Clinical Electrocardiography

A Simplified Approach

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THIRD EDITION

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Preface

This book is an introduction to electrocardiography. It is written particularly for medical students, nurses, and paramedical assistants and assumes no previous instruction in ECG reading. Physicians wishing to review basic electrocardiography have also found the material useful. The text derives from the cumulative experience of both authors in the field of electrocardiography, beginning in 1944 with the invention of the aV_R, aV_L, and aV_F leads by one of us (E.G.). The text itself is closely based on a series of lectures given over the past decade by the other (A.G.) to physician's associates, medical students, nurses, house officers, and emergency room physicians.

We have divided the book into three sections. Part I covers the basic principles of electrocardiography, normal ECG patterns, and the major abnormal P-QRS-T patterns. Part II describes the major abnormalities of heart rhythm and conduction. Part III is an extensive collection of practice questions and problems for review. In addition, we have interspersed practice questions throughout the text. In reading ECGs, as in learning a new language, fluency is attained only with repetition and review.

The clinical applications of ECG reading have been stressed throughout the book. Each time an abnormal pattern is mentioned, there is a brief discussion of the conditions that might have produced it. Although this is not intended as a manual of therapeutics, general principles of treatment and clinical management are briefly discussed. In addition, students are encouraged to approach ECGs in terms of a rational simple differential diagnosis, rather than through the tedium of rote memorization. It is comforting for most students to discover that the number of possible arrhythmias that can produce a heart rate of 170 beats per minute is limited to just a handful of choices. Only three basic ECG patterns are found with cardiac arrest. Similarly, there are only a few causes of low-voltage patterns, of patterns in which the QRS complex is abnormally wide, etc.

In approaching any given ECG, there are always three essential questions. First, what does the ECG show; second, what are the possible causes of this pattern; and, third, what if anything should be done about it? Most conventional ECG books focus on the first question, emphasizing pattern recognition. However, it is only a first step, for example, to note the premature ventricular contractions (PVCs) on an ECG. The more important question is what could have produced these abnormal beats: excessive caffeine intake, digitalis toxicity, hypoxemia, myocardial ischemia, hypokalemia, or something else? Treatment, of course, will depend on the answers to these questions.

The aim of this book, therefore, is to present the ECG as it is used on the hospital wards, in the outpatient clinics, and in the intensive and coronary care units, where recognition of normal and abnormal patterns is only the starting point in patient care and management.

The third edition contains new information on multiple topics, including arrhythmias, conduction disturbances, myocardial ischemia and infarction, and pacemakers. A new section on common pitfalls in ECG interpretation has been added (Chapter 19). Furthermore, the self-assessment problems in Part III have been expanded.

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Finally, we would like to thank Blanche and Ellen Goldberger for their encouragement and support. Ellen Goldberger contributed both artwork and editorial assistance and, once again, endured the karmic clutter of ECG clippings.

Ary L. Goldberger
Emanuel Goldberger

Contents

PART ONE

Basic Principles and Patterns

1 Introductory Principles

DEFINITION

An *electrocardiogram* (ECG)* records cardiac electrical currents (voltages, potentials) by means of metal *electrodes* placed on the surface of the body. As described in Chapter 3, these metal electrodes are placed on the arms, legs, and chest wall (precordium).

BASIC CARDIAC ELECTROPHYSIOLOGY

Before discussing the basic ECG patterns, we will review some elementary aspects of cardiac electrophysiology. Fortunately, only certain simple principles are required for clinical interpretation of ECGs. In addition, it is worth mentioning now that no special knowledge of electronics or electrophysiology is necessary despite the connotations of the term "electrocardiography."

In simplest terms the function of the heart is to contract and pump blood to the lungs for oxygenation and then to pump this oxygenated blood into the general (systemic) circulation. The signal for cardiac contraction is the spread of electrical currents through the heart muscle. These currents are produced both by specialized nervous conducting tissue within the heart and by the heart muscle itself. The ECG records the currents produced by the heart muscle.

*The abbreviation *EKG* is sometimes used.

ELECTRICAL STIMULATION OF THE HEART

The electrical "wiring" of the heart is outlined in Fig. 1-1. Normally the signal for cardiac electrical stimulation starts in the *sinus node* (also called the sinoatrial or SA node). The sinus node is located in the right atrium near the opening of the superior vena cava. It is a small collection of specialized cells capable of spontaneously generating electrical stimuli (signals). From the sinus node, this electrical stimulus spreads first through the right atrium and then into the left atrium. In this way the sinus node functions as the normal *pacemaker* of the heart.

