Rapid ECG Interpretation

Contemporary Cardiology

Christopher P. Cannon, MD
Series Editor

Annemarie M. Armani, MD
Executive Editor

Nuclear Cardiology: *The Basics: How to Set Up and Maintain a Laboratory, Second Edition,* by *Frans Wackers, MD, PhD, Barry L. Zaret, MD, PhD, and Wendy Bruni, CNMT, 2008*

Rapid ECG Interpretation, *Third Edition,* by *M. Gabriel Khan, MD, FRCP (London), FRCP(C), FACP, FACC, 2008*

Therapeutic Lipidology, edited by *Michael H. Davidson, MD, Kevin C. Maki, PhD, and Peter P. Toth, MD, PhD, 2007*

Essentials of Restenosis: *For the Interventional Cardiologist,* edited by *Henricus J. Duckers, PhD, MD, Patrick W. Serruys, MD, and Elizabeth G. Nabel, MD, 2007*

Cardiac Drug Therapy, *Seventh Edition,* by *M. Gabriel Khan, MD, FRCP (London), FRCP(C), FACP, FACC, 2007*

Cardiovascular Magnetic Resonance Imaging, edited by *Raymond Y. Kwong, MD, 2007*

Essential Echocardiography: *A Practical Handbook With DVD,* edited by *Scott D. Solomon, MD, 2007*

Cardiac Rehabilitation, edited by *William Kraus, MD, and Steven Keteyian, MD, 2007*

Management of Acute Pulmonary Embolism, edited by *Stavros Konstantinides, MD, 2007*

Stem Cells and Myocardial Regeneration, edited by *Marc S. Penn, MD, PhD, 2007*

Handbook of Complex Percutaneous Carotid Intervention, edited by *Jacqueline Saw, MD, Jose Exaire, MD, David S. Lee, MD, Sanjay Yadav, MD, 2007*

Preventive Cardiology: *Insights Into the Prevention and Treatment of Cardiovascular Disease, Second Edition,* edited by *JoAnne Micale Foody, MD, 2006*

The Art and Science of Cardiac Physical Examination: *With Heart Sounds and Pulse Wave Forms on CD,* by *Narasimhan Ranganathan, MD, Vahe Sivaciyan, MD, and Franklin B. Saksena, MD, 2006*

Cardiovascular Biomarkers: *Pathophysiology and Disease Management,* edited by *David A. Morrow, MD, 2006*

Cardiovascular Disease in the Elderly, edited by *Gary Gerstenblith, MD, 2005*

Platelet Function: *Assessment, Diagnosis, and Treatment,* edited by *Martin Quinn, MB BCh BAO, PhD, and Desmond Fitzgerald, MD, FRCPI, FESC, APP, 2005*

Diabetes and Cardiovascular Disease, *Second Edition,* edited by *Michael T. Johnstone, MD, CM, FRCP(C), and Aristidis Veves, MD, DSc, 2005*

Angiogenesis and Direct Myocardial Revascularization, edited by *Roger J. Laham, MD, and Donald S. Baim, MD, 2005*

Interventional Cardiology: *Percutaneous Noncoronary Intervention,* edited by *Howard C. Herrmann, MD, 2005*

Principles of Molecular Cardiology, edited by *Marschall S. Runge, MD, and Cam Patterson, MD, 2005*

Heart Disease Diagnosis and Therapy: *A Practical Approach, Second Edition,* by *M. Gabriel Khan, MD, FRCP(LONDON), FRCP(C), FACP, FACC, 2005*

Cardiovascular Genomics: *Gene Mining for Pharmacogenomics and Gene Therapy,* edited by *Mohan K. Raizada, PhD, Julian F. R. Paton, PhD, Michael J. Katovich, PhD, and Sergey Kasparov, MD, PhD, 2005*

Surgical Management of Congestive Heart Failure, edited by *James C. Fang, MD and Gregory S. Couper, MD, 2005*

Cardiopulmonary Resuscitation, edited by *Joseph P. Ornato, MD, FACP, FACC, FACEP and Mary Ann Peberdy, MD, FACC, 2005*

CT of the Heart: *Principles and Applications,* edited by *U. Joseph Schoepf, MD, 2005*

Coronary Disease in Women: *Evidence-Based Diagnosis and Treatment,* edited by *Leslee J. Shaw, PhD and Rita F. Redberg, MD, FACC, 2004*

Cardiac Transplantation: *The Columbia University Medical Center/New York-Presbyterian Hospital Manual,* edited by *Niloo M. Edwards, MD, Jonathan M. Chen, MD, and Pamela A. Mazzeo, 2004*

Heart Disease and Erectile Dysfunction, edited by *Robert A. Kloner, MD, PhD, 2004*

Complementary and Alternative Cardiovascular Medicine, edited by *Richard A. Stein, MD and Mehmet C. Oz, MD, 2004*

Nuclear Cardiology, The Basics: *How to Set Up and Maintain a Laboratory,* by *Frans J. Th. Wackers, MD, PhD, Wendy Bruni, BS, CNMT, and Barry L. Zaret, MD, 2004*

Minimally Invasive Cardiac Surgery, *Second Edition,* edited by *Daniel J. Goldstein, MD, and Mehmet C. Oz, MD 2004*

Cardiovascular Health Care Economics, edited by *William S. Weintraub, MD, 2003*

Platelet Glycoprotein IIb/IIIa Inhibitors in Cardiovascular Disease, *Second Edition,* edited by *A. Michael Lincoff, MD, 2003*

Heart Failure: *A Clinician's Guide to Ambulatory Diagnosis and Treatment,* edited by *Mariell L. Jessup, MD and Evan Loh, MD, 2003*

