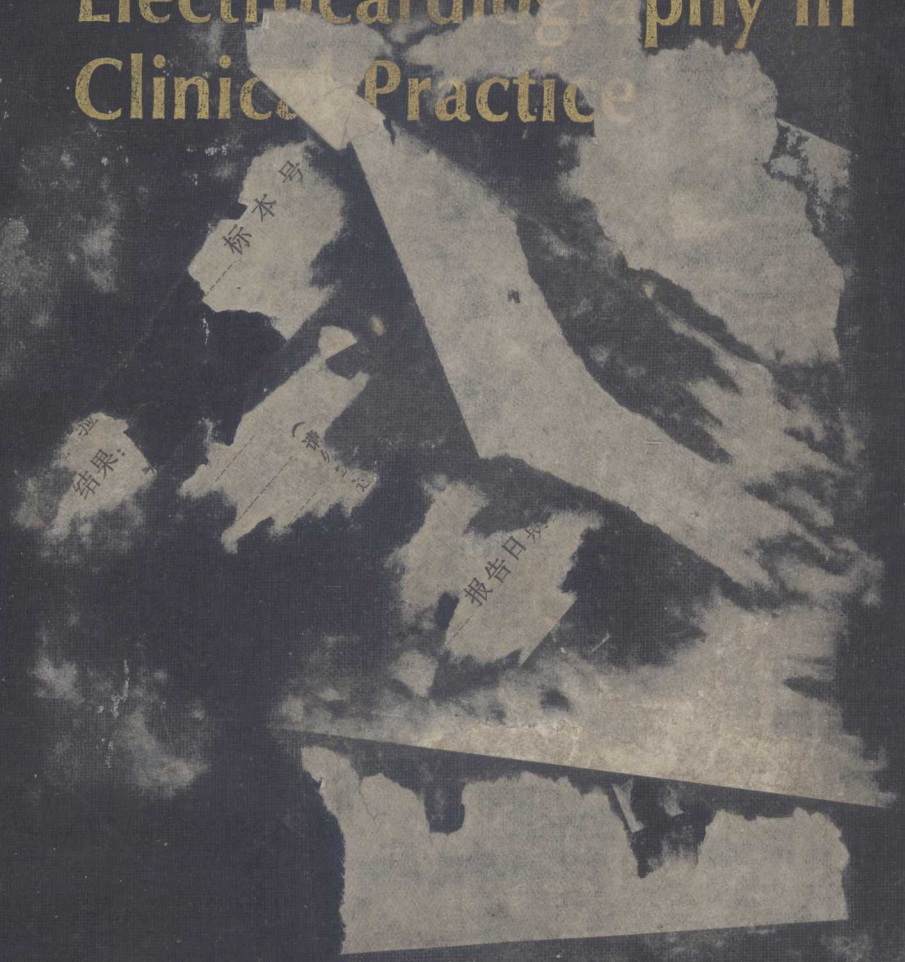
third edition

Electrocardiography in Clinical Practice



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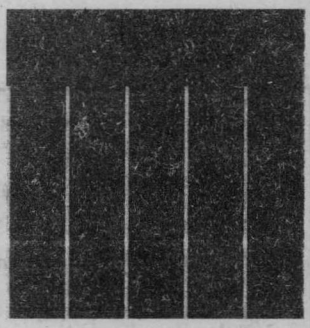

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FOREWORD FROM THE FIRST EDITION

I am told that the purpose of a foreword is to say something good about a forthcoming book that the author would be too modest to say himself. In the case of Dr. Chou's new book this is easy to do. Dr. Chou, with Dr. Robert Helm, has already written a best seller in the field of vectorcardiography. I predict that this publication will be equally successful. One's first reaction upon hearing the title of a new book on electrocardiography is "What? Who needs another book on electrocardiography?", but this is not just another book on electrocardiography. It will be unique in the field. It is the kind of book that I would like to have written myself because I have long believed that there is a need for this kind of presentation.