The first phase of cardiac activation consists of the electrical stimulation of the right and left atria. This electrical stimulation, in turn, signals the atria to contract and to pump blood simultaneously through the tricuspid and mitral valves into the right and left ventricles respectively. The electrical stimulus then spreads to specialized conduction tissues in the *atrioventricular* (AV) *junction* (which includes the *AV node* and *bundle of His*) and then into the *left and right bundle branches,* which carry the stimulus to the ventricular muscle cells.

The AV junction, which functions as an electrical "bridge" connecting the atria and ventricles, is located at the base of the interatrial septum and extends into the ventricular septum (as shown in Fig. 1-1). It has two subdivisions: The upper (proximal) part is

CONDUCTION SYSTEM OF THE HEART

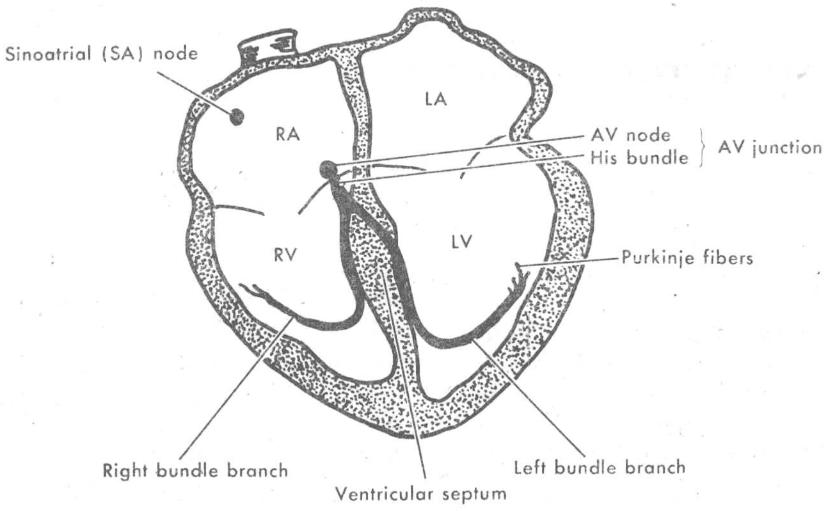

Fig. 1-1. Normally the cardiac stimulus is generated in the *SA node* (located in the right atrium, *RA*). It then spreads through the *RA* and *LA* (right and left atria). Next, it spreads through the *AV node* and *His bundle,* which comprise the *AV junction.* It then passes into the left and right ventricles *(LV* and *RV)* by way of the *left* and *right bundle branches,* which are continuations of the His bundle. Finally, it spreads to the ventricular muscle cells through the *Purkinje fibers.*

the AV node. (In older texts the terms "AV node" and "AV junction" are used synonymously.) The lower (distal) segment of the AV junction is called the bundle of His, after the physiologist who described it. The bundle of His then divides into two main branches: the right bundle branch, which brings the electrical stimulus to the right ventricle, and the left bundle branch,* which brings the electrical stimulus to the left ventricle (Fig. 1-1).

The electrical stimulus spreads simultaneously down the left and right bundle branches into the ventricular muscle itself *(ventricular myocardium).* The stimulus spreads itself into the ventricular myocardium by way of specialized conducting cells, called *Purkinje fibers,* located in the ventricular muscle.

Under normal circumstances, when the sinus node is pacing the heart (normal sinus rhythm), the AV junction appears to function primarily as a shuttle, directing the electrical stimulus into the ventricles. However, under some circumstances (described later) the AV junction can also function as an independent pacemaker of the heart. For example, if the sinus node fails to function properly, the AV junction may act as an *escape* pacemaker. In such cases an AV junc-

*The left bundle actually divides into two subbranches called *fascicles* (which are discussed on pp. 219 to 221).

tional rhythm (and *not* sinus rhythm) is present. This produces a distinct ECG pattern (described in Chapter 12).

Just as the spread of electrical stimuli through the atria leads to atrial contraction, so the spread of the electrical stimuli through the ventricles leads to ventricular contraction with pumping of blood to the lungs and into the general circulation.