Management of Acute Coronary Syndromes, *Second Edition,* edited by *Christopher P. Cannon, MD 2003*

Aging, Heart Disease, and Its Management: *Facts and Controversies,* edited by *Niloo M. Edwards, MD, Mathew S. Maurer, MD, and Rachel B. Wellner, MPH, 2003*

Peripheral Arterial Disease: *Diagnosis and Treatment,* edited by *Jay D. Coffman, MD and Robert T. Eberhardt, MD, 2003*

Cardiac Repolarization: *Bridging Basic and Clinical Science,* edited by *Ihor Gussak, MD, PhD, Charles Antzelevitch, PhD, Stephen C. Hammill, MD, Win K. Shen, MD, and Preben Bjerregaard, MD, DMSc, 2003*

Rapid ECG Interpretation

Third Edition

M. Gabriel Khan, MD, FRCP (London), FRCP(C), FACP, FACC

Associate Professor of Medicine, University of Ottawa
Cardiologist, The Ottawa Hospital
Ottawa, Ontario, Canada

With a Foreword by
Christopher P. Cannon, MD
TIMI Study Group, Brigham and Women's Hospital
Harvard Medical School
Boston, MA

Humana Press

M. Gabriel Khan, MD, FRCP
(LONDON), FRCP (C), FACP, FACC
Associate Professor of Medicine
University of Ottawa Cardiologist
The Ottawa Hospital
Ottawa, Ontario
Canada

ISBN 978-1-58829-979-6 e-ISBN 978-1-59745-408-7
DOI 10.1007/978-1-59745-408-7
Springer Dordrecht Heidelberg London New York

Library of Congress Control Number: 2007922066

Printed on acid-free paper

(Corrected at 2nd printing 2010)

Humana Press is part of Springer Science+Business Media (www.springer.com)

To my wife, Brigid

Foreword

The electrocardiogram (ECG) is the first test performed on most cardiac patients–one that helps make the first part of the diagnosis and one that can frequently direct treatment decisions. Thus, for any physician, a solid understanding of the ECG is critical. Learning the basics and subtleties of the ECG is a right of passage for all physicians and healthcare providers during their training.

So, what would we want from a book on ECGs? Ideally, such a book would be comprehensive, yet concise, practically oriented, and explain pathophysiology and its application to practice.

Dr. Khan has written such a book. *Rapid ECG Interpretation* is comprehensive, yet concise, and very practically oriented. More important, it takes a step-by-step approach, walking the reader through a thorough evaluation of the ECG. This, as many of us have been taught, is the "right" way to look at an ECG. This edition includes a new opening chapter that covers basic concepts. This quickly orients the reader to the physiology, anatomy, and geometry of the electrical system of the heart.

After reviewing each component of the ECG, the next section describes the unique ECG patterns of specific cardiac conditions, including pulmonary embolism and long QT syndrome. This is followed by a chapter with each of the arrhythmias. Finally, Dr. Khan includes an invaluable section—an ECG Board Review and Self-Assessment Quiz. With this, the reader can really see if the basic concepts and ECG fundamentals have been learned.

Dr. Khan is to be congratulated on an outstanding text that will help readers at all levels become very familiar and facile in rapid interpretation of the ECG.

Christopher P. Cannon, MD
TIMI Study Group, Brigham and Women's Hospital
Harvard Medical School, Boston, MA

Preface

A new approach for the interpretation of the electrocardiogram (ECG), *a step-by-step method for the accurate interpretation of the ECG,* is outlined in this text.

The most important addition in the second edition of *Rapid ECG Interpretatio*n was a new chapter, Basic Concepts. This chapter gives considerable practical details with 16 instructive illustrations so that the reader can fully understand the genesis of each wave and deflection of the ECG and the reason 12 carefully positioned leads are needed to capture 12 views of the heart's electrical currents and vector forces. Also, more than 35 new ECG tracings were added to the chapters that discuss topics that will be of value to postgraduates and internists.

The major addition in this third edition is a new chapter: ECG Board Self-Assessment Quiz. The chapter provides 90 selected ECG tracings that should sharpen the skills of all who wish to interpret ECGs. This small-volume text contains more than 320 ECGs and instructive illustrations.

The ECG is the oldest cardiologic test, but even 100 years after its inception, it continues as the most commonly used cardiologic test. Despite the advent of expensive and sophisticated alternatives, the ECG remains the most reliable tool for the confirmation of acute myocardial infarction (MI). The ECG—not CK-MB, troponins, echocardiogram, or SPECT or PET scan—dictates the timely administration of life-saving PCI or thrombolytic therapy. There is no test to rival the ECG in the diagnosis of arrhythmias, which is a common and bothersome clinical cardiologic problem. Also, the clinical diagnosis of pericarditis and myocardial ischemia is made mainly by ECG findings.

This text gives a systematic step-by-step approach but departs somewhat from the conventional sequence and gives steps that are consistent with the changes in cardiology practice that have evolved over the past decade. The early diagnosis of acute MI depends on astute observation for ST segment changes. New terms have emerged: ST elevation MI and non–ST elevation MI (non–Q wave MI). The ST segment holds the key to the diagnosis. Currently, ambulance crews are being trained in Europe, the United States, and Canada to recognize ST segment abnormalities and to make the diagnosis of ST elevation MI (STEMI)

and non–ST elevation MI. Thus, patients can be rapidly shuttled to special cardiac centers for coronary angiography and angioplasty/stent or thrombolytic therapy; rapid triage in emergency rooms is crucial. These lifesaving measures depend on the accurate and rapid interpretation of the ECG by clinicians who must be adequately trained to interpret tracings.

