all points around the boundary of the region of interest. This region may be a wave front, zone of infarction, or any other region in the heart.

The solid angle theorem states that the potential recorded by a remote electrode (\( \Phi \)) is defined by the following equation:

\[
\Phi = \Omega (V_{m2} - V_{m1})/K
\]

where \( \Omega \) is the solid angle, \( V_{m2} - V_{m1} \) is the potential difference across the boundary under study, and \( K \) is a constant reflecting differences in conductivity.

This equation suggests that the recorded potential equals the product of two factors. First, the solid angle reflects spatial parameters, such as the size of the boundary of the region under study and the distance from the electrode to that boundary. The potential will increase as the boundary size increases and as the distance to the electrode decreases. A second set of parameters includes nonspatial factors, such as the potential difference across the surface (i.e., \( V_{m2} - V_{m1} \)) and intracellular and extracellular conductivity (i.e., \( K \)). Nonspatial effects include, as one example, myocardial ischemia which changes transmembrane action potential shapes and alters conductivity.

The dipole model, although useful in describing cardiac fields and understanding clinical electrocardiography, has significant theoretical limitations. These limits result primarily from the inability of a single dipole to represent more than one wave front that is propagating through the heart at any one instant accurately. As will be discussed, during much of the time of ventricular excitation, more than one wave front is present. Other approaches, such as the direct estimation of epicardial potentials from body surface recordings, represent other important, albeit more complex, approaches.

**CARDIAC ELECTRICAL FIELD GENERATION DURING VENTRICULAR RECOVERY.** The cardiac electrical field during recovery (phases 1 through 3 of the action potential) is generated by forces analogous to those described during activation. However, recovery differs in several important ways from activation. First, intercellular potential differences and, hence, the directions of current flow during recovery are the opposite of those described for activation. As a cell undergoes recovery, its intracellular potential becomes progressively more negative. For two adjacent cells, the intracellular potential of the cell whose recovery has progressed further is more negative than that of the adjacent, less recovered cell. Intracellular currents then flow from the less recovered toward the more recovered cell. An equivalent dipole can then be constructed for recovery, just as for activation. Its orientation points from less to more recovered cell. An equivalent dipole can then be constructed for recovery, just as for activation. Its orientation points from less to more recovered cell. This moment, or strength, of the recovery dipole also differs from that of the activation dipole. As noted, the strength of the activation dipole is proportional to the rate of change in transmembrane potential. Rates of change in potential during the recovery phases of the action potential are considerably slower than during activation, so that the dipole moment at any one instant during recovery is less than during activation.

A third difference between activation and recovery is the rate of movement of the activation and recovery dipoles. Activation is rapid (as fast as 1 msec in duration) and occurs over only a small distance along the fiber. Recovery, in contrast, lasts 100 msec or longer and occurs simultaneously over extensive portions of the fiber.

These features result in characteristic electrocardiographic differences between activation and recovery patterns. All other factors being equal (an assumption that is often not true, as described later), electrocardiographic waveforms generated during recovery of a linear fiber with uniform recovery properties may be expected to be of opposite polarity, lower amplitude, and longer duration than those generated by activation. As will be described, these features are explicitly demonstrated in the clinical ECG.

**ROLE OF TRANSMISSION FACTORS.** These activation and recovery fields exist within a complex three-dimensional physical environment (the volume conductor) that modifies the cardiac electrical field in significant ways. The contents of the volume conductor are called transmission factors to emphasize their effects on transmission of the cardiac electrical field throughout the body. They may be grouped into four broad categories—cellular factors, cardiac factors, extracardiac factors, and physical factors.

---

**FIGURE 13-1** Example of potentials and currents generated by the activation of a single (e.g., ventricular) cardiac fiber. **A,** Intracellular (\( \Phi_i \)) and extracellular (\( \Phi_e \)) potentials are recorded with a voltmeter (\( V_m \)) from a fiber 20 mm in length. The fiber is stimulated at site \( X = 0 \) mm, and propagation proceeds from left to right. **B,** Plot of transmembrane potential (\( V_m \)) at the instant in time at which activation reaches point \( X_0 \) as a function of the length of the fiber. Positive potentials are recorded from activated tissue to the left of site \( X_0 \), and negative potentials are recorded from not yet excited areas to the right of site \( X_0 \). **C,** Membrane current (\( I_m \)) flows along the length of the fiber at time \( t_0 \). The outward current is the depolarizing current that propagates ahead of activation site \( X_0 \), whereas an inward one flows behind site \( X_0 \). **D,** Representation of the sites of peak inward and outward current flow as two point sources, a sink (at the site of peak inward current flow) and a source (at the site of peak outward current flow) separated by distance \( d \). The dipole produced by the source-sink pair is represented by the arrow. (Modified from Barl RC. Genesis of the electrocardiogram. In MacFarlane PW, Lawrie TDV (eds). Comprehensive Electrocardiography. New York, Pergamon Press, 1989.)
Cellular factors determine the intensity of current fluxes that result from local transmembrane potential gradients. These include intracellular and extracellular resistances and the concentrations of relevant ions, especially the sodium ion. Lower ion concentrations, for example, reduce the intensity of current flow and reduce extracellular potentials.

Cardiac factors affect the relationship of one cardiac cell to another. The following are two major factors: (1) anisotropy, the property of cardiac tissue that results in greater current flow and more rapid propagation along the length of a fiber than across its width; and (2) the presence of connective tissue between cardiac fibers that disrupts effective electrical coupling of adjacent fibers. Recording electrodes oriented along the long axis of a cardiac fiber register higher potentials than electrodes oriented perpendicular to the long axis. Waveforms recorded from fibers with little or no intervening connective tissue are narrow in width and smooth in contour, whereas those recorded from tissues with abnormal fibrosis are prolonged, with prominent notching.

Extracardiac factors encompass all the tissues and structures that lie between the activation region and the body surface, including the ventricular walls, intracardiac blood, lungs, skeletal muscle, subcutaneous fat, and skin. These tissues alter the cardiac field because of differences in the electrical resistivity of adjacent tissues—that is, the presence of electrical inhomogeneities within the torso. For example, intracardiac blood has much lower resistivity (162 Ω cm) than the lungs (2150 Ω cm). When the cardiac field encounters the boundary between two tissues with differing resistivity, the field is altered. Differences in torso inhomogeneities can have significant effects on ECG potentials, especially when the differences are exaggerated as in patients with congestive heart failure.

Other transmission factors reflect basic laws of physics. Changes in the distance between the heart and recording electrode reduce potential magnitudes in proportion to the square of the distance. A related factor is the eccentricity of the heart within the chest. The right ventricle and anteroseptal aspect of the left ventricle are located closer to the anterior chest wall than other parts of the left ventricle and atria. Therefore, electrocardiographic potentials will be higher on the anterior than on the posterior chest, and waveforms projected from the anterior left ventricle to the chest wall will be greater than those generated by posterior regions.

An additional physical factor affecting the recording of cardiac signals is cancellation. This results when two or more wave fronts that are simultaneously active during activation or repolarization have different orientations. The vectorial components of the wave fronts that are oriented in opposite directions cancel each other when viewed from remote electrode positions. The magnitude of this effect is substantial. During both the QRS and ST-T waves, as much as 90% of cardiac activity is obscured by cancellation.

As a result of all these factors, body surface potentials have an amplitude of only 1% of the amplitude of transmembrane potentials, are smoothed in detail so that surface potentials have only a general spatial relationship to the underlying cardiac events, preferentially reflect electrical activity in some cardiac regions over others, and reflect only limited amounts of total cardiac electrical activity.

### Recording Electrodes and Leads

Potentials generated by the cardiac electrical generator and modified by transmission factors are sensed by electrodes placed on the torso that are configured to form various types of leads. **Electrode Characteristics.** Electrocardiographic potentials are affected by the properties of the dermal and epidermal layers of the skin, the electrolytic paste applied to the skin, the electrode itself, and the mechanical contact between the electrode and skin. The net effect is equivalent to a complex electrical circuit that includes resistances, capacitances, and voltages produced by these different components and the interfaces between them.

**Electrocardiographic Lead Systems.** Electrodes are connected to form leads. The electrocardiographic leads are bipolar leads that record the potential difference between two electrodes. One electrode is designated as the positive input. The potential at the other, or negative, electrode is subtracted from the potential at the positive electrode to yield the bipolar potential. The actual potential at either electrode is not known, and only the difference between them is recorded.

In some cases, as described later, multiple electrodes are electrically connected together to represent the negative member of the bipolar pair. This electrode network or compound electrode is referred to as the reference electrode. The lead then records the potential difference between a single electrode serving as the positive input, the exploring electrode, and the potential in the reference electrode.

### TABLE 13-1 Location of Electrodes and Lead Connections for the Standard 12-Lead Electrocardiogram and Additional Leads

<table>
<thead>
<tr>
<th>LEAD TYPE</th>
<th>POSITIVE INPUT</th>
<th>NEGATIVE INPUT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Standard Limb Leads</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lead I</td>
<td>Left arm</td>
<td>Right arm</td>
</tr>
<tr>
<td>Lead II</td>
<td>Left leg</td>
<td>Right arm</td>
</tr>
<tr>
<td>Lead III</td>
<td>Left leg</td>
<td>Left arm</td>
</tr>
<tr>
<td><strong>Augmented Limb Leads</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aVR</td>
<td>Right arm</td>
<td>Left arm plus left leg</td>
</tr>
<tr>
<td>aVL</td>
<td>Left arm</td>
<td>Right arm plus left leg</td>
</tr>
<tr>
<td>aVF</td>
<td>Left leg</td>
<td>Left arm plus right arm</td>
</tr>
<tr>
<td><strong>Precordial Leads</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V 1</td>
<td>Right sternal margin, fourth intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 2</td>
<td>Left sternal margin, fourth intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 3</td>
<td>Midway between V 2 and V 4</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 4</td>
<td>Left midclavicular line, 5th intercostal space</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 5</td>
<td>Left anterior axillary line</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 6</td>
<td>Left midaxillary line</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 7</td>
<td>Posterior axillary line</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 8</td>
<td>Posterior scapular line</td>
<td>Wilson central terminal</td>
</tr>
<tr>
<td>V 9</td>
<td>Left border of spine</td>
<td>Wilson central terminal</td>
</tr>
</tbody>
</table>

*The right-sided precordial leads V R to V R are taken in mirror image positions on the right side of the chest.
†The exploring electrodes for leads V to V are placed at the same horizontal plane as the electrode for V.
This modified reference system was designed to produce a larger amplitude signal than if the full Wilson central terminal were used as the reference electrode. When the Wilson central terminal was used, the output was small, in part because the same electrode potential was included in both the exploring and the reference potential inputs. Eliminating this duplication results in a theoretical increase in amplitude of 50%.

The 12 leads are commonly divided into subgroups corresponding to the cardiac regions to which they are thought to be most sensitive. Various definitions of these groupings have been offered in the literature—for example, anterior lead groups have been defined as including V2 through V4 or only V2 and V3, and leads I and aVL have been described as being lateral or anterobasal. These designations are nonspecific and the recommendation of expert committees has been not to use them in electrocardiographic interpretation, except in the case of localizing myocardial infarction. We recommend that the conventional names of the leads be used to describe the distribution of electrocardiographic findings.

OTHER LEAD SYSTEMS. Other lead systems have been developed to detect diagnostically important information not recorded by the standard 12-lead ECG and to increase the efficiency of recording, transmitting, and storing an ECG. Expanded lead systems include the recording of additional right precordial leads to assess right ventricular abnormalities, such as right ventricular infarction in patients with evidence of inferior infarction, and left posterior leads (see Table 13-1) to help detect acute posterolateral infarctions.

Electrode arrays of 80 or more electrodes deployed on the anterior and posterior torso can be used to display body surface potentials as

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Electrode arrays of 80 or more electrodes deployed on the anterior and posterior torso can be used to display body surface potentials as
remain important for understanding the origins of electrocardiographic waveforms. **LEAD VECTORS AND HEART VECTORS.** A lead can be represented as a vector referred to as the lead vector. For simple two-electrode leads, such as leads I, II, and III, the lead vectors are directed from the negative electrode toward the positive one (Fig. 13-4). For the augmented limb and precordial leads, the origin of the lead vectors lies at the midpoint of the axis connecting the electrodes that make up the compound electrode. That is, for lead aVL, the vector points from the midpoint of the axis connecting the right arm and left leg electrodes toward the left arm (see Fig. 13-4, left). For each precordial lead, the lead vector points from the center of the triangle formed by the three standard limb leads to the precordial electrode site (see Fig. 13-4, right).

As described earlier, instantaneous cardiac activity can also be approximated as a single vector (the heart vector) or dipole representing the vector sum of the various active wave fronts. Its location, orientation, and intensity vary from instant to instant as cardiac activation proceeds.

Other lead systems that have had clinical usefulness include those designed to record a vectorcardiogram (VCG). The VCG depicts the orientation and strength of a single cardiac dipole or vector that best represents overall cardiac activity at each instant during the cardiac cycle. Lead systems for recording the VCG record the three orthogonal or mutually perpendicular components of the dipole moment—the horizontal ($x$), frontal ($y$), and sagittal or anteroposterior ($z$) axes. Clinical use of the VCG has waned in recent years but, as described later, vectorial principles remain important for understanding the origins of electrocardiographic waveforms.

Other arrays have sought to reduce the number of electrodes to reduce the time and mechanical complexity of a full recording, especially during emergency situations. For example, a full 12-lead ECG can be reconstructed, with high accuracy, from lead sets requiring only five electrodes. Modified lead systems are also used in ambulatory ECG recording, exercise stress testing, and bedside cardiac monitoring, as described in other chapters, to simplify application and reduce motion artifact during exercise. The waveforms they produce are significantly different than those recorded from the standard electrocardiographic sites, including diagnostically important changes in waveform intervals and amplitudes.

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As described earlier, instantaneous cardiac activity can also be approximated as a single vector (the heart vector) or dipole representing the vector sum of the various active wave fronts. Its location, orientation, and intensity vary from instant to instant as cardiac activation proceeds.

The amplitude of the potentials sensed in a lead equals the length of the projection of the heart vector on the lead vector, multiplied by the length of the lead vector:

$$V_i = (H)(\cos \tau)(L)$$

where $L$ and $H$ are the length of the lead and heart vectors, respectively, and $\tau$ is the angle between the two vectors, as illustrated in Figure 13-5.
Thus, if the projection of the heart vector on the lead vector points toward the positive pole of the lead axis, the lead will record a positive potential. If the projection is directed away from the positive pole of the lead axis, the potential will be negative. If the projection is perpendicular to the lead axis, the lead will record zero potential.

**Hexaxial Reference Frame and Electrical Axis.** The lead axes of the six frontal plane leads can be superimposed to produce the hexaxial reference system. As depicted in Figure 13-6, the six lead axes divide the frontal plane into 12 segments, each subtending 30 degrees. These axes divide the plane into 12 segments, each subtending 30 degrees. Positive ends of each axis are labeled with the name of the lead.

**Thus,** if the projection of the heart vector on the lead vector points toward the positive pole of the lead axis, the lead will record a positive potential. If the projection is directed away from the positive pole of the lead axis, the potential will be negative. If the projection is perpendicular to the lead axis, the lead will record zero potential.

**Hexaxial Reference Frame and Electrical Axis.** The lead axes of the six frontal plane leads can be superimposed to produce the hexaxial reference system. As depicted in Figure 13-6, the six lead axes divide the frontal plane into 12 segments, each subtending 30 degrees.

These concepts allow calculation of the mean electrical axis of the heart. The orientation of the mean electrical axis represents the direction of activation in an “average” cardiac fiber. This direction is determined by the properties of the cardiac conduction system and activation properties of the myocardium. Differences in the relation of cardiac to torso anatomy contribute relatively little to shifts in the axis.

The process for computing the mean electrical axis during ventricular activation in the frontal plane is illustrated in Figure 13-7. First, the mean force during activation is represented by the area under the QRS wave. Areas above the baseline are assigned positive values and those below the baseline have a negative polarity. The overall area equals the sum of the positive and the negative areas.

Second, the area in each lead (typically two are chosen) is represented as a vector oriented along the appropriate lead axis in the hexaxial reference system (see Fig. 13-6), and the mean electrical axis equals the resultant or vector sum of the two vectors. An axis directed toward the positive end of the lead axis of lead I—that is, oriented directly away from the right arm and toward the left arm—is designated as an axis of 0 degrees. Axes oriented in a clockwise direction from this zero level are assigned positive values and those oriented in a counterclockwise direction are assigned negative values.

The mean electrical axis during ventricular activation in the horizontal plane can be computed in an analogous manner by using the areas under and lead axes of the six precordial leads (see Fig. 13-4, right). A horizontal plane axis located along the lead axis of lead II—that is, directed more anteriorly has positive values.

This process can be applied to compute the mean electrical axis for other phases of cardiac activity. Thus, the mean force during atrial activation is represented by the areas under the P wave, and the mean force during ventricular recovery is represented by the areas under the ST-T wave.

**Electrocardiographic Processing and Display Systems.**

Electrocardiographic waveforms are also influenced by the characteristics of the electronic systems used to digitize, amplify, and filter the sensed signals. In computerized systems, analog signals are converted to a digital form at rates of 1000/sec (Hz) to as high as 15,000 Hz. Too low a sampling rate will miss brief signals such as notches in QRS complexes or pacemaker spikes, and will reduce the accuracy of waveform morphologies. Too fast a sampling rate may introduce artifacts, including high-frequency noise, and requires extensive digital storage capacity. In general, the sampling rate should be at least twice the frequency of the highest frequencies of interest in the signal being recorded (up to approximately 500 Hz in adults).

The standard amplifier gain for routine electrocardiography is 1000. Lower (e.g., 500 or half-standard) or higher (e.g., 2000 or double standard) gains may be used to compensate for unusually large or small signals, respectively.

Electrocardiographic amplifiers respond differently to the range of signal frequencies included in an electrophysiologic signal system, and they include filters to reduce the amplitude of specific frequency ranges of the signal intentionally. Low-pass filters reduce the distortions caused by high-frequency interference from, for example, muscle tremor and external electrical devices. High-pass filters reduce the effects of body motion or respiration.

The bandwidth of an amplifier defines the frequency range over which the amplifier accurately amplifies the input signals and is determined by specific characteristics of the amplifier and any added filters. Waveform components with frequencies outside the bandwidth of the amplifier will be artifactually reduced or increased in amplitude. For
routine electrocardiography, the standards of the American Heart Association require an overall bandwidth of 0.05 to 150 Hz for adults, with an extension to 250 Hz for children. Electrocardiographic amplifiers include a capacitor stage between the input and output; that is, they are capacitor coupled. This configuration blocks unwanted direct current (DC) potentials, such as those produced by the electrode interfaces, while permitting flow of alternating current (AC) signals, which accounts for the waveform shape. The elimination of the DC potential from the final product means that electrocardiographic potentials are not calibrated against an external reference level (e.g., a ground potential). Rather, electrocardiographic potentials are measured in relation to another portion of the waveform that serves as a baseline. The T-P segment, which begins at the end of the T wave of one cardiac cycle and ends with the onset of the P wave of the next cycle, is usually the most appropriate internal ECG baseline (e.g., for measuring ST-segment deviation).

