SECTION I
Basic Concepts

Cardiac Electrical Activity

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THE BOOK: MARRIOTT’S PRACTICAL ELECTROCARDIOGRAPHY, 12TH EDITION

What Can This Book Do for Me?

This 12th edition of Marriott’s Practical Electrocardiography has been specifically designed to provide you with a practical approach to reading electrocardiograms (ECGs). No previous text or experience is required. You should consider how you learn best before deciding how to approach this book. If you are most comfortable acquiring a basic understanding of a subject even before you encounter a need to use the subject information, you probably want to read the first section [Basic Concepts] carefully. However, if you have found that such understanding is not really helpful to you until you encounter a specific problem, you probably want to quickly scan this first section.

All medical terms are defined in a glossary at the end of each chapter. Each individual “practical concept” is presented in a “Learning Unit.” Each Learning Unit begins on a new page with a heading that is underscored with a green line. The Learning Units are listed in the Table of Contents for easy reference. This book will be more useful if you make your own annotations; blank space is provided for this purpose.

The illustrations are fully integrated into the text, eliminating the need for extensive figure legends. A pink background is used for the ECG examples to provide contrast with the recordings, which appear in black. Because ECG reading is a visual experience, most of the book’s illustrations are typical examples of the various clinical situations for which ECGs are recorded. Reference to these examples should provide you with support for accurately reading the ECGs you encounter in your own clinical experience.

To better understand the basic concepts the ECG provides, we have added a digital content to the 12th edition to provide the learner with visuospatial orientation of common cardiac abnormalities. The digital content is not a stand-alone educational tool but should be used to visually conceptualize.

What Can I Expect From Myself When I Have “Completed” This Book?

This book is not intended for you to “complete.” Rather, it is intended as a reference for the ECG problems you encounter. There will be evidence that this is your book, with dog-eared pages and your own notes in the sections you have already used. Through your experience with this book, you should develop confidence in identifying a “normal” ECG and be able to accurately diagnose the many common ECG abnormalities. You should also have an understanding of the practical aspects of the pathophysiologic basis for each of these common ECG abnormalities.
THE ELECTROCARDIOGRAM

What Is an Electrocardiogram?

An ECG is the recording (gram) of the electrical activity (electro) generated by the cells of the heart (cardio) that reaches the body surface. This electrical activity initiates the heart’s muscular contraction that pumps the blood to the body. Each ECG recording electrode provides one of the poles of a lead, which gives the view of this electrical activity that it “sees” from its particular position on the body surface. Observation of the 12 views provided by the routine clinical ECG allows you to “move around” this electrical activity just as though you were seeing the heart from various viewpoints. Indeed, reversal of the poles of each lead provides a reciprocal or mirrorlike view. You should probably have your own ECG recorded and then ask an experienced ECG reader to explain it to you. This experience removes the mystery surrounding the ECG and prepares you for the “Basic Concepts” section of this book.

What Does an Electrocardiogram Actually Measure?

The ECG recording plots voltage on its vertical axis against time on its horizontal axis. Measurements along the horizontal axis indicate the overall heart rate, regularity, and the time intervals during electrical activation that move from one part of the heart to another. Measurements along the vertical axis indicate the voltage measured on the body surface. This voltage represents the “summation” of the electrical activation of all of the cardiac cells. Some abnormalities can be detected by measurements on a single ECG recording, but others become apparent only by observing serial recordings over time.

What Medical Problems Can Be Diagnosed With an Electrocardiogram?

Many cardiac abnormalities can be detected by ECG interpretation, including enlargement of heart muscle, electrical conduction blocks, insufficient blood flow, and death of heart muscle due to a coronary thrombosis. The ECG can even identify which of the heart’s coronary arteries contains this occlusion when it is still only threatening to destroy a region of heart muscle. The ECG is also the primary method for identifying problems with heart rate and regularity. In addition to its value for understanding cardiac problems, the ECG can be used to aid in diagnosing medical conditions throughout the body. For example, the ECG can reveal abnormal levels of ions in the blood, such as potassium and calcium, and abnormal function of glands such as the thyroid. It can also detect potentially dangerous levels of certain drugs.