Dr. Chou does not take the usual approach of a textbook on electrocardiography; namely, that of a discussion of electrophysiology, followed by the theory of the abnormalities in various major disease states, followed by a series of a small number of clinical examples. Rather, he has written a book of clinical correlations that is extensively illustrated.

Dr. Chou's book deals with clinical electrocardiographic correlations in every major field of medicine, ranging from the common ones of ventricular hypertrophy, myocardial infarction, and T-wave abnormalities to the more uncommon ones of hypothermia, and changes with cerebrovascular accidents.

I have read Dr. Chou's manuscript carefully, and would make the following comments: It is clear, well-written, inclusive, and extensively referenced. I think that everyone who is interested in clinical electrocardiography will find something of value in Dr. Chou's book, even the expert cardiologist. It is so well-referenced that it provides a source for intensive reading on any given clinical subject.

Dr. Chou has had a quarter of a century of experience in clinical-electrocardiographic correlations, correlations with autopsy findings, and correlations evaluated by clinical investigation in the Division of Cardiology of the University of Cincinnati. He has also had many years of interaction with Dr. Samuel Kaplan's group in pediatric cardiology at the Cincinnati Children's Hospital. His extensive knowledge, research, and wide reading are clearly evidenced in this book

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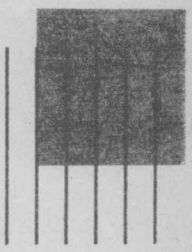
PREFACE TO THE THIRD EDITION



In keeping with the goals of previous editions, the third edition of *Electrocardiography in Clinical Practice* continues to emphasize the value and limitations of the electrocardiogram, the differential diagnosis of the electrocardiographic findings, and their correlation with the clinical and anatomical data. The chapter on myocardial infarction, injury, and ischemia has been extensively revised. The clinical and anatomical correlates of Q-wave and non–Q-wave infarction are discussed. Electrocardiographic changes after coronary reperfusion, prognostic value of the electrocardiogram in myocardial infarction, and the subject of silent myocardial ischemia have been added.

In the sections on cardiac arrhythmias, the discussion on the differential diagnosis of narrow and wide QRS complex tachycardia, especially the latter, has been expanded. The newer antiarrhythmic agents such as encainide, flecainide, propafenone, and adenosine, as well as the proarrhythmic effects of the antiarrhythmic drugs in general, are included.

A new chapter on traumatic heart disease and cardiac transplantation has been added to this edition. Some changes have been made in the discussion of artificial electronic pacemakers in keeping with the advances in pacemaker technology. A total of 54 new illustrations have been added.

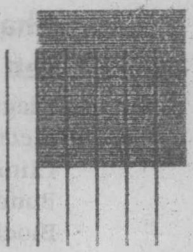
It is hoped that this text will continue to serve as a reminder that the electrocardiogram, if used properly, is still one of the most important diagnostic tools in modern medicine.

I would like to thank Dr. Noble O. Fowler, Professor Emeritus of Medicine, Division of Cardiology, University of Cincinnati College of Medicine, for reviewing Chapter 26. As during the preparation of previous editions of this text, his comments and suggestions were most helpful. Drs. Winston Gaum and David Schwartz have kindly allowed me to use some of their patients' electrocardiograms from the Children's Hospital Medical Center, Cincinnati. Ms. Zenia Frantz has helped me in selecting instructive tracings from our Holter Laboratory. My other colleagues and fellows in the Division of Cardiology have continued to provide me with new case materials.

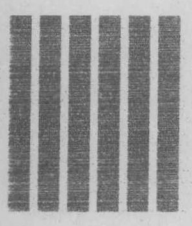
Mrs. Jean Johnson typed the manuscript and organized the references. Her many years of excellent secretarial support are appreciated.

TE-CHUAN CHOU, M.D.