This text describes ST segment abnormalities in detail. For example, the recent observation that ST segment elevation in lead aVR (a commonly ignored lead) is a marker for left main coronary artery (LMCA) occlusion is of lifesaving value. Because LMCA occlusion is a serious condition, any noninvasive diagnostic clue represents a valuable addition to our armamentarium. Thus, only after detailed assessment of the ST segment is completed are the QRS complex, T waves, atrial and ventricular hypertrophy, and lastly the axis assessed. This change in the analytical sequence is necessary so that the most crucial diagnoses can be made accurately and rapidly.

In addition, the standard teaching is for the interpreter to assess all leads and all deflections and waves before entertaining diagnoses. This text gives presumptive diagnoses as soon as a clue is uncovered in the tracing. Also, a few rare but life-threatening conditions are excluded early in the assessment sequence. For example, although Wolff-Parkinson-White (WPW) syndrome is uncommon, it is an important diagnosis that may be missed by computer analysis and by physicians. Because WPW syndrome is a result of widening of the QRS complex, it is logical to consider this diagnosis in the same framework as bundle branch blocks; this approach avoids the danger and embarrassment of missing the diagnosis. No text considers WPW syndrome in the assessment of the 10 essential ECG features. Most important, it is imperative to exclude mimics of MI early in the sequence. WPW syndrome may mimic MI. Right bundle branch block (RBBB) may reveal Q waves in leads III and aVF that may be erroneously interpreted as MI. Left bundle branch block (LBBB) may mimic MI and must be quickly documented because its presence hinders the accurate diagnosis of acute coronary syndromes. Furthermore, the ECG manifestation of acute MI may be a new LBBB pattern. Thus, the assessment for blocks is performed early, in step 2 of the 11 steps outlined.

Because RBBB and LBBB are best revealed in leads V_1 and V_2, the clinician is advised to screen these leads before assessing other leads. The text advises the clinician or senior resident that the assessment of V_1 and V_2 may assist with the diagnosis of Brugada syndrome and right ventricular dysplasia, which may display particular forms of right

bundle branch block and recently have been shown to be causes of sudden death in young adults. Many rare syndromes are described in medicine, but those that cause sudden death should be made familiar to trainees and clinicians. We should not fear divulging information about such rare syndromes at an early stage to students and residents, because these topics may serve to motivate them to higher levels of excellence.

This text presents a unique 11-step method for accurate and rapid ECG interpretation in a user-friendly synopsis format. Medical house staff should welcome this step-by-step method, because it simplifies ECG interpretation and provides for greater accuracy than the approaches given in texts published over the past 50 years. The succinct writing style allows a wealth of information to be presented in a small text that is highlighted with bullets to allow for rapid retrieval. The 11 steps are illustrated in algorithms and outlined in Chapter 2 with references to later chapters, each of which expands on one of the steps and provides advanced material for senior internal medicine residents, cardiology residents, and internists. The text moves rapidly from basics to advanced material.

All diagnostic ECG criteria are given with relevant and instructive ECGs, providing a quick review or refresher for proficiency tests and for physicians preparing for the ECG section of the Cardiovascular Diseases Board Examination. This text can be a valuable tool for all those who wish to be proficient in the interpretation of ECGs.

M. Gabriel Khan

Acknowledgments

I had the privilege of borrowing several ECG tracings from *Electrocardiography in Clinical Practice* by the late Dr. Te-Chuan Chou and from *Practical Electrocardiography* by Dr. Henry J.L. Marriott; I am grateful to these authors. A special note of thanks to my editor, Paul Dolgert.

About the Author

Dr. M. Gabriel Khan is a cardiologist at the Ottawa Hospital and an Associate Professor of Medicine, at the University of Ottawa. Dr. Khan graduated MB, BCh, with First-Class Honours at The Queen's University of Belfast. He was appointed Staff Physician in charge of a Clinical Teaching Unit at the Ottawa General Hospital and is a Fellow of the American College of Cardiology, the American College of Physicians, and the Royal College of Physicians of London and Canada. He is the author of *On Call Cardiology*, 3rd ed., Elsevier, Philadelphia (2006); *Heart Disease Diagnosis and Therapy*, 2nd ed., Humana Press (2006); *Cardiac and Pulmonary Management*, Elsevier, Philadelphia, PA (1993), *Medical Diagnosis and Therapy* (1994), *Heart Attacks, Hypertension and Heart Drugs* (1986), *Heart Trouble Encyclopedia* (1996), and *Encyclopedia of Heart Diseases* (2006), Academic Press/Elsevier, San Diego; and *Cardiac Drug Therapy*, 7th ed., Humana Press (2007).

Dr. Khan's books have been translated into Chinese, Czech, Farsi, French, German, Greek, Italian, Japanese, Polish, Portuguese, Russian, Spanish, and Turkish. He has built a reputation as a clinician-teacher and has become an internationally acclaimed cardiologist through his writings.

His peers have acknowledged the merits of his books by their reviews of *Cardiac Drug Therapy*: Review of the 5th edition in *Clinical Cardiology*: "this is an excellent book. It succeeds in being practical while presenting the major evidence in relation to its recommendations. Of value to absolutely anyone who prescribes for cardiac patients on the day-to-day basis. From the trainee to the experienced consultant, all will find it useful. The author stamps his authority very clearly throughout the text by very clear assertions of his own recommendations even when these recommendations are at odds with those of official bodies. In such situations the 'official' recommendations are also stated but clearly are not preferred."

And for the fourth edition a cardiologist reviewer states that it is "by far the best handbook on cardiovascular therapeutics I have ever had the pleasure of reading. The information given in each chapter is up-to-date, accurate, clearly written, eminently readable and well referenced."