Would It Be Helpful to Have My Own Electrocardiogram Recorded?

In the process of learning electrocardiography, it may be useful to have your own ECG recorded. Here is a list of possible reasons why:

- You will be able to understand the importance of ECG lead placement and orientation because you have experienced the electrodes being placed on your body.
- You can carry your ECG with you as reference if an abnormality is ever suspected.
- You can compare it to someone else’s ECG to see normal variations.
- You can compare it at different times of your life to see how it changes.
- You can take deep breaths to see how the resulting slight movement of your heart affects your ECG.
- You can move the electrodes to incorrect positions to see how this distorts the recording.
ANATOMIC ORIENTATION OF THE HEART

The position of the heart within the body determines the “view” of the cardiac electrical activity that can be observed from any site on the body surface. A frontal plane magnetic resonance image of the heart within the thorax is seen in Figure 1.1A. The atria are located in the top or base of the heart, and the ventricles taper toward the bottom or apex. The long axis of the heart, which extends from base to apex, is tilted to the left at its apical end in the schematic drawing of this frontal plane view (see Fig. 1.1B).

**FIGURE 1.1.** A. Frontal plane magnetic resonance image. B. Chambers of the heart. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
However, the right atrium/right ventricle and left atrium/left ventricle are not directly aligned with the right and left sides of the body as viewed in the transverse plane magnetic resonance image of the heart within the thorax (Fig. 1.2A). The schematic drawing shows how the right-sided chambers of the heart are located anterior to the left-sided chambers, with the result that the interatrial and interventricular septa form a diagonal in this transverse plane view (see Fig. 1.2B).\textsuperscript{1,2}

**FIGURE 1.2.** A. Transverse plane magnetic resonance image, as viewed from below. B. Chambers of the heart. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
THE CARDIAC CYCLE

The mechanical pumping action of the heart is produced by cardiac muscle ("myocardial") cells that contain contractile proteins. The timing and synchronization of contraction of these myocardial cells are controlled by noncontractile cells of the pacemaking and conduction system. Impulses generated within these specialized cells create a rhythmic repetition of events called cardiac cycles. Each cycle includes electrical and mechanical activation (systole) and recovery (diastole). The terms commonly applied to these components of the cardiac cycle are listed in Table 1.1. Because the electrical events initiate the mechanical events, there is a brief delay between the onsets of electrical and mechanical systole and of electrical and mechanical diastole.

The electrical recording from inside a single myocardial cell as it progresses through a cardiac cycle is illustrated in Figure 1.3. During electrical diastole, the cell has a baseline negative electrical potential and is also in mechanical diastole, with separation of the contractile

**Table 1.1.**

<table>
<thead>
<tr>
<th>Terms Describing Cardiac Cycle</th>
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<tr>
<td>Systole</td>
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<tr>
<td><strong>Electrical</strong></td>
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<tr>
<td>Activation</td>
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<td>Excitation</td>
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<td>Depolarization</td>
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proteins. At top, a single cardiac cell is shown at three points in time, during which it is relaxed, contracted, and relaxed again. An electrical impulse arriving at the cell allows positively charged ions to cross the cell membrane, causing its depolarization. This movement of ions initiates “electrical systole,” which is characterized by an action potential. This electrical event then initiates mechanical systole, in which the contractile proteins within the myocardial cell slide over each other, thereby shortening the cell. Electrical systole continues until the positively charged ions are pumped out of the cell, causing its repolarization. Below the cell is a representation of an internal electrical recording that returns to its negative resting level. The repolarization process begins with an initial brief component that is followed by a “plateau” that varies among myocardial cells. Repolarization is completed by a rapid component. This return of “electrical diastole” causes the contractile proteins within the cell to separate. The cell is then capable of being reactivated when another electrical impulse arrives at its membrane.

The electrical and mechanical changes in a series of myocardial cells (aligned end to end) as they progress through a cardiac cycle are illustrated in Figure 1.4. In Figure 1.4A, the four representative cells are in their resting or repolarized state. Electrically, the cells have negative charges; mechanically, their contractile proteins are separated. An electrical stimulus arrives at the second myocardial cell in Figure 1.4B, causing electrical and then mechanical systole.