CONTENTS



I NORMAL AND ABNORMAL ELECTROCARDIOGRAMS



NORMAL AND ABNORMAL ELECTROCARDIOGRAMS

NORMAL AND ABNORMAL ELECTROCARDIOGRAMS

Normal Electrocardiogram

1

P WAVE

Atrial activation begins in the sinoatrial (SA) node and spreads in a radial fashion to depolarize the right atrium, the interatrial septum, and then the left atrium[4,12] (Fig. 1–1A). The last region of the left atrium to be activated is the tip of the left atrial appendage, or in the posteroinferior left atrium beneath the left inferior pulmonary vein.[4] Three specialized pathways that contain Purkinje fibers have been identified to connect the SA node to the atrioventricular (AV) node. They are called the anterior, the middle, and the posterior internodal pathways. An interatrial pathway, the Bachmann bundle, connects the right and left atria. The role of these fibers in human atrial impulse conduction under normal circumstances is still unclear, however. For practical purposes, the early part of the P wave may be considered to represent the electrical potential generated by the right atrium, the late portion by the left atrium, and the midportion by both atria and the interatrial septum.

The results of recent studies of sinus node electrograms suggest that the sinus pacemaker may sometimes shift spontaneously within the SA node.[16] Atrial epicardial mapping during cardiac surgery also revealed that, in patients with "sinus" rhythm, the pacemaker impulse is sometimes initiated in the right atrium outside of the sinus node and may be multicentric in origin.[4] These findings may explain, at least in part, the changes in the morphology of the P waves often seen in healthy subjects during sinus rhythm.

In normal adults, the duration of the P wave, which represents the duration of the atrial activation, varies from 0.08 to 0.11 second.[6] In a study that involved a large population, a significant number of subjects had a duration of 0.12 to 0.13 second. Lepeschkin gives a mean duration of 0.085 second with a standard deviation of 0.015 second.[28] For practical purposes, a value above 0.11 second can usually be considered as abnormal.[19]

The P-wave axis in the frontal plane is directed inferiorly and leftward, which correlates well with the general direction of the spread of atrial excitation. The P axis determined from the limb leads varies from 0° to +75°,[24] with most falling between +45° and +60°. Therefore, the P wave is always upright in leads I and II and inverted in lead aVR. In lead III, it may be upright, diphasic, or inverted. When it is diphasic, the initial deflection is positive and the second component is negative. The P wave in lead aVL is also variable in polarity. A negative P wave is relatively common in this lead. When it is diphasic, it has a negative–positive deflection. In lead aVF, the P wave is usually upright, but a diphasic or flat P wave may occasionally be seen.

In the precordial leads, the P wave is often diphasic in leads V_1 and V_2. The early right atrial forces are directed anteriorly and the late left atrial forces posteriorly. Therefore, the diphasic P wave has a positive–negative configuration. An entirely positive or negative deflection may also be seen in lead V_1, but the P wave in lead V_2 is seldom entirely negative. In the rest of the precordial leads, the P wave is always upright because of the essentially right-to-left spread of the atrial activation impulse.

The normal P wave may show small notches or slurring. This is probably related to the transition from right atrial to left atrial depolarization. Some claim that, when the normal P wave is notched, the interval between the two peaks should not exceed 0.03 second.[52] In my experience, however, many healthy young individuals with notched P waves may have a peak-to-peak interval greater than this value, although it seldom reaches 0.05 second.

The amplitude of the P wave in the limb leads seldom exceeds 0.25 mV in normal individuals at rest. In the precordial leads, the positive component of the P wave is less than

0.15 mV. In lead V_1, the negative deflection is usually less than 0.1 mV. The total area of the negative component is a more accurate measure of the normality of the P wave in this lead, however. Morris suggested that the product of the maximum negative amplitude in millivolts and the duration of the negative deflection in seconds should not exceed 0.003.[34] During routine interpretation of the electrocardiogram (ECG), if the area of the negative component of the P wave in lead V_1 occupies one small square or more of the recording paper, the P wave is considered abnormal.