Additional signal processing steps are included in computerized systems. Multiple cardiac cycles are recorded for each lead and are processed to form a single representative beat for each lead that is used for interpretation. This step reduces the effects of beat-to-beat variation in the waveforms.

The representative beats from all leads may then be electronically overlaid on each other to produce a single, global pattern. Electrocardiographic intervals are then measured from this single pattern. This approach has the advantage of identifying the earliest onset and latest ending of an electrocardiographic interval in all leads. The beginning or end of a waveform may appear to be isoelectric in a given lead if the electrical forces at that time are perpendicular to the lead axis. The forces will, however, be detected in other leads, so that a more accurate measurement may be made than if determined only from a recording from a single lead.

Cardiac potentials are most commonly displayed as the classic scalar ECG. Scalar recordings depict the potentials recorded from each lead as a function of time. For standard electrocardiography, amplitudes are displayed on a scale of 1 mV to 10 mm on the vertical axis and time as 400 msec/cm on the horizontal scale. Leads are generally displayed in three groups, the three standard limb leads followed by the three augmented limb leads followed by the six precordial leads.

Alternative display formats have been proposed in which the six limb leads are displayed in the sequence of the frontal plane reference frame (see page 13-6). In addition, the polarity of lead aVR is reversed. That is, waveforms are displayed in this order: lead aVL, lead I, lead aVF (reversed in polarity), lead II, lead aVF, and lead III. Proposed advantages of this system include facilitating estimation of the electrical axis by presenting the leads in the order in which they appear on the frontal plane reference frame and emphasizing the relevance of abnormalities in lead aVR by reversing its polarity. This approach has also been extended to display the inverted form of all 12 leads, producing a 24-lead ECG in which waveforms are shown at 30-degree increments in the frontal plane and as if recorded on the back and right torso in the horizontal plane.

Interpreting the Electrocardiogram

The recorded or displayed electrocardiographic tracings are, finally, compared with various diagnostic criteria to identify specific abnormalities. In some cases, the electrocardiographic criteria are derived from physiologic constructs and are the sole basis for a diagnosis with no anatomic or physiologic correlation. For example, the electrocardiographic criteria for intraventricular conduction defects (see later) are diagnostic without reference to an anatomic standard.

For other electrocardiographic diagnoses, the criteria are based on statistical correlations between anatomic or physiologic findings and electrocardiographic measurements in large populations. For example, the electrocardiographic diagnostic criteria for ventricular hypertrophy depend on correlations between various electrocardiographic patterns and anatomic measures of chamber size in large populations. As a result, many different sets of criteria have been proposed for common abnormalities (e.g., left ventricular hypertrophy). The various criteria have different predictive accuracies that are empirically determined, so that the final electrocardiographic diagnosis is not absolute but represents a statistical probability that the abnormality exists based on the presence (positive predictive accuracy) or absence (negative predictive accuracy) of a specific set of electrocardiographic findings.

Another issue related to electrocardiographic interpretation is the variability in the terminology used to describe waveform patterns. Often, several different diagnostic statements, which may contain vague terminology, may be used to describe identical or similar findings. A lexicon of preferred diagnostic statements has recently been proposed to reduce these problems.

Normal Electrocardiogram

The waveforms and intervals that make up the standard ECG are displayed in Figure 13-8, and a normal 12-lead ECG is shown in Figure 13-9. The P wave is generated by activation of the atria, the PR segment represents the duration of atrioventricular (AV) conduction, the QRS complex is produced by the activation of the two ventricles, and the ST-T wave reflects ventricular recovery (see Chap. 35).

Table 13-2 lists normal values for the various intervals and waveforms of the ECG. The range of normal values of these measurements reflects the substantial interindividual variability related to, among other factors, differences in age, gender, body habitus, heart orientation, and physiology. In addition, significant differences in electrocardiographic patterns may occur within the same person in ECGs recorded days, hours, or even minutes apart. These intradividual variations may be caused by technical issues (e.g., changes in electrode position) or the biologic effects of changes in posture, temperature, or eating, and may be sufficiently large to alter diagnostic evidence for conditions such as chamber hypertrophy.

The values shown in Table 13-2 have been typically used in clinical electrocardiography. Other normal ranges for various measures, as will be described in the sections that follow, have been suggested. These proposals are based on changes in the demographics of the population as well as differences in recording methods, especially the use of digital signals and computerized analysis systems, which have occurred over the past decades. Computerization of electrocardiographic interpretation facilitates the identification and use of different criteria for various population subgroups based on, for example, age, gender, and race, that were not previously feasible. For example, the substantial differences in the duration of the normal PR interval between genders and among different age groups suggest that a single range of normal values for all subjects may be inappropriate and could lead to over- or underdiagnosis of clinically important conditions.

Atrial Activation and the P Wave

**Atrial Activation.** Atrial activation begins with impulse generation in the atrial pacemaker complex in or near the sinoatrial (SA) node. Once the impulse leaves this pacemaker site, atrial activation begins in one or, in many cases, simultaneously in several areas of the right atrium. Propagation then proceeds rapidly along the crista terminals and moves anteriorly toward the lower portion of the right atrium.
Nor \textit{mal P wave.} Activation beginning high in the right atrium and proceeding simultaneously leftward toward the left atrium and inferiorly toward the AV node corresponds to a mean frontal plane P wave axis of approximately 60 degrees. Based on this orientation of the heart vector, normal atrial activation projects positive or upright P waves in leads II and usually in leads I, aVL, and aVF. The pattern in leads aVL and III may be upright or downward, depending on the exact orientation of the mean axis.

**NORMAL P WAVE.** These patterns of atrial activation produce the normal P wave. Activation beginning high in the right atrium and proceeding simultaneously leftward toward the left atrium and inferiorly toward the AV node corresponds to a mean frontal plane P wave axis of approximately 60 degrees. Based on this orientation of the heart vector, normal atrial activation projects positive or upright P waves in leads II and usually in leads I, aVL, and aVF. The pattern in leads aVL and III may be upright or downward, depending on the exact orientation of the mean axis.

**TABLE 13-2 Normal Values for Durations of Electrocardiographic Waves and Intervals in Adults**

<table>
<thead>
<tr>
<th>WAVE OR INTERVAL</th>
<th>DURATION (MSEC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave duration</td>
<td>&lt;120</td>
</tr>
<tr>
<td>PR interval</td>
<td>&lt;200</td>
</tr>
<tr>
<td>QRS duration</td>
<td>&lt;110-120*</td>
</tr>
<tr>
<td>QT interval (corrected)</td>
<td>≤440-450*</td>
</tr>
</tbody>
</table>

*See text for further discussion.

Interaltrial spread is more complex. In most people, the left atrium is activated first by propagation across Bachmann’s bundle, which extends from the anterior right atrium, above the fossa ovalis to the left atrium, near the right upper pulmonary vein. Other routes of interatrial spread include paths within the fossa ovalis or near the coronary sinus either alone or, more commonly, in combination with conduction in Bachmann’s bundle.$^{16,17}$

At the same time, activation spreads through the interatrial septum, beginning high on the right side and moving around the fossa ovalis to reach the top of the interventricular septum. The last area to be activated is over the inferolateral aspect of the left atrium.

Thus, right atrial activation begins before activation of the left atrium, left atrial activation continues after the end of right atrial activation, and both atria undergo activation during much of the middle of the overall atrial activation period.

**HEART RATE VARIABILITY.** Ongoing attention continues to be directed to the analysis of beat-to-beat changes in heart rate and related dynamics, termed heart rate variability (HRV), to gain insight into neuroautonomic control mechanisms (see Chaps. 36 and 94) and their perturbations with aging, disease, and drug effects. For example, relatively high-frequency (0.15 to 0.5 Hz) fluctuations mediated by the vagus nerve traffic occur phasically with heart rate increasing during inspiration and decreasing during expiration. Attenuation of this respiratory sinus arrhythmia, and related short-term heart rate variability, is a consistent marker of physiologic aging and also occurs with diabetes mellitus, congestive heart failure, and a wide range of other cardiac and noncardiac conditions that alter autonomic tone. Relatively lower frequency (0.05 to 0.15 Hz) physiologic oscillations in heart rate are associated with baroreflex activation and appear to be jointly regulated by sympathetic and parasympathetic interactions. Various complementary signal processing techniques are being developed to analyze heart rate variability and its interactions with other physiologic signals.$^{18}$ These methods include time domain statistics, frequency domain techniques based on spectral methods (Fourier and other wave decomposition), and newer computational tools.

**Atrial Repolarization.** The potentials generated by atrial repolarization are not usually seen on the surface ECG because of their low amplitude (usually less than 100 µV) and because they are superimposed on the much higher amplitude QRS complex. They may be observed as a low-amplitude wave with a polarity opposite that of the P wave (the T wave) during atrioventricular block and may have special significance in influencing ST-segment patterns during exercise testing. Deviation of the PR segment is, as described later, also an important marker of acute pericarditis and, less commonly, of atrial infarction.

**Figure 13-9** Normal electrocardiogram recorded from a 48-year-old woman. The vertical lines of the grid represent time, with lines spaced at 40-msec intervals. Horizontal lines represent voltage amplitude, with lines spaced at 0.1-mV intervals. Every fifth line in each direction is typically darkened. The heart rate is approximately 72 beats/min, the PR interval, QRS, and QT durations measure about 140, 84, and 400 msec, respectively, and the mean QRS axis is approximately +35 degrees.
Atrioventricular Node Conduction and the PR Segment

The PR segment is the usually isoelectric region beginning with the end of the P wave and ending with the onset of the QRS complex. It forms part of the PR interval, which extends from the onset of the P wave to the onset of the QRS complex. The overall PR interval is best determined from the lead with the shortest PR intervals (to avoid missing various preexcitation syndromes). The normal PR interval measures 120 to 200 msec in duration in adults.

The PR segment is the temporal bridge between atrial activation and ventricular activation. Most of the time during this segment represents slow conduction within the AV node. On exiting the AV node, the impulse rapidly traverses the bundle of His to enter the bundle branches, and it then travels through the specialized intraventricular conduction paths to activate ventricular myocardium. The segment ends when enough ventricular myocardium has been activated to initiate the recording of the QRS complex.

The PR segment appears isoelectric because the potentials generated by the conduction system structures are too small to produce detectable voltages on the body surface at the normal amplifier gains used in clinical electrocardiography. Signals from elements of the conduction system can be recorded from intracardiac recording electrodes placed against the base of the interventricular septum near the bundle of His (see Chap. 35).

Ventricular Activation and the QRS Complex

Normal ventricular activation is a complex process that is dependent on interactions between the physiology and anatomy of the specialized ventricular conducting system and the ventricular myocardium.

VENTRICULAR ACTIVATION. Ventricular activation is the product of two temporally overlapping events, endocardial activation and transmural activation.20 Endocardial activation is guided by the anatomic distribution and physiology of the His-Purkinje system. The broadly dispersed ramifications of this treelike (fractal) system and the rapid conduction within it result in the near-simultaneous activation of multiple endocardial sites and the depolarization of most of the endocardial surfaces of both ventricles within several msec.

The sequence of ventricular endocardial activation is depicted in Figure 13-10. Earliest activity begins in three sites: (1) the anterior paraseptal wall of the left ventricle; (2) the posterior paraseptal wall of the left ventricle; and (3) the center of the left side of the septum. These loci generally correspond to the sites of insertion of the branches of the left bundle branch. Septal activation begins on the left side and spreads across the septum from left to right and from apex to base. Wave fronts sweep from these initial sites of activation in anterior and inferior and then superior directions to activate the anterior and lateral walls of the left ventricle. The posterobasal areas of the left ventricle are the last to be activated.

Excitation of the right ventricle begins near the insertion point of the right bundle branch, close to the base of the anterior papillary muscle, and spreads to the free wall. The final areas to be activated are the pulmonary conus and the posterobasal areas. Thus, in both ventricles, the overall endocardial excitation pattern begins on septal surfaces and sweeps down toward the apex and around the free walls to the posterior and basal regions in an apex to base direction.

The activation fronts then move from endocardium to epicardium. Excitation of the endocardium begins at sites of Purkinje–ventricular muscle junctions and proceeds by muscle cell to muscle cell conduction in an oblique direction toward the epicardium.

NORMAL QRS COMPLEX. The sequence of endocardial and transmural activation results in the characteristic waveforms of the QRS complex.

Terminology for the QRS Complex

QRS patterns are described by the sequence of waves constituting the complex. An initial negative deflection is called the Q wave, the first positive wave is the R wave, and the first negative wave after a positive wave is the S wave. A second upright wave following an S wave is an R′ wave. Tall waves are denoted by upper case letters and smaller ones by lower case letters. A monophasic negative complex is referred to as a QS complex. Thus, for example, the overall QRS complex may be described as qRS if it consists of an initial small negative wave (the q wave) followed by a tall upright one (the R wave) and a deep negative one (an S wave). In an RSR′ complex, initial R and S waves are followed by a small positive wave (the R′ wave). In each case, the deflection must cross the baseline to be designated a discrete wave; changes in waveform direction that do not cross the baseline result in notches.

EARLY QRS PATTERNS. The complex pattern of activation described earlier can be simplified into two vectors, the first representing septal activation and the second representing left ventricular free wall activation (Fig. 13-11). Initial activation of the interventricular septum corresponds to a vector oriented from left to right in the frontal plane and anteriorly in the horizontal plane, corresponding to the anatomic position of the septum within the chest. This vector produces an initial positive wave in leads with axes directed to the right (lead aVR) or anteriorly (lead V1). Leads with axes directed to the left (leads I, aVL, V5, and V6) will register initial negative waves (septal q waves). These initial forces are normally of low amplitude and are brief (less than 30 msec). The absence of these septal q waves, with QS complexes in the right precordial leads or as initial R waves in leads I and V5 or V6 is usually a normal variant and not associated with any cardiac disease.21

MID AND LATE QRS PATTERNS. Subsequent parts of the QRS complex reflect activation of the free walls of the left and right ventricles. Because right ventricular muscle mass is considerably smaller than that of the left ventricle, it contributes little to normal QRS complexes recorded in the standard ECG. Thus, the normal QRS can be considered to represent only septal and left ventricular activity; with relatively little meaningful oversimplification.

The complex interrelationships among cardiac position, conduction system function, and ventricular geometry22 result in a wide range of normal QRS patterns in the six limb leads. The QRS pattern in leads II, III, and aVF may be predominantly upright with QR complexes or these leads may show Rs or Rs patterns. Lead I may record an isoelectric Rs pattern or a predominantly upright qr pattern.
The wide range of QRS patterns, especially in the inferior leads, can be interpreted by referring to the hexaxial reference system in Figure 13-6. The normal mean QRS axis in adults lies between −30 degrees and +90 degrees. If the mean axis is near 90 degrees, the QRS complex in leads II, III, and aVF will be predominantly upright, with Q complexes; lead I will record an isoelectric RS pattern because the heart vector lies perpendicular to the lead axis. If the mean axis is nearer 0 degrees, the patterns will be reversed; leads I and aVL will register a predominantly upright qR pattern, and leads II, III, and aVF will show Rs or RS patterns.

Mean QRS axes more positive than +90 degrees (usually with an Rs pattern in lead I) represent right axis deviation (RAD), with axes between +90 and +120 degrees referred to as moderate RAD and axes between +120 and +180 degrees referred to as marked RAD. Axes more negative than −30 degrees (with an rS pattern in lead II) represent left axis deviation (LAD), with axes between −30 and −45 degrees called moderate LAD and those between −45 and −90 degrees called marked LAD. Mean QRS axes of approximately −80 to −90 degrees are sometimes referred to as superior axis deviation, and have been reported in cases of severe chronic obstructive lung disease.

Mean axes lying between −90 degrees and −180 degrees (or, equivalently, between +180 degrees and +270 degrees) are referred to as extreme axis deviations or, alternatively, as right superior axis deviations. The term indeterminate axis is applied when all six extremity leads show biphasic (QR or RS) patterns, indicating a mean axis that is perpendicular to the frontal plane. This finding can occur as a normal variant or may be seen in a variety of pathologic conditions.

Normal QRS patterns in the precordial leads follow an orderly progression from right (V1) to left (V6). In leads V1 and V2, left ventricular free wall activation generates S waves following the initial r waves generated by septal activation (an rS pattern). These S waves are produced by the spread of activation in the free wall to the left and posteriorly, with generation of a heart vector directed away from the axes of these leads.

Patterns in the midprecordial leads V3 and V4 reflect the activation front in the ventricular free wall, first approaching the exploring electrode, and followed by its moving leftward and posteriorly to more remote regions of the left ventricle and away from the exploring electrode. In leads V1 and V2, this generates an R or r wave as it moves toward the electrode, followed by an S wave as it moves away from the electrode to produce Rs or R complex patterns. As the exploring electrode moves laterally to the left, the R wave becomes more dominant and the S wave becomes smaller (or is totally lost) because of the greater time interval during which the activation front moves toward the positive end of the electrode. In the leftmost leads (i.e., leads V3 and V4), the pattern also includes the septal q wave to produce a qRs or QR pattern.

Thus, in the precordial leads, the QRS complex is usually characterized by a consistent progression from an rS complex in the right precordial leads to a QR pattern in the left precordial leads. The site during this transition at which the pattern changes from an rS to an Rs configuration—the lead in which an isoelectric RS pattern is present—is defined as the transition zone and normally occurs in leads V3 or V4. An example of a normal precordial QRS pattern is shown in Figure 13-9. An altered location of the transition zone may occur for a variety of reasons; transition zones that are shifted to the right to lead V4 are early transitions, and those that are shifted leftward to V3 or V2 are delayed transitions.

Normal variabilities in QRS amplitudes, axes, and duration QRS are related to demographic and physiologic factors. QRS amplitudes are greater in men than in women, with higher amplitudes in African Americans than in those of other races. In addition, the location of the mitral papillary muscles in relation to the septum affects duration and frontal plane axis,22 and left ventricular mass (within the normal range) affects both QRS amplitude and duration.23

QRS DURATION. The upper normal value for QRS duration is traditionally reported as less than 120 msec (and often as less than 110 msec) measured in the lead with the widest QRS duration. Women, on average, have somewhat shorter QRS durations than men (by ~5 to 8 msec).

**The Intrinsicoid Deflection.** An additional feature of the QRS complex is the intrinsicoid deflection. An electrode overlying the ventricular free wall will record a rising R wave as transmural activation proceeds toward it. Once the activation front reaches the epicardium, the full thickness of the wall under the electrode will be in an active state. At that moment, the electrode will register negative potentials as activation proceeds in remote or noncardiac areas. The sudden reversal of potential, with a sharp downslope, is the intrinsicoid deflection and approximates the timing of activation of the epicardium under the electrode. The term ventricular activation time (VAT) is sometimes used with reference to the surface ECG.