The wave of depolarization in Figure 1.4C spreads throughout all the myocardial cells. In Figure 1.4D, the recovery or repolarization process begins in the second cell, which was the first to depolarize. Finally, in Figure 1.4E, the wave of repolarization spreads throughout all of the myocardial cells, and they await the coming of another electrical stimulus.3–6

FIGURE 1.4. Cardiac cycle in a series of myocardial cells. The symbols are the same as in Figure 1.3. (Modified from Thaler MS. The Only EKG Book You’ll Ever Need. Philadelphia, PA: JB Lippincott; 1988:9.) See Animation 1.2.

CHAPTER 1: Cardiac Electrical Activity
SECTION I: Basic Concepts

In Figure 1.5, the relationship between the intracellular electrical recording from a single myocardial cell presented in Figure 1.3 is combined with an ECG recording on a "lead" that has its positive and negative electrodes on the body surface. The ECG recording is the summation of electrical signals from all of the myocardial cells. There is a flat baseline in two very different situations: (a) when the cells are in their resting state electrically and (b) when the summation of cardiac electrical activity is directed perpendicular to a line between the positive and negative electrodes. The depolarization of the cells produces a high-frequency ECG waveform. Then, between the initial transient and final complete phases of repolarization, the ECG returns to the baseline. Completion of repolarization of the myocardial cells is represented on the ECG by a lower frequency waveform in the opposite direction from that representing depolarization.

FIGURE 1.5. Single-cell recording combined with an ECG. The symbols are the same as in Figure 1.3. (Modified from Thaler MS. The Only EKG Book You'll Ever Need. Philadelphia, PA: JB Lippincott; 1988:11.) See Animation 1.3.

In Figure 1.5, the relationship between the intracellular electrical recording from a single myocardial cell presented in Figure 1.3 is combined with an ECG recording on a "lead" that has its positive and negative electrodes on the body surface. The ECG recording is the summation of electrical signals from all of the myocardial cells. There is a flat baseline in two very different situations: (a) when the cells are in their resting state electrically and (b) when the summation of cardiac electrical activity is directed perpendicular to a line between the positive and negative electrodes. The depolarization of the cells produces a high-frequency ECG waveform. Then, between the initial transient and final complete phases of repolarization, the ECG returns to the baseline. Completion of repolarization of the myocardial cells is represented on the ECG by a lower frequency waveform in the opposite direction from that representing depolarization.

Animation 1.3
In Figure 1.6, a lead with its positive and negative electrodes has been placed on the body surface and connected to a single-channel ECG recorder. The process of production of the ECG recording by waves of depolarization and repolarization spreading from the negative toward the positive electrode is illustrated. In Figure 1.6A, the first of the four cells shown is electrically activated, and the activation then spreads into the second cell. This spread of depolarization toward the positive electrode produces a positive (upward) \textit{deflection} on the ECG. In Figure 1.6B, all of the cells are in their depolarized state, and the ECG recording returns to its baseline level. In Figure 1.6C, repolarization begins in the same cell in which depolarization was initiated, and the wave of repolarization spreads into the adjoining cell. This produces the oppositely directed negative (downward) waveform on the ECG recording.

\textbf{FIGURE 1.6.} Single-channel ECG recording. The symbols are the same as in Figure 1.3. Black semiovals, electrodes. (Modified from Thaler MS. The Only EKG Book You’ll Ever Need. Philadelphia, PA: JB Lippincott; 1988:29,31.) See Animation 1.3.
FIGURE 1.7  Special cells of the cardiac pacemaking and conduction system. In A, the anterior aspect of all chambers has been removed to reveal the entire AV and ventricular conduction system. In B, the lateral aspect of the right atrium and ventricle has been removed. In C, the lateral aspect of the left atrium and ventricle has been removed to reveal the right and left bundle branches, respectively. AV, atrioventricular; SA, sinoatrial. (Modified from Netter FH. In: Yonkman FF, ed. The Ciba Collection of Medical Illustrations. Vol 5: Heart. Summit, NJ: Ciba–Geigy; 1978:13,49.)
The electrical activation of a single cardiac cell or even of a small group of cells does not produce enough voltage to be recorded on the body surface. Clinical electrocardiography is made possible by the activation of large groups of atrial and ventricular myocardial cells, whose numbers are of sufficient magnitude for their electrical activity to be recorded on the body surface.