In summary, the electrical stimulation of the heart normally follows a repetitive sequence of five steps:

1. Production of a stimulus from pacemaker cells in the sinus node (in the right atrium)
2. Stimulation of the right and left atria
3. Spread of the stimulus to the AV junction (AV node and bundle of His)
4. Spread of the stimulus simultaneously through the left and right bundle branches
5. Stimulation of the left and right ventricular myocardium

CARDIAC CONDUCTIVITY AND AUTOMATICITY

The speed with which the electrical impulses are conducted through different parts of the heart varies. For example, conduction speed is *slowest* through the AV node and *fastest* through the Purkinje fibers. The relatively slow conduction speed through the AV node is of functional importance because it allows the ventricles time to fill with blood before the signal for cardiac contraction arrives.

In addition to *conductivity* the other major electrical feature of the heart is *automaticity*. Automaticity refers to the capacity of certain myocardial cells to function as pacemakers, to spontaneously generate electrical impulses that spread throughout the heart. Normally, as mentioned earlier, the sinus node is the pacemaker of the heart because of its inherent automaticity. Under special conditions other cells outside the sinus node (in the atria, the AV junction, or the ventricles) can also act as independent pacemakers. For example, as mentioned before, if the automaticity of the sinus node is depressed, the AV junction may function as an escape pacemaker. In other conditions the automaticity of pacemakers outside the sinus node may become abnormally increased, and these *ectopic* (non-sinus) pacemakers may compete with the sinus node for control of the heartbeat. *Ectopy* is discussed in detail in Part II of this book (in the section on cardiac arrhythmias).

If you understand the normal physiologic stimulation of the heart, you have the basis for understanding the abnormalities of heart rhythm (arrhythmias) and conduction that produce distinctive ECG patterns. For example, failure of the sinus node to stimulate the heart properly may result in various rhythm disturbances, such as sinoatrial block (SA block), discussed in Chapter 11. Similarly, blockage of the spread of the stimulus through the AV junction produces various degrees of AV heart block (Chapter 15). Disease of the bundle branches may produce left or right bundle branch block (Chapter 7). Finally, any disease process that involves the ventricular muscle itself (for example, destruction of the heart muscle by myocardial infarction) also produces marked changes in the normal ECG patterns.

The first part of this book therefore is devoted to explaining the basis of the normal ECG, followed by a detailed look at major conditions causing abnormal P, QRS, and T* patterns. The second part of the book is devoted to describing the various abnormal rhythms (arrhythmias) and AV conduction disturbances that can occur. The third part of the book is a collection of review questions and examples. A short selective bibliography is provided at the end of the text.

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*P, QRS, and T are defined in Chapter 2.

REVIEW OF CHAPTER 1

An ECG records the electrical voltages (potentials) produced in the heart. It does this by means of metal electrodes (connected to an electrocardiograph) placed on the patient's extremities and chest wall.

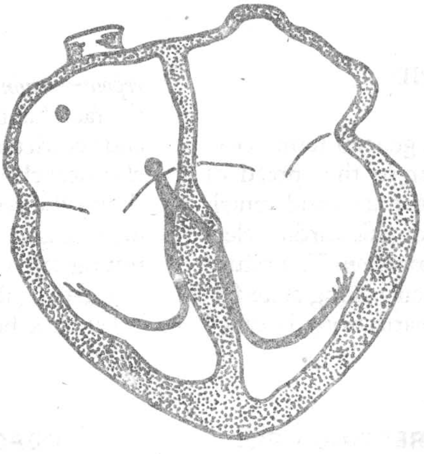
The electrical voltages of the heart are produced by specialized conduction fibers within the heart and by the heart muscle fibers themselves.

Normally a stimulus starts in the sinus (SA) node, located high in the right atrium near the superior vena cava. From here the stimulus spreads downward and to the left through the right and left atria and reaches the AV node, located at the beginning of the interventricular septum (Fig. 1-1). After a delay, the stimulus spreads through the AV junction (AV node and bundle of His). The bundle of His then subdivides into right and left branches. The right bundle branch runs down the interventricular septum and into the right ventricle. From there, small fibers, the Purkinje fibers, bring the stimulus outward into the main muscle mass of the right ventricle. Simultaneously, the left main bundle branch carries the stimulus down the interventricular septum to the muscle mass of the left ventricle, also by way of the Purkinje fibers.