PR INTERVAL

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. The term *PQ interval* is preferred by some electrocardiographers because it is the period actually measured unless the Q wave is absent. Electrophysiologically, the PR interval represents the interval between the onset of atrial depolarization and the onset of ventricular depolarization. In normal AV conduction, it involves the time required for the activation impulse to advance from the atria through the AV node, bundle of His, bundle branches, and the Purkinje fibers until the ventricular myocardium begins to depolarize (see Fig. 1–1B). It does not include the duration of conduction from the SA node proper to the right atrium.

In adults, the normal PR interval is 0.12 to 0.20 second.[28] The most common value is 0.16 second. It is generally shorter in children and longer in older persons. The interval also may become shorter as the sinus rate increases. Although lead II is usually used for this measurement, for the sake of accuracy, the interval should be taken from the lead with the largest and widest P wave and longest QRS duration. Such a selection avoids inaccuracies incurred by using the leads in which the early part of the P wave or QRS complex is isoelectric. Because most modern ECGs record several leads simultaneously, the points of onset of the P wave and the QRS complex can be verified by examining the other simultaneously recorded leads.

With His bundle recording, it has been

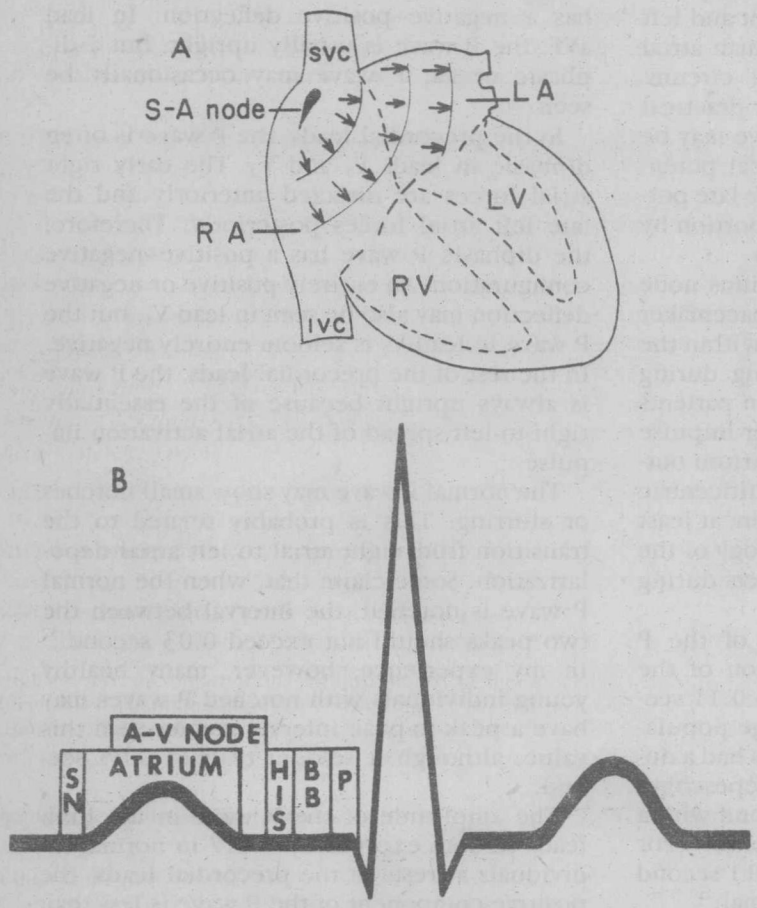


FIGURE 1–1. (A) Schematic diagram of the heart depicting the radial spread of atrial activation from the SA node. (Reproduced from Chou TC, Helm RA, Kaplan S: Clinical Vectorcardiography, 2nd ed, Chapter 3, p 31. New York, 1974, by permission of Grune & Stratton.) (B) Schematic diagram of a P-QRS-T complex illustrating the sequence of activation of the atria and specialized conduction fibers. SN = sinoatrial node; HIS = bundle of His; BB = bundle branches; P = Purkinje fibers. (Reproduced from Damato AN, Lau SH: Clinical value of the electrogram of the conduction system. Prog Cardiovasc Dis 13:119, 1970, by permission of the author and Grune & Stratton.)

shown that most of the AV conduction time is consumed by impulse conduction proximal to the His bundle. The AH time, the time between the intracavitary potential recorded from the lower part of the right atrium and the His bundle spikes, is between 50 and 130 msec.[8,11,36] The HV time, or the interval between the His spikes and the onset of ventricular deflections, is between 35 and 55 msec. Slower conduction through the AV node is mainly responsible for the longer AH time.