**Ventricular Recovery and the ST-T Wave.**

**SEQUENCE OF VENTRICULAR RECOVERY.** The ST-T wave of the electrocardiogram reflects activity during the plateau phase (the ST segment) and the later repolarization phases (the T wave) of the cardiac action potential.

Ventricular repolarization, like activation, occurs in a characteristic geometric pattern. Differences in recovery timing occur both across the ventricular wall and between regions of the left ventricle.24 Transmural differences in recovery times are the net result of two effects, differences in action potential duration across the ventricular wall and the relatively slow spread of activation across the wall. As activation moves from endocardium to epicardium, sites further away from the endocardium are activated later in time. However, action potential durations are longest near the endocardium and shortest near the epicardium. Differences in action potential duration are greater than differences in activation times, so that recovery is completed near the epicardium before it is completed near the endocardium. For example, one endocardial site may be excited 10 msec earlier than the overlying epicardium (i.e., transmural activation may require 10 msec), and the action potential duration at the endocardium may be 22 msec longer than on the epicardium. As a result, recovery will be completed 12 msec earlier in the epicardium than in the endocardium.

The resulting recovery dipole will then be directed away from sites of less recovery (the endocardium) toward sites of greater recovery (near the epicardium). The orientation of this dipole is in the same direction as transmural activation dipole, as described earlier. The result, in normal persons, is relatively concordant QRS and ST-T wave vectorial patterns.

The identification of midwall cells (M cells; see Chap. 35) has suggested that the origins of the ST-T wave may be more complex. These cells have action potentials that are longer than those of endocardial or epicardial cells.25 The ST-T wave begins when epicardial cells begin to recover ahead of both M and endocardial cells, with current flowing from midmyocardial and endocardial regions toward the epicardium. The result is the rising phase of the T wave. These currents reach maximal intensity when the epicardium is fully repolarized at the peak of the T wave. As endocardial regions begin to repolarize, a second set of currents flowing from M cells to endocardial cells is generated. This current, flowing in the opposite direction to the first, initiates the descending limb of the T wave. These relationships suggest that the interval between the peak and the end of the T wave (the TpTe interval) may be a measure of transmural dispersion of recovery properties and may be related to arrhythmogenesis.
Similarly, regional differences in recovery properties exist. Action potentials are shortest in the anterior-basal region and longest in the posterior-apical region of the left ventricle. Under normal conditions, it is the transmural gradients that predominantly determine ST patterns. These regional differences, however, account for the discordant ST-T patterns observed with intraventricular conduction defects, as will be described.

NORMAL ST-T WAVE. The normal ST-T wave begins as a low-amplitude, slowly changing wave (the ST segment) that gradually evolves into a larger wave, the T wave. The onset of the ST’ wave is the junction, or J point, and it is normally at or near the isoelectric baseline of the ECG (see Fig. 13-9). The level of the ST segment is generally measured at the J point or, in some applications such as exercise testing, 40 or 80 msec after the J point.

The polarity of the ST wave is generally the same as the net polarity of the preceding QRS complex. Thus, T waves are usually upright in leads I, II, aVL, and aVF and the lateral precordial leads. They are negative in lead aVR and variable in leads III, V1, and V2.

The amplitude of the normal J point and ST segment varies with race, gender, and age. It is typically highest in lead V1, and it is higher in young men than in young women and higher in African Americans than in whites. Recent recommendations for the upper limits of normal ST segment elevation in leads V1 and V2 are 0.2 mV for men older than 40 years, 0.25 mV for men younger than 40 years, and 0.15 mV for women. In other leads, the recommended upper limit is 0.1 mV.

J WAVE. A J wave is a dome or hump-shaped wave that appears at the end of the J wave. It is typically measured from a composite of all leads, with the interval simultaneous in every lead, the QT interval duration will vary from lead to lead by as much as 50 to 65 msec. When the interval is to be measured from a single lead, the lead in which the interval is the longest, most commonly lead V1 or V6, and in which a prominent U wave is absent should be used. In automated electrocardiographic systems, the interval is typically measured from a composite of all leads, with the interval beginning with the earliest onset of the QRS in any lead and ending with the latest onset of the T wave in any lead.

The duration of the QT interval varies widely in the general population. This is a result of the substantial variation in measurements between repeated recordings in the same person (explaining as much as one third of the variation), as well as interindividual variations in various biologic, pharmacologic, metabolic, and genetic factors. The normal QT interval decreases as heart rate increases, as does the duration of the normal ventricular action potential duration and refractoriness. Thus, the normal range for the QT interval is rate-dependent. This variability is related in part to genotypic differences affecting heart rate as well as the structure and function of repolarization channels. Rate adaptation occurs in two phases, with a rapid phase that changes the QT interval within 30 seconds of a rate change and a slower one that responds over several minutes.

Numerous formulas have been proposed to correct the measured QT interval for this rate effect. A commonly used formula was described in 1920. The result is a corrected QT interval, or QTc, defined by the following equation:

\[
QT_c = \frac{QT}{\sqrt{RR}}
\]

where the QT and RR intervals are measured in seconds. The normal QTc is generally accepted to be less than or equal to 440 and is slightly longer, on average, in women younger than 40 years. Others have suggested that the upper limit be set at 450 or even 460.

The Bazett formula has limited accuracy in predicting the effects of heart rate on the QT interval. Large data base studies have, for example, shown that the QTc interval based on the Bazett formula remains significantly affected by heart rate and that as many as 30% of normal ECGs would be diagnosed as having a prolonged QT interval when this formula is used. Several linear models have been proposed; one formula that has been shown to be relatively insensitive to heart rate is

\[
QT_c = QT + 1.75 (HR - 60)
\]

where HR is the heart rate and the intervals are measured in milliseconds.

QT DISPERSION. As noted, the QT interval varies from lead to lead. In normal persons, the QT interval varies by up to 65 msec between leads, and is typically longest in leads V1 and V6. This range of intervals, referred to as QT dispersion, has been related to electrical instability and the risk of ventricular arrhythmogenesis, as described further below, although its practical clinical usefulness remains limited.

QRST ANGLE. The concordance between the orientation of the normal QRS complex and the normal ST-T wave described earlier can be expressed vectorially. An angle can be visualized in three-dimensional space between the vector representing the mean QRS force and the vector representing the mean ST-T force. This angle is the spatial QTST angle. The angle between the two vectors in the frontal plane represents a reasonable simplification and is normally less than 60 degrees and usually less than 30 degrees. Abnormalities of the QTST angle reflect abnormal relationships between the properties of activation and recovery.

VENTRICULAR GRADIENT. If the two vectors representing mean activation and mean recovery forces are added, a third vector known as the ventricular gradient is created. This vector represents the net area under the QTST complex. The concept of the ventricular gradient was originally developed to assess the variability that exists in regional repolarization properties; the greater these differences, the larger will be the ventricular gradient. In addition, because changes in activation patterns produced, for example, by bundle branch block cause corresponding changes in recovery patterns (see later), no change in the ventricular gradient typically results. The ventricular gradient should thus allow a measure of regional recovery properties that is independent of the activation pattern. This measurement has possible relevance to the genesis of reentrant arrhythmias that may be caused, in part, by abnormal regional variations in refractory periods.

Normal Variants

These descriptions of the waveforms of the normal ECG represent patterns most often observed in normal adults. Numerous variations occur in subjects without heart disease. These variations are important to recognize because they may be mistaken for significant

*The QTc, traditionally reported in units of seconds or msec: However, the units of the QTc will vary with the formula used for the rate correction. Based on the Bazett formula, for example, it is a ratio of seconds to the square root of seconds, which is equivalent to the square root of seconds. If the denominator of the Bazett formula is considered as unitless, then the QTc, by this formula will have traditional units of seconds or msec.
Abnormal Electrocardiogram*

Atrial Abnormalities

Various pathophysiologic events alter the normal sequence of atrial activation to produce abnormal P wave patterns. Three general categories of P wave changes are described here—abnormal patterns of activation, left atrial abnormalities, and right atrial abnormalities.

ABNORMAL ATRIAL ACTIVATION AND CONDUCTION. Small shifts in the site of initial activation within the SA node or away from the SA node to other ectopic sites within the atria can lead to major changes in the pattern of atrial activation and, hence, in the morphology of P waves. These shifts can occur as escape rhythms if the normal SA nodal pacemaker fails or as accelerated ectopic rhythms if the automaticity of an ectopic site is enhanced (see Chap. 39).

P wave patterns can suggest the site of impulse formation and the path of subsequent activation based on simple vectorial principles. For example, a negative P wave in lead I suggests that the origin of activation is in the left atrium. Inverted P waves in the inferior leads generally correspond to a posterior atrial site. However, the correlations of P wave patterns with location of origin are highly variable. Because of this, these electrocardiographic patterns may as a group, be best referred to as atrial ectopic rhythms.

Conduction delays within the atria alter the duration and pattern of P waves. When conduction from the right to the left atrium within Bachmann's bundle is delayed, P wave duration is prolonged beyond 120 msec and P waves appear to have two humps in lead II (often referred to as P mitrale; see Chap. 66). With more advanced block or when interatrial conduction is predominantly through the coronary sinus, the sinus node impulses reach the left atrium only after passing inferiorly to near the AV junction and then superiorly through the left atrium. In this case, P waves are wide and biphasic (an initial positive wave followed by a negative deflection) in the inferior leads.

LEFT ATRIAL ABNORMALITY. Anatomic or functional abnormalities of the left atrium that alter the P waves in the clinical ECG include atrial dilation, atrial muscular hypertrophy, elevated intra-atrial pressures, and delayed conduction. Because these abnormalities commonly coexist and can produce the same electrocardiographic effects, the resulting electrocardiographic patterns may best be referred to as left atrial abnormality (LAA) rather than in terms suggesting a particular pathologic basis.

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*All the diagnostic criteria that will be presented here apply only to adults.
Diagnostic Criteria

The most commonly used criteria for diagnosing left atrial abnormality are listed in Table 13-3 and illustrated in Figures 13-14 and 13-15.

**MECHANISMS FOR ELECTROCARDIOGRAPHIC ABNORMALITIES.** Increases in left atrial mass or chamber size cause increases in P wave amplitudes and durations. Because the left atrium is generally activated relatively late during P wave inscription, the changes produce prolonged P wave durations and increased P terminal forces in the right precordial leads.

**DIAGNOSTIC ACCURACY.** Comparisons of the various electrocardiographic abnormalities to echocardiographic criteria for left atrial enlargement have demonstrated the limited sensitivity and high specificity for standard electrocardiographic criteria. Based on two-dimensional echocardiographic standards, the classic P wave patterns of LAA have sensitivities of only 12% to 70% and specificities of over 90% for detecting enlarged left atria. In general, the low overall predictive accuracy of these criteria render them of limited clinical value for assessing atrial size.

**CLINICAL SIGNIFICANCE.** The electrocardiographic findings of left atrial abnormality are associated with more severe left ventricular dysfunction in patients with ischemic heart disease (see Chap. 54) and with more severe valve damage in patients with mitral or aortic valve disease (see Chap. 66). Patients with left atrial abnormalities also have a higher than normal incidence of paroxysmal atrial tachyarrhythmias, including atrial fibrillation.

**RIGHT ATRIAL ABNORMALITY.** The electrocardiographic features of right atrial abnormality are illustrated in Figures 13-14 and 13-15. They include abnormally high P wave amplitudes in the limb and right precordial leads. As in the case of left atrial abnormality, the term right atrial abnormality is preferred over other terms, such as right atrial enlargement, which suggest a particular underlying pathophysiology.

**Diagnostic Criteria**

Criteria commonly used to diagnose right atrial abnormality are listed in Table 13-3.

**MECHANISMS FOR ELECTROCARDIOGRAPHIC ABNORMALITIES.** Greater right atrial mass generates greater electrical force early during atrial activation, producing taller P waves in limb leads and increasing the initial P wave deflection in lead V1.

**DIAGNOSTIC ACCURACY.** Imaging studies have shown that the electrocardiographic findings of right atrial abnormality have limited sensitivity but high specificity for detecting right atrial enlargement.

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**TABLE 13-3 Common Diagnostic Criteria for Left and Right Atrial Abnormalities**

<table>
<thead>
<tr>
<th>LEFT ATRIAL ABNORMALITY</th>
<th>RIGHT ATRIAL ABNORMALITY*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged P wave duration &gt; 120 msec in lead II</td>
<td>Peaked P waves with amplitudes in lead II &gt; 0.25 mV (P pulmonale)</td>
</tr>
<tr>
<td>Prominent notching of P wave, usually most obvious in lead II, with interval between notches of 0.40 msec (P mitrale)</td>
<td>Prominent initial positivity in lead V1 or V2 &gt; 0.15 mV</td>
</tr>
<tr>
<td>Ratio between the duration of the P wave in lead II and duration of the PR segment &gt; 1.6</td>
<td>Increased area under initial positive portion of the P wave in lead V1 to &gt; 0.06 mm-sec</td>
</tr>
<tr>
<td>Increased duration and depth of terminal-negative portion of P wave in lead V1 (P terminal force) so that area subtended by it &gt; 0.04 mm-sec</td>
<td>Rightward shift of mean P wave axis to more than +75 degrees</td>
</tr>
<tr>
<td>Leftward shift of mean P wave axis to between −30 and −45 degrees</td>
<td></td>
</tr>
</tbody>
</table>

*In addition to criteria based on P wave morphologies, right atrial abnormalities are suggested by QRS changes, including (1) Q waves (especially qR patterns) in the right precordial leads without evidence of myocardial infarction and (2) low-amplitude (≤500 μV) QRS complexes in lead V1, with a threefold or greater increase in lead V5.
CLINICAL SIGNIFICANCE. Patients with chronic obstructive pulmonary disease and this electrocardiographic pattern (often referred to as P pulmonale) have more severe pulmonary dysfunction and significantly reduced survival than do others (see Chap. 78). However, comparison of electrocardiographic and hemodynamic parameters has not demonstrated a close correlation of P wave patterns and right atrial hypertension.

OTHER ATRIAL ABNORMALITIES. Patients with abnormalities in both atria—that is, biatrial abnormality—can have electrocardiographic patterns reflecting each defect. Suggestive findings include large biphasic P waves in lead V1 and tall and broad P waves in leads II, III, and aVF (see Fig. 13-15). P wave and PR segment changes can also be seen in patients with atrial infarction or pericarditis, as described later in this chapter.

Ventricular Hypertrophy

LEFT VENTRICULAR HYPERTROPHY. Left ventricular hypertrophy (LVH) produces changes in the QRS complex, the ST segment, and the T wave. The most characteristic finding is increased amplitude of the QRS complex. R waves in leads facing the left ventricle (i.e., leads I, aVL, V5, and V6) are taller than normal, and S waves in leads overlying the right ventricle (i.e., V1 and V2) are deeper than normal. These changes are illustrated in Figure 13-16.

ST-T wave patterns vary widely in patients with LVH. The ST segment may be normal or elevated in leads with tall R waves. In many patients, however, the ST segment is depressed and followed by an inverted T wave (Fig. 13-17). In most cases, the ST segment slopes downward from a depressed J point and the T wave is asymmetrically inverted. These LVH-related repolarization changes usually occur in patients with QRS changes but may appear alone. Particularly prominent inverted T waves, so-called giant negative T waves, are characteristic of hypertrophic cardiomyopathy with predominant apical thickening (Yamaguchi syndrome; see Fig. 13-46).

Other QRS changes seen in cases of LVH include widening of the QRS complex beyond 110 msec, a delay in the intrinsicoid deflection, and notching of the QRS complex. Additional electrocardiographic abnormalities may include prolongation of the QT interval and evidence of left atrial abnormality.
These electrocardiographic features are most typical of LVH induced by pressure overload of the left ventricle. Volume overload can produce a somewhat different electrocardiographic pattern, including tall, upright T waves, and sometimes narrow (less than 25 msec) but deep (0.2 mV or greater) Q waves in leads facing the left side of the septum or the left ventricular free wall (Fig. 13-18). These distinctions have limited value in diagnosing hemodynamic conditions and their use in electrocardiographic diagnoses has been discouraged.

Mechanisms for Electrocardiographic Abnormalities. Electrocardiographic changes of LVH result from abnormalities at the cellular, tissue, and volume conductor levels. These abnormalities may be compounded by changes caused by concomitant clinical conditions, such as myocardial ischemia.

At the cellular level, hypertrophy is associated with a form of electrical remodeling that alters action potential shape and duration. These include heterogeneous changes in the function and distribution of ion channels, myocyte size and branching patterns, and intercellular matrix.

These effects are augmented by an increase in the size of activation fronts moving across the thickened wall. These larger wave fronts subtend larger solid angles and result in higher body surface voltage. Prolonged transmural activation time required to activate the thickened wall, combined with delayed endocardial activation, contribute to the high voltage as well as QRS prolongation. Notching of the QRS complex can be produced by the fractionation of activation wave fronts by intramural scarring associated with wall thickening and damage.

In addition, LVH can shift the position of the heart so that the lateral free wall lies closer than normal to the chest wall. This causes an increase in body surface potentials in accordance with the inverse square law. Also, ventricular dilatation increases the size of the highly conductive intraventricular blood pool that increases the potentials produced by transmural activation fronts, a phenomenon termed the Brody effect.

Recent genome studies have also suggested that genetic factors may influence the emergence of electrocardiographic abnormalities of LVH. For example, electrocardiographic abnormalities are more common in carriers of genetic defects related to hypertrophic cardiomyopathy who do not have ventricular hypertrophy than in noncarriers.

ST-T segment abnormalities may reflect a primary disorder of repolarization that accompanies the cellular processes of hypertrophy or they may reflect subendocardial ischemia. Ischemia can be induced in the absence of coronary artery disease by the combination of high oxygen demand caused by high wall tension and limited blood flow to the subendocardium of the thickened wall. There is no association between the ST segment shifts and hemodynamic work, so the term strain used to describe these changes is best avoided and the term secondary ST-T wave abnormalities should be used.

![Image of electrocardiogram showing LVH with prominent positive anterior T waves from a patient with severe aortic regurgitation. The serum potassium level was normal.](image)

**Figure 13-18** LVH with prominent positive anterior T waves from a patient with severe aortic regurgitation. The serum potassium level was normal.

### Table 13-4 Common Diagnostic Criteria for Left Ventricular Hypertrophy

<table>
<thead>
<tr>
<th>MEASUREMENT</th>
<th>CRITERIA</th>
</tr>
</thead>
</table>
| Sokolow-Lyon voltages | $SV_1 + RV_1 > 3.5\, mV$  
|               | $RaVL > 1.1\, mV$ |
| Romhilt-Estes point score system | Any limb lead R wave or S wave $> 2.0\, mV$ (3 points)  
|               | or $SV_1$ or $SV_2$ $> 3.0\, mV$ (3 points)  
|               | or RV, to $RV_1$ $> 3.0\, mV$ (3 points)  
|               | ST-T wave abnormality, no digitalis therapy (3 points)  
|               | ST-T wave abnormality, digitalis therapy (1 point)  
|               | Left atrial abnormality (3 points)  
|               | Left axis deviation $> 30$ degrees (2 points)  
|               | QRS duration $> 90$ msec (1 point)  
|               | Intrinsicsoid deflection in $V_1$ or $V_2$ $> 50$ msec (1 point) |
| Cornell voltage criteria | $SV_1 + RaVL ≥ 2.8\, mV$ (for men)  
|               | $SV_1 + RaVL > 2.0\, mV$ (for women) |
| Cornell regression equation | Risk of LVH = $1/(1+e^{-0.15S})$ |
| Cornell voltage duration measurement | $QR$ duration $×$ Cornell voltage $> 2,436\, \text{mm-sec}$  
|               | $QR$ duration $×$ sum of voltages in all leads $> 1,742\, \text{mm-sec}$ |

Voltages are in mV, QRS is QRS duration in msec, PTF is the area under the P terminal force in lead $V_1$ (in mm-sec), and gender = 1 for men and 2 for women. LVH is diagnosed present if $p < 0.05$.