Contents

1 Basic Concepts

CONTENTS

ELECTRICAL ACTIVITY OF THE HEART

Each contraction of the heart is preceded by excitation waves of electrical activity that originate in the sinoatrial (SA) node. Figure 1-1 depicts the radial spread of activation from the SA node. The waves of electrical activity spread through the atria and reach the atrioventricular (AV) node. Note that the SA node tracing shows no steady resting potential, as does the ventricular muscle tracing. The SA node's spontaneous depolarization and repolarization provides a unique and miraculous automatic pacemaker stimulus that activates the atria and the AV node, which conducts the activation current down the bundle branches to activate the ventricular muscle mass. Cardiac cells outside the SA node normally do not exhibit spontaneous depolarization; thus they must be activated.

Depolarization

In a resting cardiac muscle cell, molecules dissociate into positively charged ions on the outer surface and negatively charged ions on the

From: *Contemporary Cardiology: Rapid ECG Interpretation, 3e*
by: M. Gabriel Khan © Humana Press, a part of
Springer Science+Business Media, LLC 2008

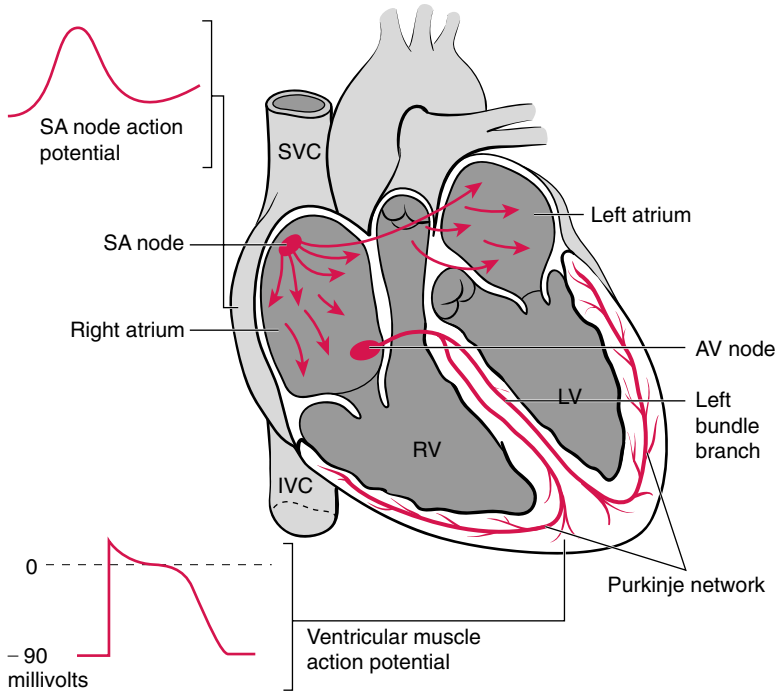

Fig. 1-1. Electrical activation of the heart by the sinoatrial (SA) node. The current of activation (arrows) spreads radially from the SA node across the atria to the atrioventricular (AV) node and down the bundle branches to the ventricular muscle and Purkinje network. The SA node tracing shows no steady resting potential and is characterized by spontaneous depolarization.

inner surface of the cell membrane; the cell is in an electrically balanced or polarized resting state (Fig. 1-2).

- When the cell is stimulated by an excitatory electrical wave, the negative ions migrate to the outer surface of the cell and the positively charged ions pass into the cell; this reversal of polarity is called depolarization (*see* Fig. 1-2).
- If an electrode is placed so that the depolarization wave flows toward the electrode, a galvanometer will record an upward or positive deflection (Fig. 1-3).

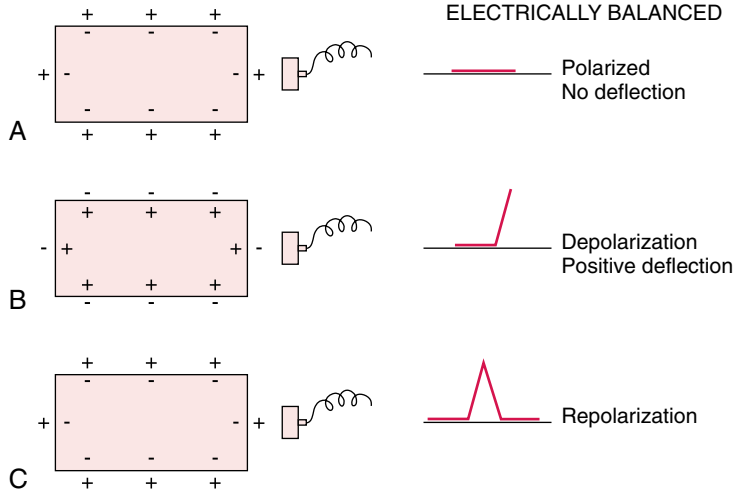

Fig. 1-2. **A**, Resting cell: Positive ions on the outer surface and negative ions inside equal an electrically balanced or polarized cell. **B**, Depolarized cell: Negative ions on the outer surface and positive ions inside. **C**, Repolarization of cell: Positive ions return to the outside.