The PR segment is the horizontal line between the end of the P wave and the beginning of the QRS complex. Its duration depends on the duration of the P wave as well as the impulse conduction through the AV junction. Although the segment is usually isoelectric, it is often displaced in a direction opposite to the polarity of the P wave. It is, therefore, depressed in most of the conventional leads except lead aVR. The displacement is mainly due to atrial repolarization. In normal subjects, the amount of PR segment depression is usually less than 0.8 mm and the amount of elevation is less than 0.5 mm.[9] Taller P waves are more likely to be associated with a greater degree of PR-segment depression and smaller P waves with lesser PR-segment changes.

QRS COMPLEX

Ventricular Activation

The QRS complex represents the resultant electrical forces generated from ventricular depolarization. In the normal sequence of activation, the ventricular excitation begins at the middle third of the left interventricular septal surface. From there, the impulse spreads in a rightward direction. Using isolated human hearts, Durrer and associates[12] demonstrated early left ventricular activation also in the anterior and posterior paraseptal areas. The forces generated from these two areas are, however, in opposite directions and are mostly neutralized by each other. The right ventricle begins to depolarize shortly after the initiation of left ventricular activation. The excitation starts at the right septal surface near the base of the anterior papillary muscle, and the impulse spreads leftward. Because a much larger area of the left septum is depolarized in comparison with that of the right septum, the net effect of the septal activation is from the left toward the right.

Soon after the beginning of the septal activation, the impulse arrives at most of the subendocardial layer of the myocardium of the apical and free wall of both ventricles through the Purkinje network. Within the inner layers of the ventricular wall, because of the deep penetration of the Purkinje fibers into the myocardium, the activation waves spread in all directions. Consequently, their effects cancel each other and little potential can be detected outside the area. The thickness of the layers to be activated in this manner has been estimated to be one third to two thirds of the entire wall. In the outer layers of the ventricles, the impulse spreads uniformly in the endocardial-to-epicardial direction. Because of the much larger muscle mass of the left ventricle, the result of its activity dominates that of the right ventricle.

The last areas of the ventricles to be depolarized are the basal portion of the septum and the posterobasal portion of the free wall of the left ventricle. The late arrival of the impulse can easily be explained by the rarity of the Purkinje fibers in these areas. The general direction of the spread of impulse in the ventricular wall is outward and backward. In the basal septum, the wave proceeds toward the base and moves from the left to the right.

In summary, because of its larger muscle mass the left ventricle contributes most of the QRS forces (Fig. 1–2). The initial left septal activation generates vectors directed rightward and anteriorly. The major ventricular forces from the free wall of the left ventricle are oriented leftward and inferiorly. The late activation of the posterobasal portion of the left ventricle is accompanied by terminal QRS

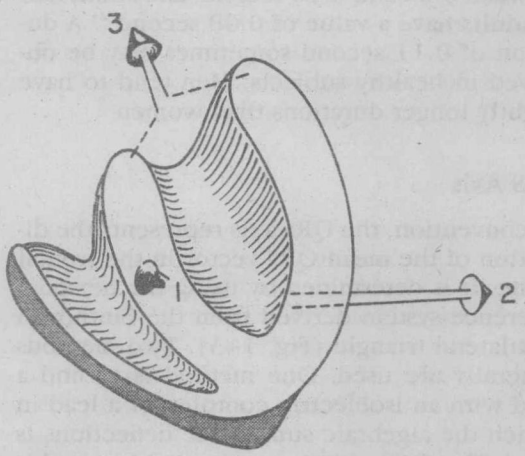


FIGURE 1–2. Representative resultant spatial vectors of ventricular activation. Vector 1 represents the resultant force of the initial septal and paraseptal activation; vector 2, the activation of the free wall of the ventricles; and vector 3, the basal portions of the ventricles.

forces directed posteriorly. Correspondingly, the polarity and amplitude of the various components of the QRS complex in the various leads are determined by the relation between these vectors and the lead axes.