*Probable left ventricular hypertrophy is diagnosed if 4 points are present and definite left ventricular hypertrophy is diagnosed if 5 or more points are present.

For subjects in sinus rhythm, exp $= 4.558 + 0.092 (SV_1 + RaVL) - 0.306 TV_1 - 0.012 QR$ $- 0.278 PTFV - 0.559$ (gender).

For women, add 8 mm.

### Diagnostic Criteria

Many sets of diagnostic criteria for LVH have been developed based on these electrocardiographic abnormalities. Several of the more commonly used criteria are presented in Table 13-4.

*A comprehensive list of criteria has been presented by Hancock and colleagues.*
Most methods assess the presence or absence of LVH as a binary function, indicating that either LVH does or does not exist based on an empirically determined set of criteria. For example, the Sokolow-Lyon and Cornell voltage criteria require that voltages in specific leads exceed certain values. The Romhilt-Estes point score system assigns point values to amplitude and other criteria, including QRS axis and P wave patterns; definite LVH is diagnosed if 5 points are computed, and probable LVH is diagnosed if 4 points are computed. The Cornell voltage-duration method includes measurement of QRS duration as well as amplitudes.

Other methods seek to quantify left ventricular mass as a continuum. Diagnosis of LVH can then be based on a computed mass that exceeds an independently determined threshold. One set of criteria applying this approach is the Cornell regression equation shown in Table 13-4.

**Diagnostic Accuracy.** The relative diagnostic accuracy of these methods has been tested using autopsy, radiographic, echocardiographic and, most recently, magnetic resonance imaging measurements of left ventricular size as standards. Results are highly variable, varying with the specific electrocardiographic criteria that are tested, the imaging method that is relied on to determine anatomic measurements, and the population that is studied. For example, sensitivities are substantially lower when tested in the general population than in a high-risk population, such as hypertensive patients.46 Accuracy also varies with gender (with women having lower voltages than men), with race (with African Americans having higher voltages than other racial groups), and with body habitus (with obesity reducing electrocardiographic voltages).

In general, these studies have reported low sensitivity and high specificity. Sensitivities vary from approximately 10% in the general population to approximately 50% in cohorts with hypertension who are at greater risk of having LVH.46 A recent review of 21 studies has reported that the median sensitivity for six commonly used electrocardiographic criteria varies from 10.5% to 21% and the median specificity varies from 89% to 99%.35 Accuracies tend to be higher for the Cornell voltage, voltage-duration, and regression methods than for others.

Thus, all methods are limited as screening tests in which high sensitivity (few false-negatives) is critical but have good reliability as diagnostic tests when few false-positives are desired. As a result, in the general population, a negative ECG has a minimal effect on the pretest likelihood of LVH, whereas a positive ECG would substantially increase the likelihood.35 Because of the variability in the accuracy of the criteria from one trial to another, no one criterion can be established as the preferred method.31 Using multiple criteria may increase the accuracy of electrocardiographic screening.

Repolarization abnormalities associated with QRS findings increase the correlation with anatomic LVH. ST and T wave abnormalities are associated with a threefold greater prevalence of anatomic LVH in patients without coronary artery disease.

**Concomitant Electrocardiographic Abnormalities.** Concomitant electrocardiographic abnormalities may interfere with the diagnosis of LVH and reduce the accuracy of standard electrocardiographic criteria. Intra-ventricular conduction defects may affect the accuracy of electrocardiographic criteria for LVH. In left anterior fascicular block, the larger R waves in leads I and aVL and smaller R waves but deeper S waves in leads V5 and V6, make criteria relying on R wave amplitude less valuable.

Left bundle branch block (LBBB) makes the diagnosis of LVH difficult because of the extensive reordering of left ventricular activation (see later). In addition, the high prevalence of LVH in patients with LBBB makes assessment of the accuracy of criteria difficult. Some have concluded that the diagnosis should not be attempted in this setting, whereas others think that the diagnosis can be made. Left atrial abnormalities and QRS durations longer than 155 msec, as well as precordial lead voltage criteria, have relatively high specificity for LVH in the presence of LBBB.

The delay in right ventricular activation that occurs with right bundle branch block (see later) increases the cancellation of left ventricular forces during the middle of the QRS complex.40 This reduces the amplitude of the S wave in the right precordial leads and of the R waves in the left precordial leads, and thus reduces the accuracy of electrocardiographic criteria for LVH. Diagnostic criteria for LVH in patients with conduction defects have been suggested.38

**Clinical Significance**

An accurate electrocardiographic diagnosis of LVH is important to detect hypertrophy, assess prognosis, and monitor progression or regression of hypertrophy during treatment. Although imaging methods may provide a more direct assessment of structural LVH, the ECG remains critical in clinical settings because of its simplicity and limited expense, and because electrocardiographic findings may provide independent, clinically important information concerning, for example, prognosis.

The presence of electrocardiographic criteria for LVH identifies a subset of the general population and of those with hypertension with a significantly increased risk for cardiovascular morbidity and mortality. Among patients with hypertension, the LIFE study41 has reported that declines in electrocardiographic measures such as the Cornell and Sokolow-Lyon voltages during antihypertensive therapy correlate with a decrease in left ventricular mass and with lower likelihoods of cardiovascular mortality and morbidity independently of the extent of blood pressure lowering. A 1-SD decrease in the Cornell product was associated with a 22% decrease in cardiovascular deaths.

Patients with repolarization abnormalities have, on average, more severe degrees of LVH and more commonly have symptoms of left ventricular dysfunction, in addition to a greater risk of future cardiovascular events. In the LIFE study cited earlier, hypertensive patients with LVH-related ST-T wave abnormalities were 1.8 times as likely to develop congestive heart failure and 2.8 times as likely to experience heart failure–related death than patients without such ST-T wave changes.31

**RIGHT VENTRICULAR HYPERTROPHY.** The electrocardiographic effects of right ventricular hypertrophy (RVH) differ in fundamental ways from those of LVH (see Chap. 78). The right ventricle is considerably smaller than the left ventricle and produces electrical forces that are largely cancelled by those generated by the larger left ventricle. Thus, for RVH to be manifested on the ECG, it must be severe enough to overcome the masking effects of the larger left ventricular forces. In addition, increasing dominance of the right ventricle changes the ECG in fundamental ways, whereas an enlarged left ventricle produces predominantly quantitative changes in underlying normal waveforms.

The electrocardiographic changes associated with moderate to severe concentric hypertrophy of the right ventricle include abnormal, tall R waves in anteriorly and rightward-directed leads (leads aVR, V1, and V4), and deep S waves and abnormally small r waves in leftward-directed leads (I, aVL, and lateral precordial leads; Fig. 13-19). These changes result in a reversal of normal R wave progression in the precordial leads, a shift in the frontal plane QRS axis to the right, and the presence of S waves in leads I, II, and III (S,S,S pattern).

Less severe hypertrophy, especially when limited to the outflow tract of the right ventricle that is activated late during the QRS complex, produces less marked changes. Electrocardiographic abnormalities may be limited to an rSr′ pattern in V1 and persistence of s (or S) waves in the left precordial leads. This pattern is typical of right ventricular volume overload such as that produced by an atrial septal defect.

**Diagnostic Criteria**

These electrocardiographic abnormalities form the basis for the diagnostic criteria for RVH. The most commonly relied on criteria for the ECG diagnosis of RVH are listed in Table 13-5. The diagnostic accuracies of these criteria remain unclear. Although the older literature has suggested very high specificities for many of the listed criteria, these estimates were often based on small and highly selective populations. The sensitivities and specificities in the general population remain to be accurately determined.

**Mechanisms for Electrocardiographic Abnormalities.** These electrocardiographic patterns result from three effects of RVH:

1. Current fluxes between hypertrophied cells are stronger than normal and produce higher than normal voltage on the body surface.
2. Activation fronts moving through the enlarged right ventricle are larger than normal and produce higher surface potentials, as predicted by the solid angle theorem.
3. The activation time of the right ventricle is prolonged.
The electrocardiographic evidence of RVH has limited value in assessing the severity of pulmonary hypertension or lung disease. QRS changes do not generally appear until ventilatory function is significantly depressed, with the earliest change commonly being a rightward shift in the mean QRS axis, and the correlation with ventilatory function or hemodynamics is poor. The presence of right atrial abnormality, an S₁S₂S₃ pattern, or both is associated with reduced survival.

Pulmonary Embolism

Acute right ventricular pressure overload such as that produced by pulmonary embolism can produce characteristic electrocardiographic patterns (Fig. 13-21; see Chap. 77). These include the following: (1) a QR or qR pattern in the right ventricular leads; (2) an S₁Q₃T₃ pattern with an S wave in lead I and new or increased Q waves in lead III and sometimes aVF, with T wave inversions in those leads; (3) ST-segment deviation and T wave inversions in leads V₁ to V₃; and (4) incomplete or complete right bundle branch block (RBBB). Sinus tachycardia is usually present. Occasionally, with massive pulmonary obstruction, a right ventricular current of injury pattern is seen, with ST elevations in the right midprecordial leads.

Electrocardiographic findings of acute right ventricular overload in patients with pulmonary embolism correspond to obstruction of much of the pulmonary arterial bed and significant pulmonary hypertension. However, even with major pulmonary artery obstruction, the ECG is notoriously deceptive and may show little more than minor or nonspecific waveform changes, or it may even be normal. The classic S₁Q₃T₃ pattern occurs in only about 10% of cases of acute pulmonary embolism (see Chap. 77). Furthermore, the specificity of this finding is limited, because it can occur with other causes of pulmonary hypertension. An analysis of the ECGs of patients with right ventricular dilation caused by acute pulmonary embolism has reported positive predictive accuracies of 23% to 69%.

BIVENTRICULAR HYPERTROPHY. Hypertrophy of both ventricles produces complex electrocardiographic patterns. In contrast to biventricular hypertrophy, the result is not the simple sum of the two sets of abnormalities. The effects of enlargement of one chamber may cancel the effects of enlargement of the other. The greater left ventricular hypertrophy may result in left axis deviation, and the right ventricle may produce a leftward shift of the QRS complex.

Right ventricular activation now ends after the completion of left ventricular activation so that its effects are no longer canceled by the more powerful forces of the left ventricle and become manifest in the ECG. Because the right ventricle is located anteriorly as well as to the right of the left ventricle, the effects produce increased potentials in leads directed anteriorly and to the right, especially late during the QRS complex.

Chronic obstructive pulmonary disease (see Chap. 78) can induce electrocardiographic changes by producing RVH, positional changes caused by insulating and positional changes produced by hyperinflation of the lungs include reduced amplitude of the QRS complex, right axis deviation in the frontal plane, and delayed transition in the precordial leads, probably reflecting a vertical and caudal shift in heart position caused by hyperinflation and a flattened diaphragm. Evidence of true RVH includes the following: (1) right axis deviation more positive than 110 degrees; (2) deep S waves in the lateral precordial leads, and (3) an S₁Q₃T₃ pattern, with an S wave in lead I (as an RS or rS complex), an abnormal Q wave in lead III, and an inverted T wave in the inferior leads.

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### TABLE 13-5 Common Diagnostic Criteria for Right Ventricular Hypertrophy

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R in V₁ ≥ 0.7 mV</td>
<td>QR in V₁ R/S in V₁ &gt; 1 with R &gt; 0.5 mV</td>
</tr>
<tr>
<td>R/S in V₁ or V₆ &lt; 1</td>
<td>S in V₁, or V₆ &gt; 0.7 mV</td>
</tr>
<tr>
<td>R in V₁, or V₆ ≥ 0.4 mV with S in V₁ ≤ 0.2 mV</td>
<td>Right axis deviation (≥90 degrees)</td>
</tr>
<tr>
<td>S₁Q₃ pattern</td>
<td>S₁S₂S₃ pattern</td>
</tr>
<tr>
<td>P pulmonale</td>
<td></td>
</tr>
</tbody>
</table>


Chronic obstructive pulmonary disease (see Chap. 78) can induce electrocardiographic changes by producing RVH, positional changes caused by insulating and positional changes produced by hyperinflation of the lungs include reduced amplitude of the QRS complex, right axis deviation in the frontal plane, and delayed transition in the precordial leads, probably reflecting a vertical and caudal shift in heart position caused by hyperinflation and a flattened diaphragm. Evidence of true RVH includes the following: (1) right axis deviation more positive than 110 degrees; (2) deep S waves in the lateral precordial leads, and (3) an S₁Q₃T₃ pattern, with an S wave in lead I (as an RS or rS complex), an abnormal Q wave in lead III, and an inverted T wave in the inferior leads.
Intraventricular Conduction Delays

Delays in intraventricular conduction of cardiac impulses may result from abnormalities in the His-Purkinje system or in ventricular muscle, and may be caused by structural changes or by the functional properties of the cardiac conduction system. Although the conduction abnormalities to be described will be referred to in specific anatomic terms (e.g., right bundle branch block), the electrocardiographic changes may be caused by abnormalities in various sites within the ventricles. Hence, the anatomic references are not intended to localize sites of impaired function precisely.

FASCICULAR BLOCK. Under normal conditions, activation of the left ventricle begins almost simultaneously at the insertion points of the fascicles of the left bundle branch. Absolute or relative delays in conduction in a fascicle, fascicular block, results in an abnormal

ventricular forces generated in LVH increase the degree of RVH needed to overcome the dominance of the left ventricle, and the anterior forces produced by RVH may cancel or be canceled by enhanced posterior forces generated by LVH.

Because of these factors, specific electrocardiographic criteria for RVH or LVH are seldom observed with biventricular enlargement. Rather, electrocardiographic patterns are usually a modification of the features of LVH, such as the following: (1) tall R waves in the right and left precordial leads; (2) vertical heart position or right axis deviation in the presence of criteria for LVH; (3) deep S waves in the left precordial leads in the presence of electrocardiographic criteria for LVH; or (4) a shift in the precordial transition zone to the left in the presence of LVH. The presence of prominent left atrial abnormality or atrial fibrillation with evidence of right ventricular or biventricular enlargement (especially LVH with a vertical or rightward QRS axis) should suggest chronic rheumatic valvular disease (Fig. 13-22; see Chap. 66).

**FIGURE 13-20** Pulmonary emphysema simulating anterior infarction in a 58-year-old man with no clinical evidence of coronary disease. Note the relative normalization of R wave progression with placement of the chest leads an interspace below their usual position (e.g., V5, V6). (From Chou TC: Pseudo-infarction [noninfarction Q waves]. In Fisch C [ed]: Complex Electrocardiography. Vol 1. Philadelphia, FA Davis, 1973.)

**FIGURE 13-21** Acute cor pulmonale secondary to pulmonary embolism simulating inferior and anterior infarction. This tracing exemplifies the classic pseudoinfarct patterns sometimes seen: an S1-Q3-T3, a QR in V1 with poor R wave progression in the right precordial leads (clockwise rotation), and right precordial to midprecordial T wave inversion (V1 to V4). Sinus tachycardia is also present. The S1-Q3 pattern is usually associated with a QR or QS complex, but not an Rs in aVR. Furthermore, acute cor pulmonale per se does not cause prominent Q waves in II (only in III and aVF). (From Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis. 4th ed. St. Louis, Mosby-Year Book, 1991.)
sequence of early left ventricular activation that, in turn, leads to characteristic electrocardiographic patterns. Even modest delays in conduction through the affected structure may be enough to alter ventricular activation patterns sufficiently to produce characteristic electrocardiographic patterns; a complete block of conduction is not required.

**Left Anterior Fascicular Block.** The electrocardiographic features of left anterior fascicular block (LAFB) are listed in Table 13-6 and illustrated in Figure 13-23. The most characteristic finding is marked left axis deviation.

The left anterior fascicle normally activates the anterosuperior portion of the left ventricle early during the QRS complex. With LAFB, this region is activated later than normal, resulting in unbalanced inferior and posterior forces early during ventricular activation (initiated by the left posterior fascicle) and unopposed anterosuperior forces later during the QRS complex (the region activated late).

**TABLE 13-6 Common Diagnostic Criteria for Unifascicular Blocks**

<table>
<thead>
<tr>
<th>Block</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left Anterior Fascicular Block</strong></td>
<td>Frontal plane mean QRS axis = −45 to −90 degrees &lt;br&gt;qR pattern in lead aVL &lt;br&gt;QRS duration &lt; 120 msec &lt;br&gt;Time to peak R wave in aVL ≥ 45 msec</td>
</tr>
<tr>
<td><strong>Left Posterior Fascicular Block</strong></td>
<td>Frontal plane mean QRS axis = +90 to +180 degrees &lt;br&gt;rS pattern in leads I and aVL with qR patterns in leads III and aVF &lt;br&gt;QRS duration &lt; 120 msec &lt;br&gt;Exclusion of other factors causing right axis deviation (e.g., right ventricular overload patterns, lateral infarction)</td>
</tr>
</tbody>
</table>
These changes are manifest on the ECG as a leftward shift of the mean frontal plane QRS axis. The characteristic pattern in the inferior leads includes initial r waves (caused by early unopposed activation of the inferoposterior left ventricle) followed by deep S waves (caused by late unopposed activation of the posteroseptal left ventricle; left axis deviation with rS patterns). The left-looking leads (e.g., leads I and aVL) show a qR pattern.

However, LAFB is not synonymous with left axis deviation. Axis shifts to between 30 and 45 degrees commonly reflect other conditions, such as LVH, without conduction system damage, and such patterns are best referred to as (moderate) left axis deviation rather than LAFB.

LAFB can also produce prominent changes in the precordial leads. Leads V3 through V6, commonly show deep S waves (a delayed transition) produced by the late activation of the anteroseptal left ventricle. The overall QRS duration is not prolonged; fascicular block alters the sequence but not the overall duration of left ventricular activation.

Damage to the left anterior fascicle is a very common occurrence because of the delicate nature of the structure. Left anterior fascicular block is common in persons without overt cardiac disease, as well as in persons with a wide range of conditions. In patients with coronary artery disease, the presence of LAFB may be associated with an increased risk of cardiac death. Commonly associated conditions include myocardial infarction, especially occlusion of the left anterior descending coronary artery, LVH, hypertrophic and dilated cardiomyopathy, and various cardiac degenerative diseases. The development of LAFB with rS complexes in leads II, III, and aVF can mask the Q waves of an inferior myocardial infarction.