Myocardial cells normally lack the ability for either spontaneous formation or rapid conduction of an electrical impulse. They depend on special cells of the cardiac pacemaking and conduction system that are located strategically through the heart for these functions [Fig. 1.7]. These cells are arranged in nodes, bundles, bundle branches, and branching networks of fascicles. The cells that form these structures lack contractile capability, but they can generate spontaneous electrical impulses (act as pacemakers) and alter the speed of electrical conduction throughout the heart. The intrinsic pacemaking rate is most rapid in the specialized cells in the atria and slowest in those in the ventricles. This intrinsic pacemaking rate is altered by the balance between the sympathetic and parasympathetic components of the autonomic nervous system.7–10

Figure 1.7 illustrates three different anatomic relationships between the cardiac pumping chambers and the specialized pacemaking and conduction system: Anterior precordium with less tilt (see Fig. 1.7A), right anterior precordium looking onto the interatrial and interventricular septa through the right atrium and ventricle (see Fig. 1.7B), and left posterior thorax looking onto the septa through the left atrium and ventricle (see Fig. 1.7C). The sinoatrial (SA) or sinus node is located high in the right atrium, near its junction with the superior vena cava. The SA node is the predominant cardiac pacemaker, and its highly developed capacity for autonomic regulation controls the heart’s pumping rate to meet the changing needs of the body. The atrioventricular (AV) node is located low in the right atrium, adjacent to the interatrial septum. Its primary function is to slow electrical conduction sufficiently to asynchronize the atrial contribution to ventricular pumping. Normally, the AV node is the only structure capable of conducting impulses from the atria to the ventricles because these chambers are otherwise completely separated by nonconducting fibrous and fatty tissue.11–13

In the atria, the electrical impulse generated by the SA node spreads through the myocardium without needing to be carried by any specialized conduction bundles. Electrical impulses reach the AV node where the impulse is delayed before continuing to the intraventricular conduction pathways.

The intraventricular conduction pathways include a common bundle (bundle of His) that leads from the AV node to the summit of the interventricular septum as well as the right and left bundle branches of the bundle of His, which proceed along the septal surfaces of their respective ventricles. The left bundle branch fans out into fascicles that proceed along the left septal endocardial surface and toward the two papillary muscles of the mitral valve. The right bundle branch remains compact until it reaches the right distal septal surface, where it branches into the interventricular septum and toward the free wall of the right ventricle. These intraventricular conduction pathways are composed of fibers of Purkinje cells, which have specialized capabilities for both pacemaking and rapid conduction of electrical impulses. Fascicles composed of Purkinje fibers form networks that extend just beneath the surface of the right and left ventricular endocardium. After reaching the ends of these Purkinje fascicles, the impulses then proceed more slowly from endocardium to epicardium throughout the right and left ventricles.14–16 This synchronization process allows activation of the myocardium at the base to be delayed until the apical region has been activated. This sequence of electrical activation is necessary to achieve the most efficient cardiac pumping because the pulmonary and aortic outflow valves are located at the ventricular bases.
RECORDING LONG-AXIS (BASE–APEX) CARDIAC ELECTRICAL ACTIVITY

FIGURE 1.8. Optimal sites for recording long-axis cardiac electrical activity. Black semi-ovals represent the electrodes. L, left; R, right. See Animation 1.4.