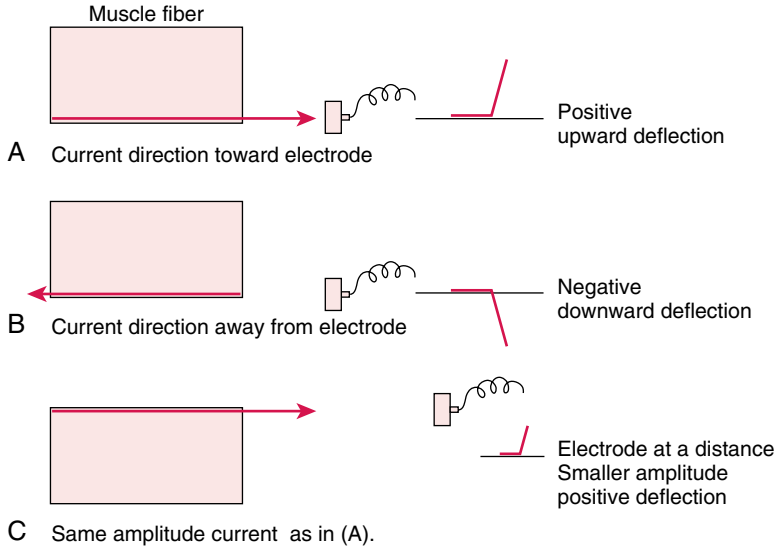

Fig. 1-3. Recording of the effects of electrical activation process. **A**, Current flows toward the electrode produce a positive upward deflection. **B**, Current flows away from the electrode produce a negative deflection. **C**, Current flows toward an electrode placed at a distance produce a positive but smaller amplitude deflection than in (**A**).

- When a depolarization current is directed away from an electrode, a negative or downward deflection is recorded (*see* Fig. 1-3).

Repolarization

- During a recovery period, positively charged ions return to the outer surface and negatively charged ions move into the cell. The electrical balance of the cell is restored; this process is called repolarization (*see* Fig. 1-2).
- The transfer of sodium (Na^+) and potassium (K^+) ions across the cell membrane plays an important role in generating cardiac electrical activity. In Fig. 1-4, the relative magnitudes of the concentration of Na^+ and K^+ ions are indicated. Intracellular concentration of K^+ is 30 times greater than extracellular K^+. Na^+ concentration is 30 times less inside the cell than outside. Because of this ionic composition, the membrane of the resting cardiac fiber is in an electrically balanced or polarized state. The potential difference across the cell membrane can be measured by a microelectrode and is observed on an oscilloscope to be –90 mV.

The Action Potential

- The inward Na^+ current results in a change in transmembrane potential; results in depolarization; and is shown as the upstroke, phase 0 of the action potential. With a decrease in Na^+ and K^+ permeability, the membrane potential remains close to 0; this represents phases 1 and 2 of the action potential (*see* Fig. 1-4). The Na^+-K^+-ATPase (adenosine triphosphatase) sodium pump, depicted in Fig. 1-4, pumps Na^+ from the intracellular to the extracellular fluid compartment; K^+ passes from the extracellular fluid to the intracellular fluid.
- Phase 3 is the phase of rapid repolarization and is followed by a period of stable resting potential, phase 4 of the action potential.

The appreciation of these four phases is important for the understanding of abnormal heart rhythms (arrhythmias) and the therapeutic actions of antiarrhythmics. For example, digoxin or excess catecholamines increase the slope of spontaneous phase 4 depolarization and therefore increase automaticity of ectopic pacemakers (Fig. 1-5); β-blockers cause inhibition or depression of spontaneous phase 4 diastolic depolarization and thus suppress catecholamine-induced arrhythmias, particularly those related to ischemia. Digitalis causes inhibition of the cellular Na^+ pump, which causes increased intracellular Na^+, which is then exchanged for calcium via the Na^+-calcium exchanger. Increased intracellular calcium during cardiac systole increases myocardial muscle contractility. Digitalis toxicity causes cellular calcium overload that potentiates arrhythmias.

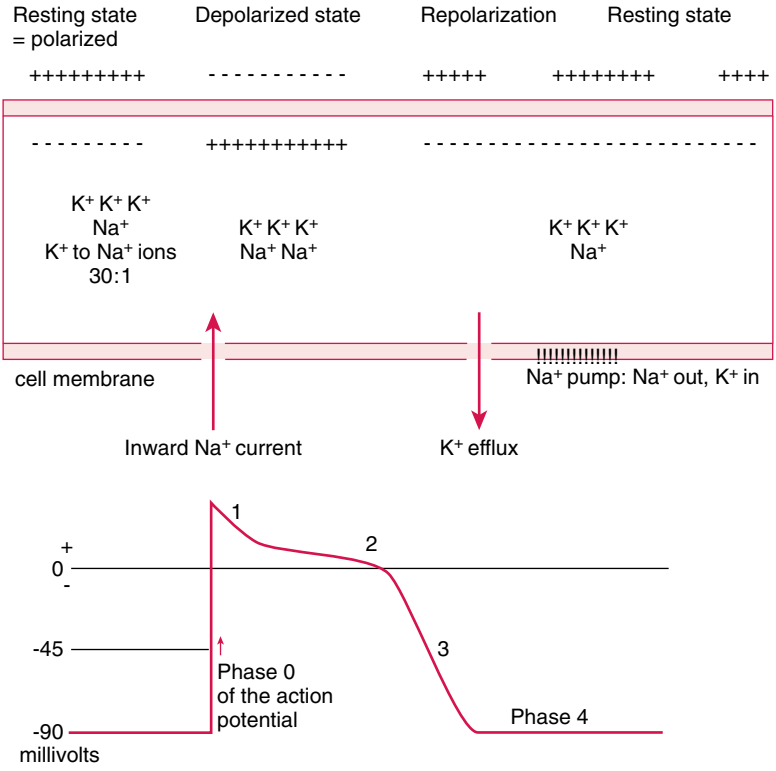

Fig. 1-4. A simplified concept of ionic exchange; the polarized, depolarized, and repolarized state of a myocardial cell; and the action potential. An electrical current arriving at the cell causes positively charged ions to cross the cell membrane, which causes depolarization, followed by repolarization, which generates an action potential: phases 0, 1, 2, 3, and 4. This electrical event traverses the heart and initiates mechanical systole, or the heartbeat (*see also* Fig. 1-7).