This sequence of stimulation of the heart is the normal basic process. Disturbances of this sequence may produce abnormalities in the rhythm of the heart (the cardiac arrhythmias) or abnormalities in cardiac conduction (SA block, AV heart block, or bundle branch block).

Review Question—Chapter 1

1. Label the major parts of the cardiac conduction system in this diagram and then trace the spread of the normal cardiac stimulus from atria to ventricles.



Answer

1. See Fig. 1-1.

2 Basic ECG Waves

DEPOLARIZATION AND REPOLARIZATION

In Chapter 1 we used the general term "electrical stimulation" to refer to the spread of electrical stimuli through the atria and ventricles. The technical term for this cardiac electrical stimulation is ***depolarization***. The return of heart muscle cells to their resting state following stimulation (depolarization) is called ***repolarization***. These terms are derived from the fact that the normal myocardial cells (atrial and ventricular) are *polarized;* that is, they carry electrical charges on their surface. Fig. 2-1, *A,* shows the resting polarized state of a normal heart muscle cell. Notice that the outside of the resting cell is positive and the inside of the cell is negative (about −90 mV).

When a heart muscle cell is stimulated, it

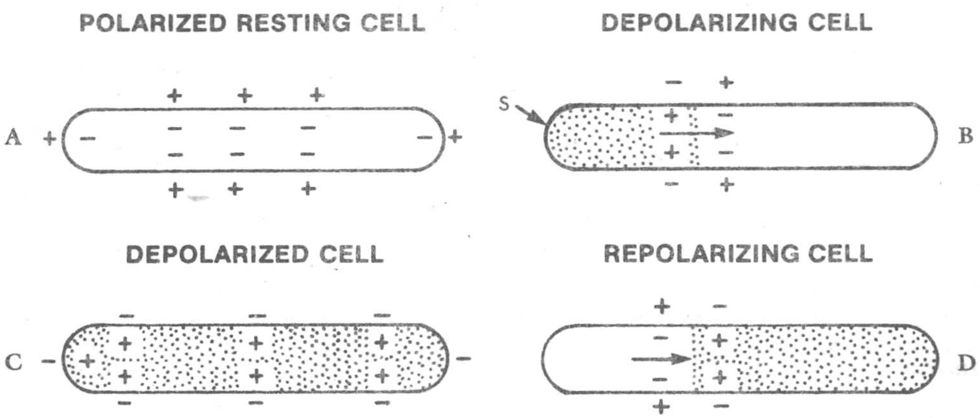


Fig. 2-1. Depolarization and repolarization. The resting heart muscle cell, **A,** is polarized; that is, it carries an electrical charge, with the outside of the cell positively charged and the inside negatively charged. When the cell is stimulated *(S),* as in **B,** it begins to depolarize *(stippled area)*. The fully depolarized cell, **C,** is positively charged on the inside and negatively charged on the outside. Repolarization, **D,** occurs when the stimulated cell returns to the resting state. The direction of depolarization and repolarization is represented by *arrows*. Depolarization (stimulation) of the atria produces the P wave on the ECG, whereas depolarization of the ventricles produces the QRS complex. Repolarization of the ventricles produces the ST-T complex.

depolarizes. As a result, the outside of the cell, in the area where the stimulation has occurred, becomes negative, while the inside of the cell becomes positive. This produces a difference in electrical voltage on the outside of the cell between the stimulated depolarized area and the unstimulated polarized area (Fig. 2-1, *B*). As a result, a small electrical current is formed. This electrical current spreads along the length of the cell as stimulation and depolarization occur until the entire cell is depolarized (Fig. 2-1, *C*). The path of depolarization can be represented by an arrow, as shown in Fig. 2-1. For individual myocardial cells (fibers) depolarization and repolarization proceed in the same direction. However, for the entire myocardium depolarization proceeds from innermost layer (endocardium) to outermost layer (epicardium) while repolarization proceeds in the opposite direction. The mechanism of this difference is not well understood.

This depolarizing electrical current is recorded by the ECG as a *P wave* (when the atria are stimulated and depolarize) and as a *QRS complex* (when the ventricles are stimulated and depolarize).