**LEFT POSTERIOR FASCICULAR BLOCK.** Conduction delay in the left posterior fascicle is considerably less common than delay in the anterior fascicle because of its thicker structure and more protected location near the left ventricular inflow tract. Conduction delay results in early unopposed activation of the anteroseptal left ventricular free wall, followed by late activation of the inferoposterior aspect of the left ventricle—that is, the reverse of the pattern observed with LAFB.

The electrocardiographic features of left posterior fascicular block (LPFB; see Table 13-6 and Fig. 13-23), reflect this altered activation pattern. Right axis deviation, with rS5 patterns in leads I and aVL as well as qR complexes in the inferior leads, is the result of early unopposed activation forces from the anteroseptal aspect of the left ventricle (activated early via the left anterior fascicle and producing the initial q and r waves) and of late unopposed forces from the inferoposterior free wall (activated late via the left posterior fascicle and generating the late S and R waves). As in the case of LAFB, the overall activation time of the ventricles is not prolonged, and the QRS duration remains normal.

LPFB can occur in patients with almost any cardiac disease but is unusual in otherwise healthy persons. Other conditions that augment or appear to augment the rightward electrical forces in the frontal plane, such as right ventricular overload syndromes and extensive lateral infarction, can produce similar electrocardiographic patterns and must be excluded before a diagnosis of LPFB can be made.

**OTHER FORMS OF FASCICULAR BLOCK.** Electrocardiographic patterns that suggest left septal fascicular block have also been described. The most common electrocardiographic finding attributed to this form of block is the absence of septal q waves. It has been recommended that this term not be used because clear diagnostic criteria have not been developed.

**LEFT BUNDLE BRANCH BLOCK.** LBBB results from conduction delay or block in any of several sites in the intraventricular conduction system, including the main left bundle branch, each of the two fascicles, the distal conduction system of the left ventricle or less commonly, fibers of the bundle of His that become the main left bundle branch. The result is extensive reorganization of the activation and recovery patterns of the left ventricle that produces extensive changes in the QRS complex and STT wave.

**ECG Abnormalities**

LBBB produces a prolonged QRS duration, abnormal QRS patterns, and STT wave abnormalities (Fig. 13-24). Commonly accepted diagnostic criteria for LBBB are listed in Table 13-7. Basic requirements include a prolonged QRS duration to 120 msec or more, broad and commonly notched R waves in leads I and aVL and the left precordial leads, narrow r waves followed by deep S waves in the right precordial leads, and absent septal q waves. R waves are typically tall and S waves are deep. The mean QRS axis with LBBB is highly variable; it can be normal, deviated to the left or rarely, deviated to the right. In addition to these features, some require a prolonged time to the peak of the R wave (> 60 msec) in the left precordial leads to diagnose LBBB.

STT wave changes are prominent with LBBB. In most cases, the ST segment and T wave are discordant with the QRS complex. That is, the ST segment is depressed and the T wave is inverted in leads with positive QRS waves (e.g., leads I, aVL, V5, and V6), and the ST segment is elevated and the T wave is upright in leads with negative QRS complexes (e.g., leads II and V1).

An incomplete form of LBBB may result from lesser degrees of conduction delay in the left bundle branch system. Electrocardiographic features include the following: (1) modest prolongation of the QRS complex (between 100 and 119 msec); (2) loss of septal q waves; (3) slurring and notching of the upstroke of tall R waves; and (4) delay in the time to the peak of the R wave in left precordial leads. The pattern commonly is similar to that of LVH.

**MECHANISMS FOR THE ELECTROCARDIOGRAPHIC ABNORMALITIES.** The electrocardiographic abnormalities of LBBB result from an almost completely...
Clinical Significance

LBBB usually appears in patients with underlying heart disease; approximately 30% of patients with heart failure have LBBB. As many as 70% of people developing LBBB had preceding ECG evidence of LVH. However, as many as 12% of patients with LBBB have no demonstrable heart disease. The prevalence and severity of left ventricular dysfunction increase progressively as QRS duration increases.

LBBB has significant prognostic implications. Even in persons without overt heart disease, LBBB is associated with a higher than normal risk of cardiovascular mortality from infarction and heart failure. The risk of heart failure increases progressively as QRS duration increases. LBBB is associated with substantially higher than expected risks of high-grade atrioventricular block and cardiac death, mostly because of sudden death outside the hospital setting. Among patients with coronary artery disease, the presence of LBBB correlates with more extensive disease, more severe left ventricular dysfunction, and reduced survival rates.

Patients with associated left or, rarely, right axis deviation have more severe clinical manifestations. Left axis deviation is associated with more severe conduction system disease that includes the fascicular and the main left bundle, whereas right axis deviation suggests dilated cardiomyopathy with biventricular enlargement.

The abnormal ventricular activation pattern of LBBB itself induces hemodynamic changes that are superimposed on the abnormalities caused by the underlying heart disease. These include premature movement of the septum into the left ventricle that is followed by delayed contraction of the posterior and lateral walls of the left ventricle. As a result, when the lateral wall does contract, blood pushes the compliant septum toward the right ventricular cavity rather than transseptal spread. Other patients with LBBB have near-normal left septal activation time, indicating a more peripheral site of block.

After septal activation, activation of the left ventricular free wall is also varied. The spread of activation may be disrupted by regions of block, which may, for example, require the activation of the anterior left ventricular myocardium by fronts moving through the inferior or apical portions of the left ventricle. These blocks may be fixed, caused by scarring, or may be transient, related to functional derangements in conduction. Overall activation may then require more than 180 msec, depending largely on the functional status of the distal left bundle and Purkinje systems. Irregular spread predominantly through working muscle fibers rather than the specialized conduction system results in notching and slurring of the wide QRS complex.

The discordant ST-T wave pattern is a result of the transventricular recovery gradients. The presence of LBBB, the right ventricle is activated and recovers earlier than the left, so recovery vectors or dipoles are directed toward the right and away from the left. Hence, positive ST-T waves will be registered over the right ventricle, and negative ones over the left ventricle. These transventricular gradients play only a minor role during normal conduction because the simultaneous activation of multiple regions cancels the forces that they produce; with bundle branch block, activation is sequential and cancellation is reduced. Because the ST-T wave changes with LBBB are generated by abnormalities in conduction, they are called secondary ST-T wave abnormalities; as will be discussed later, ST-T wave changes produced by direct abnormalities of the recovery process are referred to as primary ST-T wave abnormalities.

RIGHT BUNDLE BRANCH BLOCK. Right bundle branch block is a result of conduction delay in any portion of the right-sided intraventricular conduction system. The delay can occur in the main right bundle branch itself, in the bundle of His, or in the distal right ventricular conduction system. The latter is the common cause of RBBB after a right ventriculotomy is performed, for example, to correct the tetralogy of Fallot.

Electrocardiographic Abnormalities

Major features of RBBB are illustrated in Figure 13-24 and commonly used diagnostic criteria are listed in Table 13-7. As with LBBB, the QRS complex duration exceeds 120 msec. The right precordial leads show prominent and notched R waves with rs′r′, rs′R′, or sR′ patterns, whereas leads I and aVL and the left precordial leads demonstrate wide S waves that are longer in duration than the preceding R wave. The ST-T waves are, as in LBBB, discordant with the QRS complex, so T waves are inverted in the right precordial leads (and other leads with a terminal R′ wave) and upright in the left precordial leads and in leads I and aVL.

The mean QRS axis is not altered by RBBB. Axis shifts can occur, however, as a result of the simultaneous occurrence of fascicular block along with RBBB (see later). This combination of RBBB with LAFB (producing left axis deviation) or LPFB (producing right axis deviation) is termed bifascicular block.

Features indicative of incomplete RBBB, produced by lesser delays in conduction in the right bundle branch system, are commonly seen. This finding is most frequently characterized by an rSr′ pattern in lead V1 with a QRS duration between 100 and 120 msec. Although these electrocardiographic changes of incomplete RBBB are commonly attributed to conduction defects, they can reflect RVH (especially with a rightward QRS axis) without intrinsic dysfunction of the conduction system. An rs′R′ morphology in lead V1, (and sometimes V2) with a narrow QRS duration (≤ 100 msec) is a common physiologic or positional variant.

Mechanism for Electrocardiographic Abnormalities. With delay or block in the proximal right bundle branch system, activation of the right side of the septum is initiated only after slow transseptal spread of activation from the left septal surface. The right ventricular anterior free wall is then excited slowly, followed by activation of the lateral right ventricular wall and, finally, the right ventricular outflow tract.

The result is delayed and slow activation of the right ventricle. Much or all of the right ventricle undergoes activation after depolarization of the left ventricle has been completed. This reduces the cancellation of right ventricular activation forces by the more powerful left ventricular activation forces. The late and unopposed emergence of right ventricular forces produces increased anterior and rightward voltage in the later half of the ECG, as well as a prolonged QRS complex. Discordant ST-T wave patterns are generated by the same mechanisms as for LBBB; with RBBB, recovery forces are directed toward the earlier activated left ventricle and away from the right.

A substantial proportion of patients with RBBB have abnormalities of left ventricular activation that are similar to those in patients with LBBB. This suggests that many patients with RBBB have a diffuse, biventricular conduction system disease.

Clinical Significance

RBBB is a common finding in the general population, and many persons with RBBB have no clinical evidence of structural heart disease. The high prevalence of RBBB is a reflection of the relative fragility of the right bundle branch, as suggested by the development of bifascicular block.
of RBBB after the minor trauma produced by right ventricular catheterization.

In some reports, RBBB is an independent predictor of cardiovascular mortality. The new onset of RBBB predicts a higher rate of coronary artery disease, congestive heart failure, and cardiovascular mortality. When cardiac disease is present, the coexistence of RBBB suggests advanced disease with, for example, more extensive multivessel disease and reduced long-term survival in patients with ischemic heart disease. An entity known as the Brugada syndrome has been described, in which an RBBB-like pattern with persistent ST-segment elevation in the right precordial leads is associated with susceptibility to ventricular tachyarrhythmias and sudden cardiac death (see Chaps. 9 and 39).

RBBB interferes with other electrocardiographic diagnoses, although to a lesser extent than LBBB. The diagnosis of RVH is more difficult to make with RBBB because of the accentuated positive potentials in lead V1; RVH is suggested, although with limited accuracy, by the presence of an R wave in lead V1 that exceeds 1.5 mV and a rightward shift of the mean QRS axis. The usual criteria for LVH can be applied but have lower sensitivities than with normal conduction. The combination of left atrial abnormality or left axis deviation with RBBB also suggests underlying LVH.

**Multifascicular Blocks.** The term *multifascicular block* refers to conduction delay or block in more than one of the structural components of the specialized conduction system—that is, the left bundle branch, the left anterior and posterior fascicles of the left bundle branch, and the right bundle branch. Conduction delay in any two fascicles is termed *bifascicular block,* and delay in all three fascicles is termed *trifascicular block.* The term *bilateral bundle branch block* has been used to refer to concomitant conduction abnormalities in both the left and right bundle branch systems. Bifascicular block can have several forms: (1) RBBB with LAFB, characterized by the electrocardiographic pattern of RBBB plus left axis deviation beyond −45 degrees (Fig. 13-25); (2) RBBB with LPFB, with an electrocardiographic pattern of RBBB and a mean QRS axis deviation to the right of +120 degrees (Fig. 13-26); or (3) LBBB alone that may be caused by delay in both the anterior and posterior fascicles. This form of LBBB represents one of the inadequacies of current electrocardiographic terminology and the simplification inherent in the trifascicular schema of the conduction system. The electrophysiologic consequences of these abnormalities are discussed in Chaps. 35 and 38.

Trifascicular block involves conduction delay in the right bundle branch plus delay in the main left bundle branch or in both the left anterior and the left posterior fascicles. The resulting electrocardiographic pattern is dependent on the relative degree of delay in the affected structures and on the shortest conduction time from the atria to the ventricles through any one part of the conducting system. Ventricular activation begins at the site of insertion of the branch with the fastest conduction time and spreads from there to the remainder of the ventricles. For example, if conduction in the right and left bundle branches exists and the delay in the right bundle branch is shorter than the delay in the left bundle branch, activation will begin in the right ventricle and the QRS pattern will resemble that of LBBB. If the delay were longer in the right bundle branch than in the left bundle branch, the electrocardiographic pattern would be that of RBBB. The fascicle with the longest delay can vary with, for example, the heart rate and lead to changing or alternating conduction patterns (Fig. 13-27).

What distinguishes electrocardiographic patterns of trifascicular block from those of bifascicular block is an increase in the overall conduction time from the AV node to the ventricles. In bifascicular block, conduction through the unaffected fascicle (and hence, overall conduction time) is normal in the absence of concomitant AV nodal conduction delay. In trifascicular block, however, the delay in conduction through even the least affected fascicle is abnormal and results in relative prolongation of the overall conduction time from the AV node to the ventricular myocardium. (Note that only delay, not block, of conduction is required. If block were present in all fascicles, conduction would fail and complete heart block would result. This situation is perhaps best illustrated by cases of alternating bundle branch block (see Fig. 13-27); if the block were total in one bundle branch, development of block in the other would produce complete AV block rather than a change in bundle branch block patterns.) Thus, a diagnosis of trifascicular block requires an electrocardiographic pattern of bifascicular block plus evidence of prolonged conduction below the AV node.

This delay in conduction is most specifically observed as a prolongation of the His-ventricular (HV) time in intracardiac recordings. On the surface ECG, the delay in conduction delay may be manifest as a prolonged PR interval. However, the PR interval includes conduction time in the AV node as well as in the intraventricular conduction system. Prolonged intraventricular conduction may be insufficient to extend the PR interval beyond normal limits, whereas a prolonged PR interval can reflect delay in the AV node rather than in all three intraventricular fascicles.

Thus, it should be noted that the finding of a prolonged PR interval in the presence of an electrocardiographic pattern consistent with

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**FIGURE 13-25** Sinus rhythm at 95 beats/min with 2:1 AV block. Conducted ventricular beats show a pattern consistent with bifascicular block with delay or block in the right bundle and left anterior fascicle. The patient underwent pacemaker implantation for presumed infrahisian block.
Sinus rhythm with a 2:1 atrioventricular block. QRS morphology in the conducted beats is consistent with bifascicular block with delay or block in the right bundle and left posterior fascicle. Subsequently, complete heart block was also noted. The patient underwent pacemaker implantation for presumed infrahisian block.

Bifascicular block is not diagnostic of trifascicular block, whereas the presence of a normal PR interval does not exclude this finding.

The major clinical implication of a multifascicular block is its relation to advanced conduction system disease. It may be a marker for severe myocardial disease and may identify patients at risk for heart block (see Figs. 13-25 and 13-26), as discussed in Chaps. 35, 38, and 39.

**Rate-Dependent Conduction Block (Aberration).** Intraventricular conduction delays can result from the effects of changes in the heart rate, as well as from fixed pathologic lesions in the conduction system. Rate-dependent block or aberration (see Chap. 39) can occur at relatively high or low heart rates. In acceleration (tachycardia)-dependent block, conduction delay occurs when the heart rate exceeds a critical value. At the cellular level, this aberration is the result of encroachment of the impulse on the relative refractory period (sometime during phase 3 of the action potential) of the preceding impulse, which results in slower conduction. This form of rate-related block is relatively common and can have the electrocardiographic pattern of RBBB or LBBB (Figs. 13-28 and 13-29).

In deceleration (bradycardia)-dependent block, conduction delay occurs when the heart rate falls below a critical level. Although the mechanism is not clearly established, it may reflect abnormal phase 4 depolarization of cells so that activation occurs at lower resting potentials. Deceleration-dependent block is less common than acceleration-dependent block and is usually seen only in patients with significant conduction system disease (Fig. 13-30).

Other mechanisms of ventricular aberration include concealed conduction (anterograde or retrograde) in the bundle branches (see Figs. 13-28 and 13-29), premature excitation, depressed myocardial conduction as a result of drug effects or hyperkalemia (see Fig. 13-49, top), and the effect of changing cycle length on refractoriness (the Ashman phenomenon; see Chaps. 29).
The duration of the refractory period is a function of the immediately preceding cycle length: the longer the preceding cycle, the longer the subsequent refractory period. Therefore, abrupt prolongation of the immediately preceding cycle can result in aberration as part of a long cycle—short cycle sequence. These so-called Ashman beats usually have an RBBB morphology (see Fig. 13-28).

**OTHER FORMS OF CONDUCTION ABNORMALITIES.**

**NOTCHING.** The presence of multiple deflections within the QRS complex (e.g., r5r, sFs, rSs′ or multiple r′ patterns) or the presence of high-frequency notches within the R and S wave without overall prolongation of the QRS complex may also reflect forms of intraventricular conduction defects, especially in patients with known coronary artery disease. These findings have reported sensitivities and specificities of over 85% for the presence of myocardial scars or ventricular aneurysms, but more definitive studies are needed. 57

**PERI-INFARCTION BLOCK.** This term refers to conduction delay in the region of a myocardial infarction. It is manifest in electrocardiographic leads with pathologic Q waves when the terminal portion of the QRS complex is wide and directed opposite to the Q wave, such as a QR complex in leads III and aVF. A related abnormality is peri-ischemic block, manifested by a reversible widening of the QRS complex in electrocardiographic leads with ST-segment elevation caused by acute injury. 54

### Myocardial Ischemia and Infarction

The ECG remains a key test for the diagnosis of acute and chronic coronary syndromes. 52-56 The findings vary considerably depending importantly on four major factors: (1) the duration of the ischemic process (acute versus evolving/chronic); (2) its extent (transmural versus nontransmural); (3) its topography (anterior versus inferior-posterior-lateral or right ventricular); and (4) the presence of other underlying abnormalities (e.g., LBBB, Wolff-Parkinson-White syndrome, or pacemaker patterns) that can mask or alter the classic patterns.

A key clinical distinction is between ST-segment elevation myocardial infarction (or ischemia; STEMI) and non-STEMI because of the therapeutic implications. Emergency coronary reperfusion therapy has only proven to be consistently efficacious in the former syndrome.

**REPOLARIZATION (ST-T WAVE) ABNORMALITIES.** The earliest and most consistent electrocardiographic finding during acute severe ischemia is deviation of the ST segment as a result of a current of injury mechanism (see Chap. 54). Under normal conditions, the ST segment is usually nearly isoelectric because almost all healthy myocardial cells attain approximately the same potential during the initial to middle phases of repolarization, corresponding to the plateau phase of the ventricular action potential. Ischemia, however, has complex time-dependent effects on the electrical properties of myocardial cells. Severe acute ischemia can reduce the resting membrane potential, shorten the duration of the action potential in the ischemic area, and decrease the rate of rise and amplitude of phase 0 (Fig. 13-31). These changes cause a voltage gradient between normal and ischemic zones that leads to electrocardiographic flow between these regions. Resulting currents of injury are represented on the surface ECG by deviation of the ST segment.