The schematic frontal plane view of the heart in the thorax is shown in Figure 1.1B, with the negative and positive electrodes located where the long axis of the heart intersects with the body surface. The optimal body surface sites for recording long-axis (base–apex) cardiac electrical activity are located where the extensions of the long axis of the heart intersect with the body surface (Fig. 1.8). The negative electrode on the right shoulder and the positive electrode on the left lower chest are aligned from the cardiac base to apex parallel to the interatrial and interventricular septa. This long-axis “ECG lead” is oriented similarly to a lead termed “aVR” on the standard 12-lead ECG (see Chapter 2). However, the lead in Figure 1.8 would actually be lead –aVR because, for lead aVR, the positive electrode is placed on the right arm. Both the positive and negative electrodes are attached to a single-channel ECG recorder that produces predominantly upright waveforms on the ECG, as explained later in this unit (see also Chapter 2).
The long-axis recording in Figure 1.8 has been magnified to illustrate the sequence of activation in structures of the pacemaking and conduction system [Fig. 1.9]. The initial wave of a cardiac cycle represents activation of the atria and is called the *P wave*. Because the SA node is located in the right atrium, the first part of the P wave represents the activation of this chamber. The middle section of the P wave represents completion of right-atrial activation and initiation of left-atrial activation. The final section of the P wave represents completion of left-atrial activation. Activation of the AV node begins by the middle of the P wave and proceeds slowly during the final portion of the P wave. The wave representing electrical recovery of the atria is usually too small to be seen on the ECG, but it may appear as a distortion of the PR segment. The bundle of His and bundle branches are activated during the PR segment but do not produce waveforms on the body surface ECG.

The next group of waves recorded is termed the *QRS complex*, representing the simultaneous activation of the right and left ventricles. On this long-axis recording, the P wave is entirely positive and the QRS complex is predominantly positive.

**FIGURE 1.9.** Wave forms. P, atrial activation; Q, R, and S, ventricular activation; T and U, ventricular recovery. AV, atrioventricular; SA, sinoatrial. See Animation 1.5.
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The QRS complex may normally appear as one (monophasic), two (diphasic), or three (triphasic) individual waveforms (Fig. 1.10). By convention, a negative wave at the onset of the QRS complex is called a Q wave. The predominant portion of the QRS complex recorded from this long-axis viewpoint is normally positive and is called the R wave, regardless of whether or not it is preceded by a Q wave. A negative deflection following an R wave is called an S wave. When a second positive deflection occurs, it is termed R’ (R prime). A monophasic negative QRS complex should be termed a QS wave (see Fig. 1.10, left). Biphasic complexes are either RS or QR (see Fig. 1.10, center), and triphasic complexes are RSR’ or QRS (see Fig. 1.10, right). Occasionally, more complex patterns of QRS waveforms occur (see Chapter 3).

**FIGURE 1.10.** QRS complex waveforms and their alphabetical terms. (From Selvester RH, Wagner GS, Hindman NB. The development and application of the Selvester QRS scoring system for estimating myocardial infarct size. Arch Intern Med. 1985;145:1879, with permission. Copyright 1985, American Medical Association.) See Animation 1.5.
The wave in the cardiac cycle that represents recovery of the ventricles is called the T wave. The frontal plane view of the right and left ventricles (as in Fig. 1.7A) is presented along with schematic recordings from left-ventricular myocardial cells on the endocardial and epicardial surfaces (Fig. 1.11). The numbers below the recordings refer to the time (in seconds) required for these sequential electrical events. As stated in the previous Learning Unit, the Purkinje fibers provide electrical activation of the endocardium, initiating a "wave front" of depolarization that spreads through the myocardial wall to the cells on the epicardial surface. Because recovery of the ventricular cells (repolarization) causes an ion flow opposite to that of depolarization, one might expect the T wave to be inverted in relation to the QRS complex, as shown in Figures 1.5 and 1.6. However, epicardial cells repolarize earlier than endocardial cells, thereby causing the wave of repolarization to spread in the direction opposite that of the wave of depolarization (epicardium to endocardium; see Fig. 1.11A). This results in the long-axis body surface ECG waveform (as in Fig. 1.9) with the T wave deflected in a similar direction as the QRS complex (see Fig. 1.11B). The T wave is sometimes followed by another small upright wave (the source of which is uncertain), called the U wave, as seen in Figure 1.9.
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The magnified recording from Figure 1.9 is again presented with the principal ECG segments (P–R and S–T) and time intervals (P–R, QRS, Q–T, and T–P) as displayed in Figure 1.12. The time from the onset of the P wave to the onset of the QRS complex is called the **PR interval**, regardless of whether the first wave in this QRS complex is a Q wave or an R wave. This interval measures the time between the onset of activation of the atrial and ventricular myocardium. The designation **PR segment** refers to the time from the end of the P wave to the onset of the QRS complex. The **QRS interval** measures the time from the beginning to the end of ventricular activation. Because activation of the thick left-ventricular free wall and interventricular septum requires more time than does activation of the right-ventricular free wall, the terminal portion of the QRS complex represents the balance of forces between the basal portions of these thicker regions.