QRS Duration

The duration of the QRS complex represents the duration of ventricular activation. It should be measured from the lead with the widest QRS complex, because in certain leads the initial or terminal vectors may be perpendicular to the lead axis and an isoelectric line is inscribed and the QRS may appear narrow. Although the interval traditionally is measured from the limb leads, the precordial leads, especially leads V_2 and V_3, may have the widest complex. Another difficulty in obtaining an accurate value arises from the fact that ventricular repolarization usually begins before the completion of depolarization. Therefore, there is an overlap and fusion of the terminal part of the QRS and the onset of the ST segment. The end point of the QRS is slurred. This problem is encountered more frequently in the precordial leads. If the leads are recorded simultaneously, the beginning and end of ventricular excitation can be identified more accurately. The interval so determined usually is slightly longer than that obtained from the single-channel trace. With the single-channel recording, it is my practice to choose the widest complex in any lead with the sharpest onset and termination regardless of whether it is a limb lead or a precordial lead.

In normal adults, the QRS duration varies between 0.06 and 0.10 second, and about half of adults have a value of 0.08 second.[24] A duration of 0.11 second sometimes may be observed in healthy subjects. Men tend to have slightly longer durations than women.

QRS Axis

By convention, the QRS axis represents the direction of the mean QRS vector in the frontal plane. It is determined by using the hexaxial reference system derived from the Einthoven equilateral triangle (Fig. 1–3). Two methods generally are used. One method is to find a lead with an isoelectric complex or a lead in which the algebraic sum of the deflections is zero. The QRS axis is perpendicular to this lead, and its positive terminus points toward the limb lead with the largest net positive deflection. The other method is to use the algebraic sum of the deflections in two leads, usually leads I and III, to plot out the axis. The results obtained from these two methods are not necessarily the same. Furthermore, the direction of the axis may be different when a different pair of leads is used. A variance of up to $\pm 35°$ occasionally may be obtained in the same individual.[39] The main reason for the discrepancies is that the three standard limb leads do not form an equilateral triangle and the adjacent axes of the hexaxial reference system are not separated by a 30° angle. The electrical axes of the limb leads form a scalene triangle as described by Burger and van Milaan[5] (Fig. 1–4). A more accurate QRS axis (as well as P and T axes) can be obtained by using the hexaxial reference system derived from it. Langner further improved the reference system by modifying the scales of the leads to compensate for their unequal strength,[25] since the same unit potential applied to the various leads does not give the same magnitude of deflection. Although it is theoretically desirable to use the reference system based on Burger's triangle, most values given in the literature were determined from leads I and III by the traditional method.

The normal range of the QRS axis in the frontal plane is between $-30°$ and $+105°$.[19,43] Most people have values between $+30°$ and $+75°$. A significant difference is found among various age groups, with a leftward shift of the axis in older individuals. In an individual under the age of 30, the axis is seldom superior to 0°. After the age of 40, the axis is seldom to the right of $+90°$. Therefore, for clinical purposes, the normal limits for persons under the age of 40 are 0° to $+105°$, and for those over the age of 40, from $-30°$ to $+90°$. There also is an association between the QRS axis in the frontal plane and body weight. A thin person is likely to have a more vertical axis, and overweight individuals tend to have a more leftward axis. The trend of leftward shift of the axis with aging is particularly pronounced in overweight subjects. There is no significant sex difference in the axis even though older women generally have less leftward shift of the axis.

QRS Morphology and Amplitude

The morphology and amplitude of the QRS complex, as in the case of the QRS axis, are significantly affected by some of the constitutional variables. With advancing age, the amplitude of the QRS complex decreases, but the changes are less apparent after the age of 40.[17,40] Men generally have a larger QRS am-