Both “diastolic” and “systolic” injury currents have been proposed to explain ischemic ST-segment elevations (Fig. 13-32). 57 According to the diastolic current of injury hypothesis, ischemic ST-segment elevation is attributable to negative (downward) displacement of the electrical diastolic baseline (the TQ segment of the ECG). Ischemic cells remain relatively depolarized during phase 4 of the ventricular action potential (i.e., lower membrane resting potential; see Fig. 13-31) and depolarized muscle carries a negative extracellular charge relative to repolarized muscle. Therefore, during electrical diastole, current (the diastolic current of injury) will flow between the partly or completely depolarized ischemic myocardium and the neighboring, normally repolarized, uninjured myocardium. The injury current vector will be directed away from the more negative ischemic zone toward the more positive normal myocardium. As a result, leads overlying the ischemic zone will record a
These electrophysiologic effects create a voltage gradient between ischemic and normal cells during different phases of the cardiac electrical cycle. The resulting currents of injury are reflected on the surface ECG by deviation of the ST segment (see Fig. 13-32).

Conventional alternating-current ECGs compensate for the baseline shift, and an apparent ST-segment elevation is produced over the ischemic zone (Fig. 13-33). Reciprocal ST depression can appear in leads reflecting the contralateral surface of the heart. Occasionally, the reciprocal changes can be more apparent than the primary ST-segment elevations. When ischemia is confined primarily to the subendocardium, the overall ST vector typically shifts toward the inner ventricular layer and the ventricular cavity such that the overlying (e.g., anterior precordial) leads show ST-segment depression, with ST-segment elevation in lead aVR (see Fig. 13-33). This subendocardial ischemia pattern is the typical finding during spontaneous episodes of angina pectoris or during symptomatic or asymptomatic (silent) ischemia induced by exercise or pharmacologic stress tests (see Chaps. 14 and 37). Multiple factors can affect the amplitude of acute ischemic ST deviations. Profound ST-segment elevation or depression in multiple leads usually indicates very severe ischemia. Conversely, prompt resolution of ST-segment elevation following thrombolytic therapy or percutaneous coronary interventions is a specific marker of successful reperfusion. These relationships are not universal, however, because severe ischemia or even infarction can occur with slight or absent ST-T changes. Furthermore, a relative increase in T wave amplitude (hyperacute T waves) can accompany or precede the ST-segment elevations as part of the injury current pattern attributable to ischemia with or without infarction (Fig. 13-34).

QRS changes. With actual infarction, depolarization (QRS) changes often accompany repolarization (ST-T) abnormalities (Fig. 13-35). Necrosis of sufficient myocardial tissue can lead to decreased R wave amplitude or Q waves in the anterior, lateral, or inferior leads as a result of loss of electromotive forces in the infarcted area. Local conduction delays caused by acute ischemia can also contribute to Q wave pathogenesis in selected cases. Abnormal Q waves were once considered markers of transmural myocardial infarction, whereas subendocardial (nontransmural) infarcts were thought not to produce Q waves. However, careful experimental and clinical electrocardiographic-pathologic correlative studies have indicated that transmural infarcts can occur in the absence of abnormal Q waves. Accordingly, infarcts are better classified electrocardiographically as Q wave or non-Q wave rather than as transmural or nontransmural, based on the ECG. The findings may be somewhat
different with posterior or lateral infarction (Fig. 13-36). Loss of depolarization forces in these regions can reciprocally increase R wave amplitude in lead V1 and sometimes V2, rarely without causing diagnostic Q waves in any of the conventional leads. The differential diagnosis of prominent right precordial R waves is presented in Table 13-8.

**FIGURE 13-33** Current of injury patterns with acute ischemia. **A**, With predominant subendocardial ischemia, the resultant ST vector is directed toward the inner layer of the affected ventricle and the ventricular cavity. Overlying leads therefore record ST depression. **B**, With ischemia involving the outer ventricular layer (transmural or epicardial injury), the ST vector is directed outward. Overlying leads record ST-segment elevation. Reciprocal ST-segment depression can appear in contralateral leads.

**FIGURE 13-34** Hyperacute phase of extensive anterolateral myocardial infarction. Marked ST-segment elevation melding with prominent T waves is present across the precordium, as well as in leads I and aVL. ST-segment depression, consistent with a reciprocal change, is seen in leads III and aVF. Q waves are present in leads V3 through V6. Marked ST-segment elevations with tall T waves caused by severe ischemia are sometimes referred to as a monophasic current of injury pattern. A paradoxical increase in R wave amplitude (V2 and V3) may accompany this pattern. This tracing also shows left axis deviation with small or absent inferior R waves, which raises the possibility of a prior inferior infarct.

**EVOLUTION OF ELECTROCARDIOGRAPHIC CHANGES.** Ischemic ST-segment elevation and hyperacute T wave changes may occur as the earliest sign of acute infarction (STEMI) and are typically followed within a period ranging from hours to days by evolving T wave inversion and sometimes Q waves in the same lead distribution (see Fig. 13-35 and Chap. 54). T wave inversion from evolving or chronic ischemia correlates with increased ventricular action potential duration, and these ischemic changes are often associated with QT prolongation. The T wave inversions can resolve after days or weeks, or persist indefinitely. The extent of the infarct may be an important determinant of T wave evolution. In one series, T waves that were persistently negative for more than 1 year in leads with Q waves were associated with a transmural infarction with fibrosis of the entire wall; in contrast, T waves that were positive in leads with Q waves correlated with nontransmural infarction, with viable myocardium within the wall.

**TABLE 13-8** Differential Diagnosis of Tall R Waves in Leads V1 and V2

<table>
<thead>
<tr>
<th>Physiologic and Positional Factors</th>
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</thead>
<tbody>
<tr>
<td>Misplacement of chest leads</td>
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<tr>
<td>Normal variants</td>
</tr>
<tr>
<td>Displacement of heart toward right side of chest (dextroversion), congenital or acquired</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Myocardial Injury</th>
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</thead>
<tbody>
<tr>
<td>Lateral or “true posterior” myocardial infarction (see Fig. 13-39)</td>
</tr>
<tr>
<td>Duchenne muscular dystrophy (see Chap. 92)</td>
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</tbody>
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<table>
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<tr>
<th>Ventricular Enlargement</th>
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</thead>
<tbody>
<tr>
<td>Right ventricular hypertrophy (usually with right axis deviation)</td>
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<tr>
<td>Hypertrophic cardiomyopathy</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Altered Ventricular Depolarization</th>
</tr>
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<tbody>
<tr>
<td>Right ventricular conduction abnormalities</td>
</tr>
<tr>
<td>Wolff-Parkinson-White patterns (caused by posterior or lateral wall preexcitation)</td>
</tr>
</tbody>
</table>

FIGURE 13-35  Sequence of depolarization and repolarization changes with acute anterior-lateral (A) and acute inferior wall (B) Q wave infarctions. With anterior-lateral infarcts, ST-segment elevation in leads I, aVL, and the precordial leads can be accompanied by reciprocal ST-segment depression in leads II, III, and aVF. Conversely, acute inferior (or posterior) infarcts can be associated with reciprocal ST-segment depression in leads V1 to V3. (Modified from Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 6th ed. St. Louis, CV Mosby, 1999.)

FIGURE 13-36  Evolving inferoposterolateral infarction. Note the prominent Q waves in II, III, and aVF, along with ST-segment elevation and T wave inversion in these leads, as well as V3 through V6. ST depression in I, aVL, V1, and V2 is consistent with a reciprocal change. Relatively tall R waves are also present in V1 and V2.

In the days to weeks or longer following infarction, the QRS changes can persist or begin to resolve. Complete normalization of the ECG following Q wave infarction is uncommon but can occur, particularly with smaller infarcts and when the left ventricular ejection fraction and regional wall motion improve. This is usually associated with spontaneous recanalization or good collateral circulation and is a positive prognostic sign. In contrast, persistent Q waves and ST-segment elevation several weeks or more after an infarct correlate strongly with a severe underlying wall motion disorder (akinetic or dyskinetic zone), although not necessarily a frank ventricular aneurysm. The presence of an rSR′ or similar complex in the midleft chest leads or lead I is another reported marker of ventricular aneurysm.

**OTHER ISCHEMIC ST-T PATTERNS.** Reversible transmural ischemia caused, for example, by coronary vasospasm may result in very transient ST-segment elevation (Fig. 13-37). This pattern is the classic electrocardiographic marker of Prinzmetal variant angina (see Chaps. 56 and 57). Depending on the severity and duration of such noninfarction ischemia, the ST-segment elevation can either resolve completely within minutes or be followed by T wave inversion that can persist for hours or even days. Some patients with ischemic chest pain have deep coronary T wave inversion in multiple precordial leads (e.g., V1 through V5), with or without cardiac enzyme level elevations. This finding is typically caused by severe ischemia associated with a high-grade stenosis in the proximal left anterior descending (LAD)
coronary artery system (referred to as the LAD-T wave pattern or Wellens T waves). The T wave inversion can actually be preceded by a transient ST-segment elevation that resolves by the time the patient arrives at the hospital. These T wave inversions, in the setting of unstable angina, can correlate with segmental hypokinesis of the anterior wall and suggest a myocardial stunning syndrome. The natural history of this syndrome is unfavorable, with a high incidence of recurrent angina and myocardial infarction. On the other hand, patients whose baseline ECG already shows abnormal T wave inversion can experience paradoxical T wave normalization (pseudonormalization) during episodes of acute transmural ischemia (Fig. 13-38). The four major classes of acute electrocardiographic–coronary artery syndromes in which myocardial ischemia leads to different electrocardiographic findings are summarized in Figure 13-39.

Ischemic U Wave Changes
Alterations in U wave amplitude or polarity have been reported with acute ischemia or infarction. For example, exercise-induced transient inversion of precordial U waves has been correlated with severe stenosis of the left anterior descending coronary artery. Rarely, U wave inversion can be the earliest electrocardiographic sign of acute coronary syndromes.

FIGURE 13-37  A, Prinzmetal angina with ST segment and T wave alternans. B, ST segment and T wave alternans associated with nonsustained ventricular tachycardia. (Courtesy of Dr. C. Fisch.)

FIGURE 13-38  Pseudo (paradoxical) T wave normalization. A, Baseline ECG of a patient with coronary artery disease shows ischemic T wave inversion. B, T wave "normalization" during an episode of ischemic chest pain. C, Following resolution of the chest pain, the T waves have reverted to their baseline appearance. (From Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis. 4th ed. St. Louis, Mosby-Year Book, 1991.)
QT INTERVAL DISPERSION. The effects of acute myocardial ischemia and infarction on the disparity among QT intervals in various electrocardiographic leads, referred to as QT dispersion, have generated interest. The greater the difference between maximum and minimum QT intervals—that is, increased QT dispersion—the greater the variability in myocardial repolarization. An increased index has been proposed as a marker of arrhythmia risk after myocardial infarction and as a marker of acute ischemia with atrial pacing. The practical usefulness of QT dispersion measurements in patients with coronary syndromes appears limited, despite initial enthusiasm.

LOCALIZATION OF ISCHEMIA OR INFARCTION. The electrocardiographic leads are more helpful in localizing regions of transmural than subendocardial ischemia. As examples, ST-segment elevation and/or hyperacute T waves are seen in the following: (1) two or more contiguous precordial leads (V1 through V3) and/or in leads I and aVL with acute transmural anterior or anterolateral wall ischemia; (2) leads V1 to V4 with anteroseptal or apical ischemia; (3) leads V5 to V6 with apical or lateral ischemia; (4) leads II, III, and aVF with inferior wall ischemia; and (5) right-sided precordial leads with right ventricular ischemia. Posterior wall infarction, which induces ST-segment elevation in leads placed over the back of the heart, such as leads V1 to V6,

New Q waves usually preceded by hyperacute T waves/ST elevations, followed by T wave inversions

Non–Q-wave/non-ST elevation infarction

ST elevations

Q-wave infarction

QT INTERVAL DISPERSION. The effects of acute myocardial ischemia and infarction on the disparity among QT intervals in various electrocardiographic leads, referred to as QT dispersion, have generated interest. The greater the difference between maximum and minimum QT intervals—that is, increased QT dispersion—the greater the variability in myocardial repolarization. An increased index has been proposed as a marker of arrhythmia risk after myocardial infarction and as a marker of acute ischemia with atrial pacing. The practical usefulness of QT dispersion measurements in patients with coronary syndromes appears limited, despite initial enthusiasm.

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The ECG can also provide more specific information about the location of the occlusion within the coronary system (the culprit lesion). In patients with an inferior wall myocardial infarction, the presence of ST-segment elevation in lead III exceeding that in lead II, particularly when combined with ST-segment elevation in lead V1, is a useful predictor of occlusion in the proximal to midportion of the right coronary artery (Fig. 13-40). In contrast, the presence of ST-segment elevation in lead II equal to or exceeding that in lead III, especially in concert with ST-segment depression in leads V1 to V3 or ST-segment elevation in leads I and aVL, suggests occlusion of the left circumflex coronary artery or a distal occlusion of a dominant right coronary artery. Right-sided ST-segment elevation is indicative of acute right ventricular injury and usually indicates occlusion of the proximal right coronary artery. Of note is the finding that acute right ventricular infarction can project an injury current pattern in leads V1 through V3 or even V6, thereby simulating anterior infarction. In other cases, simultaneous ST-segment elevation in V1 (V3R) and ST-segment depression in V2 (V4R) can occur (see Fig. 13-40). Lead aVR may provide important clues to artery occlusion in myocardial infarction (MI). Left main (or severe multivessel) disease should be considered when leads aVR and V1 show ST-segment elevation, especially in concert with diffuse prominent ST depression in other leads.

These and other criteria proposed for localization of the site of coronary occlusion based on the initial ECG still require additional validation in larger populations. Current and future criteria will always be subject to limitations and exceptions based on variations in coronary anatomy, the dynamic nature of acute electrocardiographic changes, the presence of multivessel involvement, collateral flow, and the presence of ventricular conduction delays.

For example, in some cases, ischemia can affect more than one region of the myocardium (e.g., infero-lateral; see Fig. 13-35). Not uncommonly, the ECG will show the characteristic findings of involvement in each region. Sometimes, however, partial normalization can result from cancellation of opposing vectorial forces. Inferior lead ST-segment elevation accompanying acute anterior wall infarction suggests either occlusion of a left anterior descending artery that extends onto the inferior wall of the left ventricle (the wrap-around vessel) or multivessel disease with jeopardized collaterals.

ELECTROCARDIOGRAPHIC DIAGNOSIS OF BUNDLE BRANCH BLOCKS AND MYOCARDIAL INFARCTION. The diagnosis of MI is often more difficult in cases in which the baseline ECG shows a bundle branch block pattern, or a bundle branch block develops as a complication of the infarct. The diagnosis of Q wave infarction is not usually impeded by the presence of RBBB, which affects primarily the terminal phase of ventricular depolarization. The net effect is that the criteria for the diagnosis of a Q wave infarct in a patient with RBBB are the same as in patients with normal conduction (Fig. 13-41). The diagnosis of infarction in the presence of LBBB is considerably more complicated and confusing, because LBBB alters the early and the late phases of ventricular depolarization and produces secondary ST-T changes. These changes may mask and/or mimic MI findings. As a result, considerable attention has been directed to the problem of diagnosing acute and chronic MI in patients with LBBB (Fig. 13-42).

Infarction of the left ventricular free (or lateral) wall ordinarily results in abnormal Q waves in the midprecordial to lateral precordial leads (and selected limb leads). However, the initial septal depolarization forces with LBBB are directed from right to left. These leftward forces produce an initial R wave in the midprecordial to lateral precordial leads, usually skewing the loss of electrical potential (Q waves) caused by the infarction. Therefore, acute or chronic left ventricular free wall infarction by
FIGURE 13-40  Acute right ventricular infarction with acute inferior wall infarction. Note the ST-segment elevation in the right precordial leads, as well as in leads II, III, and aVF, with reciprocal changes in leads I and aVL. ST-segment elevation in lead III greater than in lead II and right precordial ST-segment elevation are consistent with proximal to middle occlusion of the right coronary artery. The combination of ST-segment elevation in conventional lead V₁ (V₁R here) and ST-segment depression in lead V₆ (lead V₆R here) has also been reported with acute right ventricular ischemia or infarction.

FIGURE 13-41  RBBB with acute anterior infarction. Loss of anterior depolarization forces results in QR-type complexes in the right precordial to midprecordial leads, with ST-segment elevations and evolving T wave inversions (V₁ through V₆).

Itself will not usually produce diagnostic Q waves in the presence of LBBB. Acute or chronic infarction involving both the free wall and septum (or the septum itself) may produce abnormal Q waves (usually as part of QR-type complexes) in leads V₁ to V₆. These initial Q waves probably reflect posterior and superior forces from the spared basal portion of the septum (Fig. 13-43). Thus, a wide Q wave (40 msec) in one or more of these leads is a reliable sign of underlying infarction. The sequence of repolarization is also altered in LBBB, with the ST-segment and T wave vectors being directed opposite the QRS complex. These changes can mask or simulate the ST segment changes of actual ischemia.

The following points summarize the ECG signs of myocardial infarction in LBBB:

1. ST-segment elevation with tall, positive T waves is frequently seen in the right precordial leads with uncomplicated LBBB. Secondary T wave inversions are characteristically seen in the lateral precordial leads. However, the appearance of ST-segment elevations in the lateral leads or ST-segment depressions or deep T wave inversions in leads V₁ to V₆ strongly suggests underlying ischemia. More marked ST-segment elevations (>0.5 mV) in leads with QS or rS waves may also be caused by acute ischemia, but false-positive findings occur, especially with large-amplitude negative QRS complexes.

2. The presence of QR complexes in leads I, V₅, or V₆ or in II, III, and aVF with LBBB strongly suggests underlying infarction.

3. Chronic infarction is also suggested by notching of the ascending part of a wide S wave in the midprecordial leads (Cabrera sign) or the ascending limb of a wide R wave in lead I, aVL, V₅, or V₆ (Chapman sign).

Similar principles can apply to the diagnosis of acute and chronic infarction in the presence of right ventricular pacing. Comparison between an ECG exhibiting the LBBB prior to the infarction and the present ECG is often helpful to show these changes.

The diagnosis of concomitant LAFB and inferior wall infarction can also pose challenges. This combination can result in loss of the small r waves in the inferior leads, so that leads II, III, and aVF show QS, not rS, complexes. LAFB, however, will occasionally hide the diagnosis of inferior wall infarction. The inferior orientation of the initial QRS forces caused by the fascicular block can mask inferior Q waves, with resultant rS complexes in leads II, III, and aVF. In other cases, the combination of LAFB and inferior wall infarction will produce qS complexes in the inferior limb leads, with the initial q wave the result of the infarct and the minuscule r wave the result of the fascicular block.

Atrial Infarction. A number of ECG clues to the diagnosis of atrial infarction have been suggested, including localized deviations of the PR segment (e.g., PR elevation in lead V₁ or V₆ or the inferior leads), changes in P wave morphology, and atrial arrhythmias. The sensitivity and specificity of these signs are limited, however. Diffuse PR-segment changes (PR elevation in aVR with depression in the inferolateral leads) with acute ventricular infarction usually indicate concomitant pericarditis (see later).