After a period of time, the fully stimulated and depolarized cell begins to return to the resting state. This is known as repolarization. A small area on the outside of the cell becomes positive again (Fig. 2-1, *D*). The repolarization spreads along the length of the cell until the entire cell is once again fully repolarized. Ventricular repolarization is recorded by the ECG as the *ST segment, T wave,* and *U wave.* (Atrial repolarization is usually obscured by ventricular potentials, as will be discussed later.)

The ECG records the electrical activity of a large mass of atrial and ventricular cells, not just the electrical activity of a single cell. Since cardiac depolarization and repolarization normally occur in a synchronized fashion, the ECG is able to record these electrical currents as specific waves (P wave, QRS complex, ST segment, T wave, and U wave).

To summarize, regardless of whether the ECG is normal or abnormal, it merely records two basic events: (1) depolarization, the spread of a stimulus through the heart muscle, and (2) repolarization, the return of the stimulated heart muscle to the resting state.

BASIC ECG COMPLEXES: P, QRS, ST, T, AND U WAVES

This spread of a stimulus through the atria and ventricles and the return of the stimulated atrial and ventricular muscle to the resting state produce, as noted previously, the electrical currents recorded on the ECG. Furthermore, each phase of cardiac electrical activity produces a specific wave or complex (shown in Fig. 2-2). These basic ECG waves are labeled alphabetically and begin with the P wave.

P wave: atrial depolarization (stimulation)
QRS complex: ventricular depolarization (stimulation)
ST segment, T wave, U wave } ventricular repolarization (recovery)

The P wave represents the spread of a stimulus through the atria (atrial depolarization). The QRS complex represents the spread of a stimulus through the ventricles (ventricular depolarization). The ST segment and T wave represent return of the stimulated ventricular muscle to the resting state (ventricular repolarization). The U wave is a small deflection sometimes seen just after the T wave. It represents the final phase of ventricular repolarization, although its exact significance is not known.

You are probably wondering why there is no wave or complex representing the return of the stimulated atria to the resting state. The atrial ST segment (STa) and atrial T wave (Ta) are generally not observed on the normal ECG because of their low amplitudes.

(An important exception is discussed on p. 147.) Similarly the routine ECG is not sensitive enough to record any electrical activity during the spread of the stimulus through the AV junction (AV node and bundle of His). The spread of the electrical stimulus through the AV junction occurs between the beginning of the P wave and the beginning of the QRS complex. This interval, which is known as the PR interval, is a measure of the time it takes for the stimulus to spread through the atria and pass through the AV junction.

To summarize, the P-QRS-ST-T-U sequence represents the repetitive cycle of the electrical activity in the heart, beginning with the spread of a stimulus through the atria (P wave) and ending with the return of the stimulated ventricular muscle to the resting state (ST-T-U sequence). As shown in the rhythm strip in Fig. 2-3, this cardiac cycle repeats itself again and again.

BASIC ECG COMPLEXES

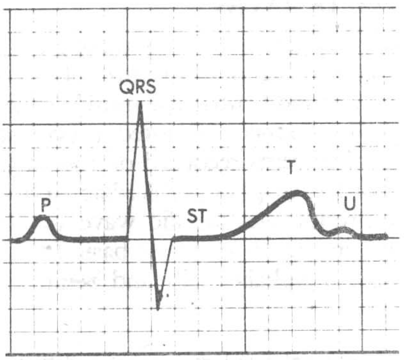


Fig. 2-2. The P wave represents atrial depolarization. The PR interval is the time from initial stimulation of the atria to initial stimulation of the ventricles. The QRS represents ventricular depolarization. The ST segment, T wave, and U wave are produced by ventricular repolarization.

ECG PAPER

The P-QRS-T sequence is recorded on special ECG paper (shown in Figs. 2-3 and 2-4). This paper is divided into gridlike boxes. Each of the small boxes is 1 millimeter square (1 mm^2). The paper usually moves out of the electrocardiograph at a speed of 25 mm/sec. Therefore, horizontally, each millimeter of the ECG paper is equal to 0.04 second (25 mm/sec $\times$ 0.04 sec = 1 mm). Notice also that between every five boxes there are heavier lines, so each of the 5 mm units horizontally corresponds to 0.2 second (5 $\times$ 0.04 = 0.2).

Fig. 2-3. Cardiac cycle. The basic cycle (P-QRS-T) repeats itself again and again.