The **ST segment** is the interval between the end of ventricular activation and the beginning of ventricular recovery. The term **ST segment** is used regardless of whether the final wave of the QRS complex is an R or an S wave. The junction of the QRS complex and the ST segment is called the **J point**. The interval from the onset of ventricular activation to the end of ventricular recovery is called the **QT interval**. This term is used regardless of whether the QRS complex begins with a Q or an R wave.

At low heart rates in a healthy person, the PR, ST, and TP segments are at approximately the same level (*isoelectric*). The TP segment between the end of the T or U wave and beginning of the P wave is typically used as the **baseline** for measuring the amplitudes of the various waveforms.16-20

**FIGURE 1.12.** Magnified cardiac long-axis viewpoint of ECG segments and time intervals. See Animation 1.7.

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It is often important to determine whether an abnormality originates from the left or right side of the heart. The optimal sites for recording left- versus right-sided cardiac electrical activity are located where the extensions of the short axis of the heart intersect with the body surface as illustrated in the schematic transverse plane view (Fig. 1.13). The negative electrode on the left posterior thorax (back) and the positive electrode on the right anterior thorax (right of sternum) are aligned perpendicular to the interatrial and interventricular septa, and they are attached to a single-channel ECG recorder. This short-axis "ECG lead" is oriented similarly to a lead termed "V1" on the standard 12-lead ECG (see Chapter 2). The positive electrode for lead V1 is placed on the anterior thorax in the fourth intercostal space at the right edge of the sternum. The typically diphasic P and T waves and the predominantly negative QRS complex recorded by electrodes at these positions are indicated on the ECG recording.
The ECG waveforms from the cardiac short-axis viewpoint (see Fig. 1.13) are magnified in Figure 1.14, with the principal ECG segments and time intervals indicated. The initial part of the P wave, representing only right-atrial activation, appears positive at this site because of the progression of electrical activity from the interatrial septum toward the right-atrial free wall and the positive electrode. The final part of the P wave, representing only left-atrial activation, appears negative because of progression of electrical activity from the interatrial septum toward the left-atrial free wall and the negative electrode. This activation sequence produces a diphasic P wave.

The initial part of the QRS complex represents the progression of activation in the interventricular septum. This movement is predominantly from the left toward the right side of the septum, producing a positive [R wave] deflection at this left- versus right-sided recording site. The midportion of the QRS complex represents progression of electrical activation through the left- and right-ventricular myocardium. Because the posteriorly positioned left-ventricular free wall is much thicker than the anteriorly placed right-ventricular free wall, its activation predominates over that of the latter, resulting in a deeply negative deflection (S wave). The final portion of the QRS complex represents the completion of activation of the left-ventricular free wall and interventricular septum. This posteriorly directed excitation is represented by the completion of the S wave. The T wave is typically biphasic in this short-axis view, and there is no U wave.
GLOSSARY

**Action potential:** the electrical potential recorded from within a cell as it is activated by an electrical current or impulse.

**Anterior:** located toward the front of the body.

**Apex:** the region of the heart where the narrowest parts of the ventricles are located.

**Atrioventricular (AV) node:** a small mass of tissue situated in the inferior aspect of the right atrium, adjacent to the septum between the right and left atria. Its function is to slow impulses traveling from the atria to the ventricles, thereby synchronizing atrial and ventricular pumping.

**Atrium:** a chamber of the heart that receives blood from the veins and passes it along to its corresponding ventricle.

**Base:** the broad top of the heart where the atria are located.

**Baseline:** see **Isoelectric line**.

**Bundle branches:** groups of Purkinje fibers that emerge from the common bundle (of His); the right bundle branch rapidly conducts electrical impulses to the right ventricle, while the left bundle branch conducts impulses to the left ventricle.