Electrocardiographic Differential Diagnosis of Ischemia and Infarction. The ECG has important limitations in sensitivity and specificity in the diagnosis of coronary syndromes.
An initially normal ECG does not exclude ischemia or even acute infarction. If the initial ECG is not diagnostic, but the patient remains symptomatic and there is high clinical suspicion for acute ischemia, it is recommended that the ECG be repeated at 5- to 10-minute intervals. However, a normal ECG throughout the course of an alleged acute infarct is distinctly uncommon. As a result, prolonged chest pain without diagnostic electrocardiographic changes on repeat ECGs should always prompt a careful search for noncoronary causes of chest pain (see Chap. 53). Pathologic Q waves can be absent, even in patients with depressed left ventricular function caused by severe coronary disease and a previous infarct. As noted, the diagnosis of acute or chronic infarction can be completely masked by ventricular conduction disturbances, especially those resulting from LBBB, as well as ventricular pacing and Wolff-Parkinson-White preexcitation. On the other hand, diagnostic confusion can arise because Q waves, ST-segment elevation, ST-segment depression, tall positive T waves, and deep T wave inversion can be seen in a wide variety of noncoronary settings.

**NONINFARCTION Q WAVES.** Q waves simulating coronary artery disease can be related to one (or a combination) of the following four factors (Table 13-9): (1) physiologic or positional variants; (2) altered ventricular conduction; (3) ventricular enlargement; and (4) myocardial damage or replacement. Depending on the electrical axis, prominent Q waves (as part of QS- or QR-type complexes) can also appear in the limb leads (aVL with a vertical axis and III and aVF with a horizontal axis). A QS complex can appear in lead V1 as a normal variant, but rarely in leads V1 and V2. Prominent Q waves can be associated with a variety of other positional factors that alter the orientation of the heart vis-à-vis a given lead axis. Poor R wave progression, sometimes with actual QS waves, can be caused solely by improper placement of chest electrodes above their usual position. In cases of dextrocardia, provided that no underlying structural abnormalities are present, normal R wave progression can be restored by recording leads V2 to V6 on the right side of the chest (with lead V6 placed in the V2 position). A rightward mediastinal shift in the left pneumothorax can contribute to the apparent loss of left precordial R waves. Other positional factors associated with slow R wave progression include pectus excavatum and congenitally corrected transposition of the great vessels.

An intrinsic change in the sequence of ventricular depolarization can lead to pathologic, noninfarct Q waves. The two most important conduction disturbances associated with pseudoinfarct Q waves are LBBB and the Wolff-Parkinson-White (WPW) preexcitation patterns. With LBBB, QS complexes can appear in the right precordial to midprecordial leads and, occasionally, in one or more of leads II, III, and aVF. Depending on the location of the bypass tract, WPW preexcitation can mimic anteroseptal,
lateral, or inferior-posterior infarction. LAFB is often cited as a cause of anterosepal infarct patterns; however, LAFB usually has only minor effects on the QRS complex in horizontal plane leads. Probably the most common findings are relatively prominent S waves in leads V₅ and V₆. Slow R wave progression is not a consistent feature of LAFB, although minuscule q waves in leads V₁ to V₃ have been reported in this setting. These small q waves can become more apparent if the leads are recorded one interspace above their usual position and disappear in leads that are one interspace below their usual position. As a general clinical rule, however, prominent Q waves (as part of QS or QR complexes) in the right precordial to midprecordial leads should not be attributed to LAFB alone.

**Slow (“Poor”) R Wave Progression**

In contrast, slow R wave progression, a nonspecific finding, is commonly observed with LVH and with acute or chronic right ventricular overload. Q waves in such settings can reflect a variety of mechanisms, including a change in the balance of early ventricular depolarization forces and altered cardiac geometry and position. A marked loss of R wave voltage, sometimes with frank Q waves from lead V₁ to the lateral chest leads, can be seen with chronic obstructive pulmonary disease (see Fig. 13-20). The presence of low limb voltage and signs of right atrial abnormality (P pulmonale) can serve as additional diagnostic clues. This loss of R wave progression may, in part, reflect right ventricular dilation. Furthermore, downward displacement of the heart in an emphysematous chest can play a major role in the genesis of poor R wave progression in this syndrome. Partial or complete normalization of R wave progression can be achieved in these cases simply by recording the chest leads an interspace lower than usual (see Fig. 13-20).

**Other Pseudoinfarct Patterns in Ventricular Overload.** A variety of pseudoinfarct patterns can occur with acute cor pulmonale caused by pulmonary embolism (see Chap. 77). Acute right ventricular overload in this setting can cause slow R wave progression and sometimes right precordial to midprecordial T wave inversion (formerly referred to as right ventricular strain), mimicking anterior ischemia or infarction. The classic S₁ Q₃ T₃ pattern can occur but is neither sensitive nor specific. A prominent Q wave (usually as part of a QR complex) can also occur in lead aVF along with this pattern (see Fig. 13-21). However, acute right overload by itself does not cause a pathologic Q wave in lead II. Right heart overload, acute or chronic, may also be associated with a QR complex in lead V₁ and simulate anterosepal infarction.

Pseudoinfarct patterns are an important finding in patients with hypertrophic cardiomyopathy, and the ECG can simulate anterior, inferior, posterior, or lateral infarction. The pathogenesis of depolarization abnormalities in this cardiomyopathy is not certain. Prominent inferolateral Q waves (leads II, III, aVF, and V₉ to V₁₂) and tall, right precordial R waves are probably related to increased depolarization forces generated by the markedly hypertrophied septum (Fig. 13-44). Abnormal septal depolarization can also contribute to bizarre QRS complexes.

**Q Wave Pathogenesis with Myocardial Damage.** Loss of electromotive force associated with myocardial necrosis contributes to R wave loss and Q wave formation in MI cases. This mechanism of Q wave pathogenesis, however, is not specific for coronary artery disease with infarction. Any process, acute or chronic, that causes sufficient loss of regional electromotive potential can result in Q waves. For example, replacement of myocardial tissue by electrically inert material such as amyloid or tumor can cause noninfarction Q waves (see Chap. 74). A variety of dilated cardiomyopathies associated with extensive myocardial fibrosis can be characterized by pseudoinfarct patterns. Ventricular hypertrophy can also contribute to Q wave pathogenesis in this setting. Finally, Q waves caused by myocardial injury, whether ischemic or nonischemic in origin, can appear transiently and do not necessarily signify irreversible heart muscle damage. Severe ischemia can cause regional loss of electromotive potential without actual cell death (electrical stunning phenomenon). Transient conduction disturbances can also cause alterations in ventricular activation and result in noninfarctional Q waves. In some cases, transient Q waves may represent unmasking of a prior Q wave infarct. New but transient Q waves have been described in patients with severe hypotension from a variety of causes, as well as with tachyarhythmias, myocarditis, Prinzmetal angina, protracted hypoglycemia, phosphorus poisoning, and hyperkalemia.

**Fragmented QRS**

Because Q waves are not always present and can regress or even disappear over time, alternative electrocardiographic markers of prior infarction are being evaluated, including a fragmented QRS complex.
Acute pericarditis is often characterized by two apparent injury currents, one atrial and the other ventricular. The atrial injury current vector (ST<sub>a</sub>) is usually directed upward and to the right and produces PR-segment elevation in aVR, with reciprocal PR depression in II, aVL, and V<sub>6</sub>. The ventricular injury current (ST<sub>v</sub>) is directed downward and to the left, associated with ST-segment elevation in leads II, V<sub>5</sub>, and V<sub>6</sub>. This characteristic PR-ST segment discordance is illustrated in the bottommost tracing. Note the diffuse distribution of ST-segment elevation in acute pericarditis (e.g., I, II, and V<sub>2</sub> through V<sub>6</sub>, with reciprocal changes in aVR and perhaps minimally in V<sub>1</sub>). (From Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis. 4th ed. St. Louis, Mosby-Year Book, 1991.)

**TABLE 13-10** Differential Diagnosis of ST-Segment Elevation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial ischemia or infarction</td>
<td>Noninfarction, transmural ischemia (e.g., Prinzmetal angina pattern, takotsubo syndrome; see Fig. 13-40)</td>
</tr>
<tr>
<td>Post myocardial infarction</td>
<td>(ventricular aneurysm pattern)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>(see Fig. 13-37)</td>
</tr>
<tr>
<td>Normal variants</td>
<td>(including the classic early repolarization pattern; see Fig. 13-16)</td>
</tr>
<tr>
<td>LVH, LBBB (V&lt;sub&gt;1&lt;/sub&gt;-V&lt;sub&gt;4&lt;/sub&gt; only)</td>
<td></td>
</tr>
<tr>
<td>Other (rarer)</td>
<td>Acute pulmonary embolism (right midchest leads)</td>
</tr>
<tr>
<td>Hypothermia (J wave, Osborn wave)</td>
<td>Myocardial injury</td>
</tr>
<tr>
<td>Myocarditis (may resemble myocardial infarction or pericarditis)</td>
<td>Tumor invading the left ventricle</td>
</tr>
<tr>
<td>Hyperkalemia*</td>
<td>Hypothermia (U wave, Osborn wave)</td>
</tr>
<tr>
<td>Other (rarer)</td>
<td>DC cardioversion (just following)</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
<td>Hyperkalemia*</td>
</tr>
<tr>
<td>Brugada pattern (RBBB-like pattern and ST-segment elevations in right precordial leads)*</td>
<td></td>
</tr>
<tr>
<td>Other (rarer)</td>
<td>Type 1C antiarrhythmic drug*</td>
</tr>
<tr>
<td>Hyperkalemia*</td>
<td></td>
</tr>
</tbody>
</table>

resulting from mitral or aortic regurgitation, among other causes. ST-segment elevation, J point elevations, and tall positive T waves are also common findings in leads V<sub>1</sub> and V<sub>2</sub>. Some adults show persistence of the juvenile T wave pattern (see Fig. 13-12), with more prominent T wave inversion in right precordial to midprecordial leads showing an R<sub>S</sub> or RS morphology. Such
patterns, especially associated with LBBB-type premature ventricular beats or relevant family history, also raise strong consideration of arrhythmogenic right ventricular cardiomyopathy (dysplasia).\textsuperscript{25-27} The other major normal variant that can be associated with notable T wave inversion is the early repolarization pattern (see Fig. 13-13). Some subjects, especially athletes, with this variant have prominent, biphasic T wave inversion in association with the ST-segment elevation. This pattern, which may simulate the initial stages of an evolving infarct, is most prevalent in young black men and athletes. These functional ST changes are probably the result of regional disparities in repolarization and can be normalized by exercise.

**Primary and Secondary T Wave Inversions**

A variety of pathologic factors can alter repolarization and cause prominent T wave inversion (Fig. 13-46). As noted earlier, T wave alterations are useful classified as primary or secondary. Primary T wave changes are caused by alterations in the duration or morphology of ventricular action potentials in the absence of changes in the activation sequence. Examples include ischemia, drug effects, and metabolic factors. Prominent primary T wave inversion (or in some cases, tall positive T waves) is also a well-described feature of the ECG in cerebrovascular accidents, particularly with subarachnoid hemorrhage. The so-called cerebrovascular accident (CVA) T wave pattern is characteristically seen in multiple leads, with a widely splayed appearance usually associated with marked QT prolongation (see Figs. 13-46 and 92-18). Some studies have implicated structural damage (myocytolysis) in the hearts of patients with such T wave changes, probably induced by excessive sympathetic stimulation mediated via the hypothalamus. A role for concomitant vagal activation in the pathogenesis of such T wave changes, which are usually associated with bradyarrhythmia, has also been postulated. Similar T wave changes have been reported after truncal vagotomy, radical neck dissection, and bilateral carotid endarterectomy. In addition, the massive diffuse T wave inversion seen in some patients after Stokes-Adams syncope may be related to a similar neurogenic mechanism. Patients with subarachnoid hemorrhage can also show transient ST elevation, as well as arrhythmias, including torsades de pointes. Ventricular dysfunction can also occur.

In contrast to these primary T wave abnormalities, secondary T wave changes are caused by altered ventricular activation, without changes in action potential characteristics. Examples include bundle branch block, Wolf-Parkinson-White preexcitation, and ventricular ectopic or paced beats. In addition, altered ventricular activation (associated with QRS interval prolongation) can induce persistent T wave changes that appear after normal ventricular depolarization has resumed. The term cardiac memory T wave changes has been used in this context to describe repolarization changes subsequent to depolarization changes caused by ventricular pacing, intermittent LBBB, intermittent Wolf-Parkinson-White preexcitation, and other alterations of ventricular activation\textsuperscript{28} (see Chaps. 36 and 39). Finally, the term idiopathic global T wave inversion has been applied in cases in which no identifiable cause for often marked diffuse repolarization abnormalities can be found. An unexplained female preponderance has been reported.

**Drug Effects**

Numerous drugs can affect the ECG, often in association with nonspecific STT alterations.\textsuperscript{52,53,58} More marked changes, as well as AV and intraventricular conduction disturbances, can occur with selected agents. The proarrhythmic effects of antiarrhythmic medications are described in Chaps. 10 and 37.

The digitalis effect\textsuperscript{59} refers to the relatively distinctive “scooped” appearance of the ST-T complex and shortening of the QT interval, which correlates with abbreviation of the ventricular action potential duration (Fig. 13-47). Digitalis-related ST-T changes can be accentuated by an increased heart rate during exercise and result in false-positive stress test results (see Chap. 14). Digitalis effect can occur with therapeutic or toxic doses of the drug. The term digitalis toxicity refers specifically to systemic effects (nausea and anorexia, among other effects) or conduction disturbances and arrhythmias caused by drug excess or increased sensitivity.

The electrocardiographic effects and toxicities of other cardiovascular agents can be anticipated, in part, from ion channel effects (see Chap. 35). Inactivation of sodium channels by class 1 agents (e.g., quinidine, procainamide, disopyramide, flecaïnide) can cause QRS prolongation. Classes 1A and 3 agents (e.g., amiodarone, dronedarone, dofetilide, ibutilide, sotalol) can induce an acquired long QT(U) syndrome (see Chap. 37). Psychotropic drugs (e.g., tricyclic antidepressants and phenothiazines), which have class 1A-like properties, can also lead to QRS and QT(U) prolongation. Toxicity can produce astysole or torsades de pointes. Right axis shift of the terminal 40 msec frontal plane QRS axis may be a helpful additional marker of tricyclic antidepressant overdose. QT prolongation has been reported with methadone, as discussed below in the section on ECG Guidelines. Cocaine (see Chap. 73) can cause a variety of ECG changes including those of STEMI, as well as life-threatening arrhythmias.

**Electrolyte and Metabolic Abnormalities**

In addition to the structural and functional cardiac conditions already discussed, numerous systemic metabolic aberrations affect the ECG, including electrolyte abnormalities and acid-base disorders, as well as systemic hypothermia.\textsuperscript{52,53}

**CALCIUM.** Hypercalcemia and hypocalcemia predominantly alter the action potential duration. An increased extracellular calcium concentration shortens the ventricular action potential duration by shortening phase 2 of the action potential. In contrast, hypocalcemia prolongs phase 2 of the action potential. These cellular changes correlate with abbreviation and prolongation of the QT interval (ST segment portion) with hypercalcemia and hypocalcemia, respectively (Fig. 13-48). Severe hypercalcemia (e.g., serum Ca\textsuperscript{2+} >15 mg/dL) can also be associated with decreased T wave amplitude, sometimes with T wave notching or inversion. Hypercalcemia sometimes produces a high takeoff of the ST segment in leads V\textsubscript{1} and V\textsubscript{2} and can thus simulate acute ischemia (see Table 13-10).
POTASSIUM. Hyperkalemia is associated with a distinctive sequence of ECG changes (Fig. 13-49A). The earliest effect is usually narrowing and peaking (tenting) of the T wave. The QT interval is shortened at this stage, associated with decreased action potential duration. Progressive extracellular hyperkalemia reduces atrial and ventricular resting membrane potentials, thereby inactivating sodium channels, which decreases \( V_{m0} \) and conduction velocity. The QRS begins to widen and P wave amplitude decreases. PR interval prolongation can occur, followed sometimes by second- or third-degree AV block. Complete loss of P waves may be associated with a junctional escape rhythm or so-called sino-ventricular rhythm. In the latter instance, sinus rhythm persists with conduction between the SA and AV nodes and occurs without producing an overt P wave. Moderate to severe hyperkalemia occasionally induces ST elevations in the right precordial leads (V₁ and V₂) and simulates an ischemic current of injury or Brugada-type patterns.

However, even severe hyperkalemia can be associated with atypical or nondiagnostic ECG findings. Very marked hyperkalemia leads to eventual asystole, sometimes preceded by a slow undulatory (“sine-wave”) ventricular flutter-like pattern. The electrocardiographic triad of (1) peaked T waves (from hyperkalemia), (2) QT prolongation (from hypocalcemia), and (3) LVH (from hypertension) is strongly suggestive of chronic renal failure (see Chap. 93).

Electrophysiologic changes associated with hypokalemia, in contrast, include hyperpolarization of myocardial cell membranes and increased action potential duration. The major electrocardiographic manifestations are ST depression with flattened T waves and increased U wave prominence (see Fig. 13-49B). The U waves can exceed the amplitude of T waves. Clinically, distinguishing T waves from U waves can be difficult or impossible from the surface ECG. Indeed, apparent U waves in hypokalemia and other pathologic settings may actually be part of T waves whose morphology is altered by the effects of voltage gradients between M, or midmyocardial, cells, and adjacent myocardial layers. The prolongation of repolarization with hypokalemia, as part of an acquired long QT(U) syndrome, predisposes to torsades de pointes. Hypokalemia also predisposes to tachyarrhythmias from digitals.

MAGNESIUM. Specific electrocardiographic effects of mild to
moderate isolated abnormalities in magnesium ion concentration are not well characterized. Severe hypermagnesemia can cause AV and intraventricular conduction disturbances that may culminate in complete heart block and cardiac arrest (Mg²⁺ > 15 mEq/L). Hypomagnesemia is usually associated with hypocalcemia or hypokalemia. Hypomagnesemia can potentiate certain digitalis toxic arrhythmias. The role of magnesium deficiency in the pathogenesis and treatment of the acquired long QT(U) syndrome with torsades de pointes is discussed in Chaps. 9 and 39.

**OTHER FACTORS.** Isolated hypernatremia or hyponatremia does not produce consistent effects on the ECG. Acidemia and alkalemia are often associated with hyperkalemia and hypokalemia, respectively. Systemic hypothermia may be associated with the appearance of a distinctive convex elevation at the junction (J point) of the ST segment and QRS complex (J wave or Osborn wave; Fig. 13-50). The cellular mechanism of this type of pathologic J wave appears to be related to an epicardial-endocardial voltage gradient associated with the localized appearance of a prominent epicardial action potential notch.