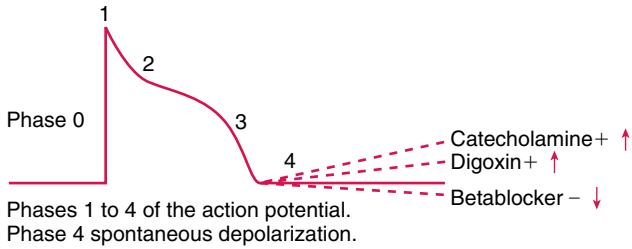

Fig. 1-5. Effects of catecholamines, digoxin, and β-blockers on spontaneous phase 4 depolarization. β-Blockers inhibit or decrease spontaneous phase 4 depolarization caused by catecholamines, especially that caused by ischemia.

Sinoatrial Node

The SA node is unique and has no steady resting potential. After repolarization, slow, spontaneous depolarization occurs during phase 4 that causes the automaticity of the SA node fibers (*see* SA node waveform in Fig. 1-1). Thus, the unique pacemaker provides individuals with an automatic infinitesimal current that sets the heart's electrical activity and contractions. The SA discharge rate, usually 50 to 100 per minute, is under autonomic, chemical, and hormonal influence.

Atrioventricular Node

The AV node provides a necessary physiologic delay of the electrical currents, which allows the atria to fill the ventricles with blood before ventricular systole.

- From the AV node and bundle of His, the excitatory electrical current rapidly traverses the right and left bundle branches, the specialized conductive tissues of the ventricles, and the Purkinje system, and the entire ventricular muscle is depolarized (*see* Fig. 1-1).
- Depolarization spreads down the intraventricular septum toward the apex of the heart and then along the free wall of the left ventricular myocardium; it always proceeds from the endocardium toward the pericardium. The specialized fine arborization of branches that constitute the Purkinje network spreads over the endocardial surfaces of the ventricles.
- The transient halt and slowing of conduction through the specialized AV node fibers play an important protective role in patients with atrial flutter and atrial fibrillation. In these common conditions, a rapid atrial rate of approximately 300 to 600 beats/min reaches the AV node; this AV "tollgate" reduces the electrical traffic that reaches the superhighway that traverses the ventricles to approximately 120 to 180 beats/min, and serious life-threatening events are prevented.

ELECTROCARDIOGRAM

The heart muscle is made up of several thousand muscle elements, about 10^{10} cells. Each instant of depolarization or repolarization represents different stages of activity for a large number of cells. The electrical activity of each element can be represented by a vector force.

- A vector is defined as a force that can be represented by direction and magnitude. The sum total of cardiac vectors is considered the electrical activity of the entire heart (Fig. 1-6). The ECG records the sequence of such instantaneous vectors.

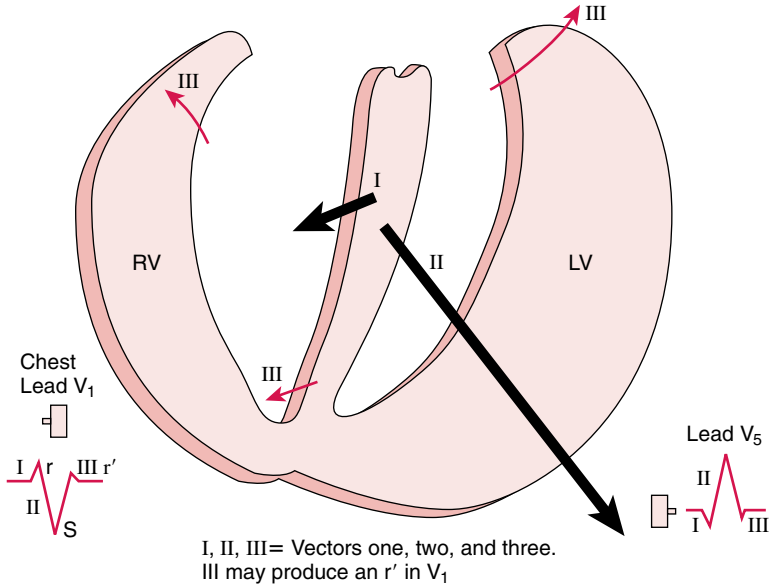

Fig. 1-6. Electrical activation of the heart: resultant vector forces. Vector III may produce an r′ in V_1.

- The heart muscle is arranged in three muscle masses: the intraventricular septum, a large left ventricular muscle mass, and a small right ventricular muscle mass. The magnitude or amplitude of the deflections recorded is influenced by the size of the muscle mass depolarized and the distance from the recording electrode (*see* Figs. 1-3 and 1-6).

The graphic representation of the heart's electrical activity recorded through electrodes positioned at strategic points on the body constitutes the electrocardiogram (ECG). The recording of the electrical currents, their direction, and their magnitude, as well as the rate of the heart's contractions, is made by the machine and electrocardiograph, which is essentially a galvanometer whose deflections are recorded on moving, specially prepared paper.

The ECG is the recording obtained, and to simplify interpretation, it suffices to state that the ECG displays the following:

- Three major deflections or waves: the P wave, the QRS complex, and a T wave (Fig. 1-7).
- Two time intervals of clinical importance: the PR interval and QRS duration (*see* Fig. 1-7).