**Cardiac cycle:** a single episode of electrical and mechanical activation and recovery of a myocardial cell or of the entire heart.

**Cardiac pacemaking and conduction system:** groups of modified myocardial cells strategically located throughout the heart and capable of forming an electrical impulse and/or of conducting impulses particularly slowly or rapidly.

**Common bundle (of His):** a compact group of Purkinje fibers that originates at the AV node and rapidly conducts electrical impulses to the right and left bundle branches.

**Deflection:** a waveform on the ECG; its direction may be either upward (positive) or downward (negative).

**Diphasic:** consisting of two components.

**Distal:** situated away from the point of attachment or origin; the opposite of proximal.

**Electrocardiogram (ECG):** the recording made by the electrocardiograph, depicting the electrical activity of the heart.

**Electrode:** an electrical contact that is placed on the skin and is connected to an ECG recorder.

**Endocardium:** the inner aspect of a myocardial wall, adjacent to the blood-filled cavity of the adjacent chamber.

**Epicardium:** the outer aspect of a myocardial wall, adjacent to the pericardial lining that closely envelops the heart.

**Fascicle:** a small bundle of Purkinje fibers that emerges from a bundle or a bundle branch to rapidly conduct impulses to the endocardial surfaces of the ventricles.

**Isoelectric line:** a horizontal line on an ECG recording that forms a baseline; representing neither a positive nor a negative electrical potential.

**J point:** junction of the QRS complex and the ST segment.

**Lateral:** situated toward either the right or left side of the heart or of the body as a whole.

**Monophasic:** consisting of a single component, being either positive or negative.

**P wave:** the first wave depicted on the ECG during a cardiac cycle; it represents atrial activation.

**PR interval:** the time from onset of the P wave to onset of the QRS complex. This interval represents the time between the onsets of activation of the atrial and the ventricular myocardium.

**PR segment:** the time from the end of the P wave to the onset of the QRS complex.

**Purkinje cells or fibers:** modified myocardial cells that are found in the distal aspects of the pacemaking and conduction system, consisting of the common bundle, the bundle branches, the fascicles, and individual strands.

**Q wave:** a negative wave at the onset of the QRS complex.
QRS complex: the second wave or group of waves depicted on the ECG during a cardiac cycle; it represents ventricular activation.

QRS interval: the time from the beginning to the end of the QRS complex, representing the duration required for activation of the ventricular myocardial cells.

QS: a monophasic negative QRS complex.

QT interval: the time from the onset of the QRS complex to the end of the T wave. This interval represents the time from the beginning of ventricular activation to the completion of ventricular recovery.

R wave: the first positive wave appearing in a QRS complex; it may appear at the onset of the QRS complex or following a Q wave.

R' wave: the second positive wave appearing in a QRS complex.

Repolarization: the transition in which the inside of the cell becomes markedly positive in relation to the outside. This condition is maintained by a pump in the cell membrane, and it is disturbed by the arrival of an electrical current.

Septum: a dividing wall between the atria or between the ventricles.

Sinoatrial (SA) node: a small mass of tissue situated in the superior aspect of the right atrium, adjacent to the entrance of the superior vena cava. It functions as the dominant pacemaker, which forms the electrical impulses that are then conducted throughout the heart.

ST segment: the interval between the end of the QRS complex and the beginning of the T wave.

Superior: situated above and closer to the head than another body part.

Superior vena cava: one of the large veins that empties into the right atrium.

Systole: the period in which the electrical and mechanical aspects of the heart are in their active state: electrical systole is characterized by depolarization and mechanical systole by contraction. During mechanical systole, blood is being pumped out of the heart.

T wave: the final major wave depicted on the ECG during a cardiac cycle; it represents ventricular recovery.

Triphasic: consisting of three components.

U wave: a wave on the ECG that follows the T wave in some individuals; it is typically small and its source is uncertain.

Ventricle: a chamber of the heart that receives blood from its corresponding atrium and pumps the blood it receives out into the arteries.

Waveform: electrocardiographic representation of either the activation or recovery phase of electrical activity of the heart.

REFERENCES