### Nonspecific QRS and ST-T Changes

Low QRS voltage is said to be present when the total amplitude of the QRS complexes in each of the six extremity leads is 0.5 mV or less or 1.0 mV or less in leads V₁ through V₆. Low QRS voltage can relate to a variety of mechanisms, including increased insulation of the heart by air (chronic obstructive pulmonary disease) or adipose tissue (obesity); replacement of myocardium, for example, by fibrous tissue (ischemic or nonischemic cardiomyopathy), amyloid, or tumor; or to short-circuiting (shunting) effects resulting from low resistance of the fluids (especially with pericardial or pleural effusions, or anasarca). The combination of relatively low limb voltage (QRS voltage < 0.8 mV in each of the limb leads), relatively prominent QRS voltage in the chest leads (SV₁ or SV₂ + RV₅ or RV₆ > 3.5 mV), and slow R wave progression (R wave less than the S wave amplitude in V₁ through V₆) has been reported as a relatively specific but not sensitive sign of dilated-type cardiomyopathies (referred to as the ECG—congestive heart failure triad).

Many factors in addition to ischemia (e.g., postural changes, meals, drugs, hypertrophy, electrolyte and metabolic disorders, central nervous system lesions, infections, pulmonary diseases) can affect the ECG. Ventricular repolarization is particularly sensitive to these effects, which can lead to a variety of nonspecific ST-T changes. The term is usually applied to slight ST depression or T wave inversion or to T wave flattening without evident cause. Care must be taken not to overinterpret such changes, especially in subjects with a low prior probability of heart disease. At the same time, subtle repolarization abnormalities can be markers of coronary or hypertensive heart disease or other types of structural heart disease; these probably account for the association of relatively minor but persistent nonspecific ST-T changes with increased cardiovascular mortality in middle-aged men and women.

### Alternans Patterns

The term alternans applies to conditions characterized by the sudden appearance of a periodic beat-to-beat change in some aspect of cardiac electrical or mechanical behavior. These abrupt (period-doubling) changes (AAAA > ABAB pattern) are reminiscent of a generic class of patterns observed in perturbed nonlinear control systems. Many different examples of electrical alternans have been described clinically; a number of others have been reported in the laboratory. Most familiar is total electrical alternans with sinus tachycardia, a specific but not highly sensitive marker of pericardial effusion with tamponade physiology (Fig. 13-51; see Chap. 75). This finding is associated with an abrupt transition from a 1:1 to a 2:1 pattern in the “to-fro” swinging motion of the heart in the effusion (see Fig. 15-72). Other alternans patterns have primary electrical rather than mechanical causes. ST-T alternans has long

![FIGURE 13-49](Image)

Electrocardiographic changes in hyperkalemia (A) and hypokalemia (B). A, On day 1, at a K⁺ level of 8.6 mEq/liter, the P wave is no longer recognizable and the QRS complex is diffusely prolonged. Initial and terminal QRS delays are characteristic of K⁺-induced intraventricular conduction slowing and are best illustrated in leads V₅ and V₆. On day 2, at a K⁺ level of 5.8 mEq/liter, the P wave is recognizable, with a PR interval of 0.24 second; the duration of the QRS complex is approximately 0.10 second, and the T waves are characteristically “tented.” B, On day 1, at a K⁺ level of 1.5 mEq/liter, the T and U waves are merged. The U wave is prominent and the QU interval is prolonged. On day 4, at a K⁺ level of 3.7 mEq/liter, the tracing is normal. (Courtesy of Dr. C. Fisch.)

![FIGURE 13-50](Image)

Systemic hypothermia. The arrowheads (leads V₁ through V₆) point to the characteristic convex J waves, termed Osborn waves. Prominent sinus bradycardia is also present. (From Goldberger AL. Clinical Electrocardiography: A Simplified Approach. 6th ed. St. Louis, CV Mosby, 1999.)
organizations; these are described and discussed in the section at the end of this chapter.

Whereas most reviews tend to focus on preventing overuse, the ECG may be underused in other important clinical situations. For example, over one third of patients evaluated for angina pectoris in outpatient settings do not have an ECG recorded and only one fourth of patients with ST-segment elevation myocardial infarction transported to emergency rooms have a prehospital ECG, with resulting delays in revascularization procedures.

**Technical Errors and Artifacts**

Technical errors can lead to clinically significant diagnostic mistakes. Artifacts that may interfere with interpretation can come from movement of the patient or electrodes, electrical disturbances related to current leakage and grounding failure, and external sources such as electrical stimulators or cauteries. Electrical artifacts can simulate life-threatening arrhythmias (Fig. 13-53), and excessive body motion can cause excessive baseline wander that could simulate an ST-segment shift of myocardial ischemia or injury.

Misplacement of one or more electrodes is a common cause for errors in interpretation of the ECG. Many limb lead switches produce electrocardiographic patterns that can aid in their identification. Reversal of the two arm electrodes, for example, results in an inverted P and QRS waveforms in lead I but not in lead V6, two leads that would normally be expected to have similar polarities. Other lead misplacements are not as obvious.

As many as one third of ECGs are recorded with significant misplacement of precordial electrodes. The most common errors are placing the V1 and V2 electrodes in the second or third rather than in the fourth intercostal space and placing the V4 to V6 electrodes too high on the lateral chest. Placing the right precordial electrodes too high on the chest can yield patterns that mimic those produced by anterior myocardial infarction (delayed R wave progression) or an intraventricular conduction delay (e.g., rSr′ patterns in lead V1).

Another very common technical error is recording the ECG with nonstandard high- and low-pass filter settings. Increasing the low-frequency cutoff to reduce baseline wander and respiratory effects

**Clinical Issues in Electrocardiographic Interpretation**

The effectiveness of the ECG as a diagnostic tool depends on factors such as the indications for the procedure, proper recording technique, and the skills of the reader of the ECG.

**Indications for an Electrocardiogram**

Relatively limited attention has been paid to the indications for an ECG, probably because of its seeming simplicity, safety, and low cost. However, the cumulative expense of low-cost tests performed at high volume is significant, and the potential risk to the patient of missed or false diagnoses of cardiac disease can be substantial. Recommendations for performing ECGs have been proposed by various organizations; these are described and discussed in the section at the end of this chapter.

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As many as one third of ECGs are recorded with significant misplacement of precordial electrodes. The most common errors are placing the V1 and V2 electrodes in the second or third rather than in the fourth intercostal space and placing the V4 to V6 electrodes too high on the lateral chest. Placing the right precordial electrodes too high on the chest can yield patterns that mimic those produced by anterior myocardial infarction (delayed R wave progression) or an intraventricular conduction delay (e.g., rSr′ patterns in lead V1).

Another very common technical error is recording the ECG with nonstandard high- and low-pass filter settings. Increasing the low-frequency cutoff to reduce baseline wander and respiratory effects
can produce a range of artifactual ST segment abnormalities. Lowering the high-frequency cutoff to reduce motion and tremor artifacts reduces R wave amplitudes and Q wave measurements and decreases the accuracy of diagnoses of hypertrophy and infarction. Low-pass filtering may also alter electrocardiographic features such as J waves in early repolarization.

Other technical issues reflect characteristics of computerized systems. Clinically relevant differences in measurements may be reported by systems of different manufacturers and by different software versions from the same manufacturer. Other differences result from the differences in the signals used for computerized interpretation and for graphic display. For example, intervals measured by eye may be significantly shorter than those reported by software because the software determines the interval from an overlay of patterns from all leads, whereas manual methods typically rely on the analysis of the waveform from a single lead. Differences in intervals, such as the duration of the Q wave or the QRS complex, may exceed 5 msec; sufficient to alter the diagnosis of conduction defects and infarction.

**Reading Competency**

Developing and maintaining competency in interpretation of the ECG is critical to successful clinical practice. The Accreditation Council for Graduate Medical Education and the American College of Cardiology recommend supervised and documented interpretation of a minimum of 3500 ECGs covering a broad spectrum of diagnoses and clinical settings over a 3-year training period. Various tools are available to assess and improve proficiency. The Adult Clinical Cardiology Self-Assessment Program (ACCSAP) contains a self-assessment examination in electrocardiography and is useful for identifying knowledge levels of proficiency and areas of specific weakness. The ECG Wave-Maven (http://ecg.bidmc.harvard.edu) provides free access to more than 400 case studies of ECGs, with answers and multimedia adjuncts.

**Future Perspectives**

Clinical electrocardiography represents a mature and canonical cardiovascular technology based on extensive electrophysiologic and clinical correlates that have been elaborated over more than a century of study. Advances in other fields have challenged the use of the ECG. For example, imaging techniques provide a more direct assessment of cardiac structural abnormalities than the ECG. Thus, it is important to assess the role of the ECG in adding value within the overall spectrum of cardiac diagnostic tests.

Recent advances in biomedical engineering and technology clinical therapeutics, and basic science suggest that important new clinically relevant information may be derived from the ECG. Methods to estimate direct cardiac potentials from surface recordings will permit a more direct understanding of the abnormal physiology underlying electrocardiographic patterns, and extracting “hidden information” in the ECG using advanced mathematics techniques will expand its clinical usefulness. Full application of the capabilities of computerized systems will permit the development of more accurate diagnostic criteria based on population subsets and clinical covariates. Continued research correlating electrocardiographic patterns with genomic and biomarker patterns will promote greater understanding of the variation in electrocardiographic patterns seen in common disorders. Also, the development of new treatments will likely expand the role of the ECG as urgent revascularization and cardiac resynchronization have done in the recent past. The historic richness of the surface ECG as a source of basic physiologic and clinical information continues to support the expectation of future unanticipated areas for exploration and discovery.
Normal Electrocardiogram


Intraventricular Conduction Delays and Preexcitation


Myocardial Ischemia and Infarction


Electrocardiography

GUIDELINES

Electrocardiography Guidelines for the performance of electrocardiograms have evolved little in recent years. The most widely cited guidelines were published by the American College of Cardiology and American Heart Association (ACC/AHA) in 1992 and are summarized in Tables 13G-1 through 13G-3. Other professional groups have also published guidelines for use in specific populations and clinical settings (see later).

The ACC/AHA guidelines 1 use the convention of classifying indications according to one of three classes. Class I indications are for which it is generally agreed that ECGs are useful. Class II indications are for which opinions differ with respect to the usefulness of ECGs. Class III indications are for which it is generally agreed that ECGs have little or no usefulness.

Indications for the ECG can be considered for several different subpopulations—those with known heart disease (Table 13G-1), those with suspected heart disease or at high risk for heart disease (Table 13G-2), and those without evidence of heart disease (Table 13G-3). In addition, more specific recommendations have been proposed for the use of the ECG in special groups, including preoperative patients, persons with dangerous occupations, athletes, and those taking medications associated with electrophysiologic effects.

PATIENTS WITH KNOWN CARDIOVASCULAR DISEASE

The guidelines 1 support the use of ECGs in the baseline evaluation of all patients with known cardiovascular disease, when important clinical changes occur, for following the course of disease, and for the evaluation of response to therapies likely to produce electrocardiographic changes (see Table 13G-1). Thus, in patients with known cardiac disease, ECGs are warranted as part of a baseline examination, after initiating therapy known to produce electrocardiographic changes that correlate with therapeutic responses, progression of disease, or adverse effects, for intermittent follow-up after changes in signs or symptoms such as syncope, chest pain, and extreme fatigue or relevant laboratory findings, and after significant intervals (usually 1 year or longer) in the absence of clinical changes. Follow-up ECGs are not considered appropriate for patients with mild chronic cardiovascular conditions that are not considered likely to progress (e.g., mild mitral valve prolapse). ECGs at each visit are considered inappropriate for patients with stable heart disease who are seen frequently (e.g., within 4 months) and have no evidence of clinical change.

PATIENTS SUSPECTED OF HAVING CARDIOVASCULAR DISEASE

In patients suspected of having cardiac disease or at high risk for cardiac disease, an ECG is appropriate as part of an initial evaluation in the presence of signs or symptoms suggesting cardiac disease, in patients with important risk factors such as cigarette abuse, diabetes mellitus, peripheral vascular disease, or a family history of cardiac disease, during therapy with cardiovascular medications, and during follow-up if clinical events develop or at prolonged intervals if clinically stable (see Table 13G-2). In the follow-up of patients at increased risk for heart disease, ECGs every 1 to 5 years are considered appropriate, but routine screening ECGs more frequently than yearly are not supported for patients who remain clinically stable.

PATIENTS WITHOUT KNOWN OR SUSPECTED CARDIOVASCULAR DISEASE

The various guidelines differ in their recommendations for the use of ECGs to screen for cardiovascular disease in healthy people. In the ACC/AHA guidelines, ECGs are considered appropriate screening tests in patients without apparent or suspected heart disease who are 40 years of age or older (see Table 13G-3). Earlier guidelines from the AHA recommended that ECGs be obtained at ages 20, 40, and 60 years. In contrast, the U.S. Preventive Services Task Force (USPSTF) and the task force assembled by the Canadian government did not find evidence to support the use of screening ECG.

The ACC/AHA guidelines also recommend ECGs for patients for whom drugs with a high incidence of cardiovascular effects (e.g., chemotherapy) or for whom exercise testing is planned, and for people of any age in occupations with high cardiovascular demands or whose cardiovascular status might affect the safety of other people (e.g., airline pilots).

In populations without known disease or significant risk factors, it has become common practice to include an ECG as part of routine health examinations and on any admission to a hospital. These ECGs are considered...
earlier sections of this chapter, that many commonly encountered cardiovascular events. Against which to compare later tracings, and assessing the future risk of unknown abnormalities, serving as a baseline for routine clinical screening by numerous organizations, including the USPSTF, do not include an ECG. It is clear, as described in earlier sections of this chapter, that many commonly encountered electrocardiographic abnormalities in clinically normal people raise the risk of various forms of cardiac morbidity and mortality. Although the proportion of persons subjected to routine screening ECGs who have these abnormalities may be substantial, the overall sensitivity and specificity of electrocardiographic findings are too low to warrant universal screening.

### TABLE 13G-1 ACC/AHA Guidelines for Electrocardiography in Patients with Known Cardiovascular Disease or Dysfunction

<table>
<thead>
<tr>
<th>INDICATION</th>
<th>CLASS I (INDICATED)</th>
<th>CLASS II (EQUIVOCAL)</th>
<th>CLASS III (NOT INDICATED)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline or initial evaluation</td>
<td>All patients</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Response to therapy</td>
<td>Patients in whom prescribed therapy is known to produce changes in the ECG that correlate with therapeutic responses or progression of disease</td>
<td>None</td>
<td>Patients receiving pharmacologic or nonpharmacologic therapy not known to produce changes in the ECG or to affect conditions that may be associated with such changes</td>
</tr>
<tr>
<td>Follow-up</td>
<td>Patients with a change in symptoms, signs, or relevant laboratory findings</td>
<td>None</td>
<td>Adult patients whose cardiovascular condition is usually benign and unlikely to progress (e.g., patients with asymptomatic mild mitral valve prolapse, mild hypertension, or premature contractions in absence of organic heart disease)</td>
</tr>
<tr>
<td>Before surgery</td>
<td>All patients with known cardiovascular disease or dysfunction, except as noted under Class II</td>
<td>Patients with hemodynamically insignificant congenital or acquired heart disease, mild systemic hypertension, or infrequent premature complexes in absence of organic heart disease</td>
<td>None</td>
</tr>
</tbody>
</table>

### TABLE 13G-2 ACC/AHA Guidelines for Electrocardiography in Patients Suspected of Having or at Increased Risk for Cardiovascular Disease or Dysfunction

<table>
<thead>
<tr>
<th>SETTING</th>
<th>CLASS I (APPROPRIATE)</th>
<th>CLASS II (EQUIVOCAL)</th>
<th>CLASS III (INAPPROPRIATE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline or initial evaluation</td>
<td>All patients suspected of having or being at increased risk for cardiovascular disease</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Response to therapy</td>
<td>To assess therapy with cardioactive drugs in patients with suspected cardiac disease</td>
<td>To assess response to administration of any agent known to alter serum electrolyte concentration</td>
<td>To assess response to administration of agents known not to influence cardiac structure or function</td>
</tr>
<tr>
<td>Follow-up</td>
<td>Presence of any change in clinical status or laboratory findings suggesting interval development of cardiac disease or dysfunction</td>
<td>None</td>
<td>Follow-up ECGs more often than yearly not indicated for patients who remain clinically stable, not at increased risk for development of cardiac disease, and not demonstrated to have cardiac disease with previous studies</td>
</tr>
<tr>
<td>Before surgery</td>
<td>As part of preoperative evaluation of any patient with suspected, or at increased risk of developing, cardiac disease or dysfunction</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>
The consequences of high rates of false-positive results, including unnecessary diagnostic testing, overtreatment, and labeling, especially in populations with a low prevalence of disease, may outweigh the benefits of screening.

**SPECIAL POPULATIONS**

**Persons with Dangerous Occupations**

Recommendations for screening of persons with dangerous jobs or jobs that place others at risk—for example, airline pilots and bus drivers—are also controversial. Although no specific data defining the value of routine screening are available, some groups, including the USPSTF, recognize the potential for benefit in relation to the possible risk to others.

**Preoperative Evaluation**

The routine recording of the ECG before noncardiac surgery in patients without other indications, although a common practice, has also been challenged. Routine electrocardiographic recording may be recommended for patients with one or more risk factors undergoing vascular surgery and for patients with known peripheral or cerebrovascular disease undergoing intermediate-risk procedures. It may be reasonable in patients without risk factors undergoing vascular procedures or with risk factors who are undergoing intermediate-risk procedures.

However, routine preoperative or postoperative ECGs are not recommended for asymptomatic persons undergoing low-risk procedures. Although electrocardiographic abnormalities are common in these patients, the findings do not have predictive value for postoperative cardiac events beyond that established by routine clinical examinations. In these cases, use of the ECG may be based on clinical judgment rather than on rigid protocol requirements.

**Screening of Athletes**

The routine screening of competitive athletes by electrocardiography remains unsettled. Proposals to include the ECG are based on its high sensitivity in detecting the most common underlying causes of athlete deaths such as hypertrophic cardiomyopathy and the long-QT interval syndromes. It is thus proposed that prospective identification of these abnormalities can reduce the occurrence of sudden death by facilitating the disqualification of high-risk affected persons.

Evidence of the success of this approach has been reported, and both the European Society of Cardiology and the International Olympic Committee recommend including the ECG as part of pre-participation medical review. In contrast, the AHA does not recommend routine electrocardiographic screening, largely because of the cost, the lack of an organized system to achieve the goal, and the risks of false-positive recordings. Rather, they recommend a more complete examination if suggestive abnormalities are found in the personal and family history or on physical examination.

**Cardioactive Drug Administration**

The role of the ECG as a baseline and in the follow-up of patients taking drugs with potential cardioactive effects, especially QT(U) interval prolongation, remains poorly defined and, in some cases, controversial. One example is the suggestion by an expert panel on electrocardiographic screening in methadone treatment to "obtain a pretreatment electrocardiogram for all patients to measure the QTc interval and a follow-up electrocardiogram within 30 days and annually. Additional electrocardiography is recommended if the methadone dosage exceeds 100 mg/day or if patients have unexplained syncope or seizures." This proposal, which has provoked considerable debate and discussion, has not been formally endorsed by national cardiology societies.

**REFERENCES**