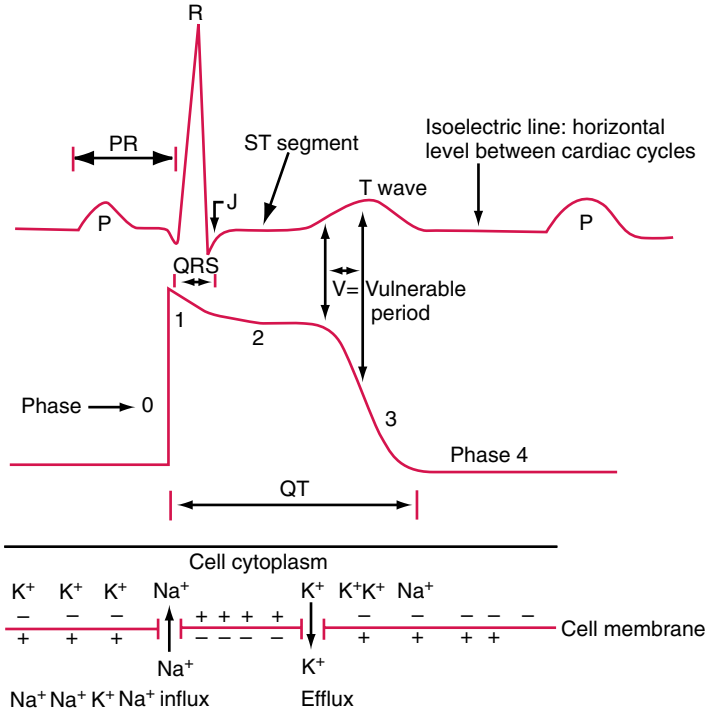

Fig. 1-7. Sodium influx, potassium efflux, the action potential, and the electrocardiogram. (From Khan, M. Gabriel: *On Call Cardiology*, 3rd ed., Philadelphia, 2006, WB Saunders, Elsevier Science.)

- The ST segment, a most important ECG component. The study of abnormalities of the ST segment reveals the early diagnosis of acute myocardial infarction (MI) and myocardial ischemia. Thus, this text devotes an in-depth chapter to abnormalities of the ST segment and does so early in the interpretive sequence; that is, before analysis of abnormalities of the P wave, ventricular hypertrophy, QRS abnormalities, and the electrical axis, all of which are discussed early in other textbooks. This approach simplifies ECG interpretation and is a strategy that is now embraced by physicians who render acute care to patients with acute MI and those with myocardial ischemia.

HOW ARE THE WAVES OF THE ELECTROCARDIOGRAM PRODUCED?

P Wave

The early part of the P wave represents the electrical activity generated by the right atrium; the middle portion of the P wave represents

completion of right atrial activation and initiation of left atrial activation; and the late portion is generated by the left atrium. The P wave is the first deflection recorded and is a small, smooth, rounded deflection that precedes the spiky-looking QRS complex (Fig. 1-8). (See Chapter 3 for an in-depth discussion of P waves.)

PR Interval

The PR interval involves the time required for the electrical impulse to advance from the atria through the AV node, bundle of His, bundle branches, and Purkinje fibers until the ventricular muscle begins to depolarize (*see* Figs. 1-7 and 1-8).

QRS Complex

The QRS complex represents the spread of electrical activation through the ventricular myocardium; the resultant electrical forces generated from ventricular depolarization is recorded on the ECG as a spiky deflection (*see* Figs. 1-7 and 1-8). The sharp, pointed deflections are labeled QRS regardless of whether they are positive (upward) or negative (downward).

Figure 1-9 indicates the conventional labeling of the QRS complex: q or Q, r or R, s or S, depending on the size of the components that

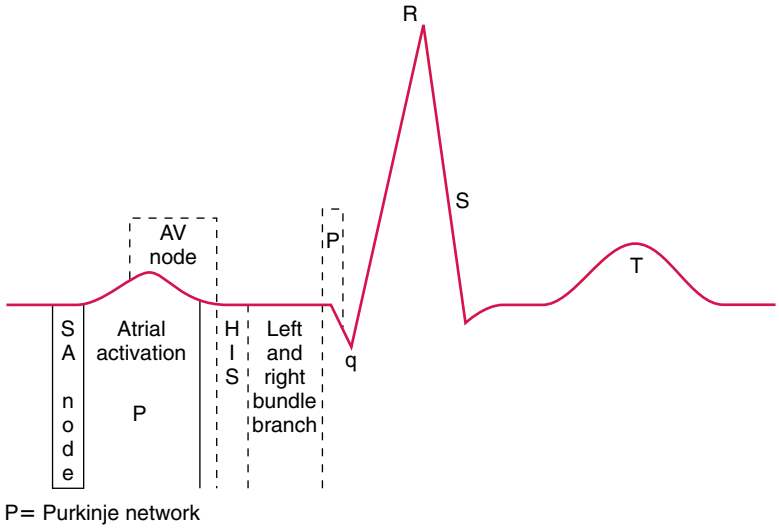

Fig. 1-8. Relationship of P wave, PR interval, and QRS complex to activation from the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His (HIS), and bundle branches. Note that the normal ST segment curves imperceptibly into the ascending limb of the T wave and is not a horizontal line.

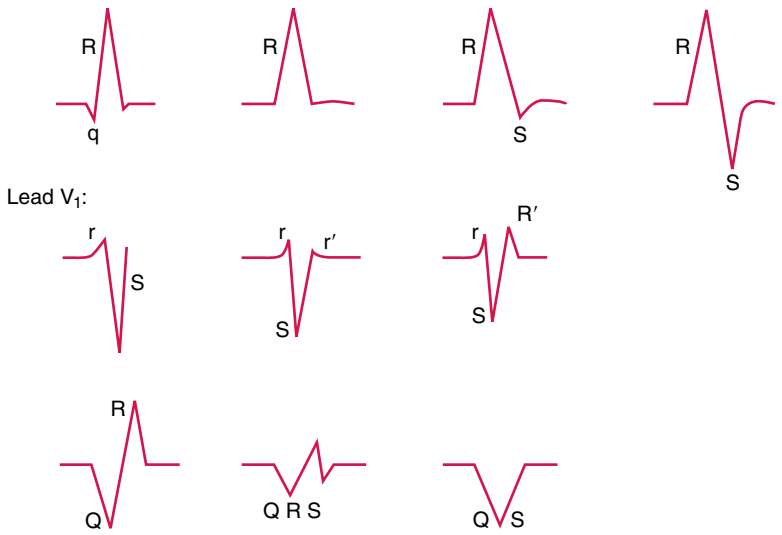

Fig. 1-9. Variation in QRS wave form. Uppercase letters are used to denote large deflection; R or r is used for first positive deflection; and R′ or r′ for second positive wave. Q or q is used for negative deflection before an r or R wave.

may be recorded (i.e., those influenced by the electrode position) and the direction of the resultant vector forces. Large deflections are labeled with uppercase letters.

The genesis of the QRS complex is intricate and is better understood after the reader has been presented with information on leads and lead positions and why 12 leads are used to capture 12 views of the heart's electrical activity. Thus the genesis of the QRS complex is discussed at the end of this chapter.

ST Segment

The ST segment is the segment that lies between the end of the QRS complex and the beginning of the T wave (*see* Figs. 1-7 and 1-8). It represents the period when all parts of the ventricles are in the depolarized state or a stage in which the terminal depolarization and the starting repolarization are superimposed and thus neutralize each other. Early repolarization may encroach on the ST segment to a variable degree. The part at which the ST segment takes off from the QRS complex is called the J, or the junction point. The ST segment normally curves imperceptibly into the ascending limb of the T wave and should not form a horizontal line nor form a sharp angle with the proximal

limb of the T wave. The student must be aware of this important diagnostic point.

This important diagnostic ECG segment is discussed in detail in Chapters 2 and 5.

T Wave

The T wave represents electrical recovery, repolarization of the ventricles, and is a broad, rounded wave (*see* Figs. 1-7 and 1-8). The T wave follows each QRS complex and is separated from the QRS by an interval that is constant for that ECG. Because ventricular recovery proceeds in the general direction of ventricular excitation, the polarity of the resultant T vector is similar to that of the QRS vector. The T wave is recorded during ventricular systole, whereas the QRS occurs immediately before mechanical systole.

- The T wave process is energy consuming, but the QRS process is not. During repolarization, cellular metabolic work and energy consumption occurs to accomplish the ionic flux associated with repolarization. Thus several metabolic, hemodynamic, and physiologic factors may affect the repolarization process and alter the morphology of the T wave. The student or clinician interpreting ECGs should be aware of the normal variations in T wave morphology and the influence of a host of factors that may alter the T wave and lead to erroneous diagnoses.
- Levine listed approximately 67 causes for T wave changes, which include the patient drinking ice water, eating, exercising, or fasting or having infections, fever, tachycardia, anoxia, shock, electrolyte derangements, acidemia, alkalemia, hormonal imbalances, subarachnoid hemorrhage, or drug or alcohol abuse.

Because of the unreliable diagnostic yield derived from the scrutiny of T waves, further details on this topic are relegated to Chapter 8.

U Wave

The U wave is a wave that follows the T wave and is observed only in the ECG tracings of some individuals. It is a small, often indistinct wave, and its source is uncertain (*see* Chapter 8).

WHY USE 12 LEADS TO RECORD THE ELECTROCARDIOGRAM?

Einthoven's discovery in 1901 was of paramount importance. His landmark paper was published in 1901, and a further paper on the galvanometric registration of the human electrocardiogram was published

in 1903. However, the initial work of Galvani (1791), Muller (1856), and Waller (1887) initiated Einthoven's accomplishment. Einthoven recognized that the heart possessed electrical activity, and he recorded this activity using two sensors attached to the two forearms and connected to a silver wire that ran between two poles of a large permanent magnet. He noted that the silver wire moved rhythmically with the heartbeats, but to visualize the small movements Einthoven shone a light beam across the wire, and the wavy movements of the wire were recorded on moving photographic paper. Einthoven recorded the waves and spiky deflection and labeled the first smooth, rounded wave, P; the spiky deflection, QRS; and the last recorded wave, T.

- Einthoven labeled the waves P, Q, R, S, and T; his lettering obeyed the convention used by geometricians: curved lines were labeled beginning with P, and points on straight lines were labeled beginning with Q.

Einthoven, Sir Thomas Lewis, and others correlated the ECG waves with the contracting heart and correlated that the P wave was related to atrial contraction and that the QRS deflection was associated with ventricular contraction. Improvements in the quality of recordings resulted from the immense work and technique of Frank Wilson, who studied with Lewis and, in Michigan (1934), described the unipolar leads that include the precordial V leads and VR, VL, and VF.

LEADS AND ELECTRODES

Why Are 12 Leads Necessary?

Figure 1-10 shows the infinite number of electrode positions arranged in a continuous circle, at the center of which is the origin of the depolarization wave. The illustration indicates that the electrode position has a profound influence on the size, or amplitude, of the recording.

- Twelve ECG leads are used to obtain 12 views of the heart's electrical activity. The heart may be considered to lie at the center of an equilateral triangle (Fig. 1-11). The leads attached to the limbs, the limb leads, act as linear conductors and have virtually identical voltages at all points along their lengths. The limbs can be regarded as extensions of a lead wire. Thus, the left arm electrode placed at the wrist, arm, or shoulder displays the same ECG record. Because the limb leads act as linear conductors, the effective sensing points and electrode locations are at the left and right shoulders and left groin, but are usually positioned and labeled as follows:

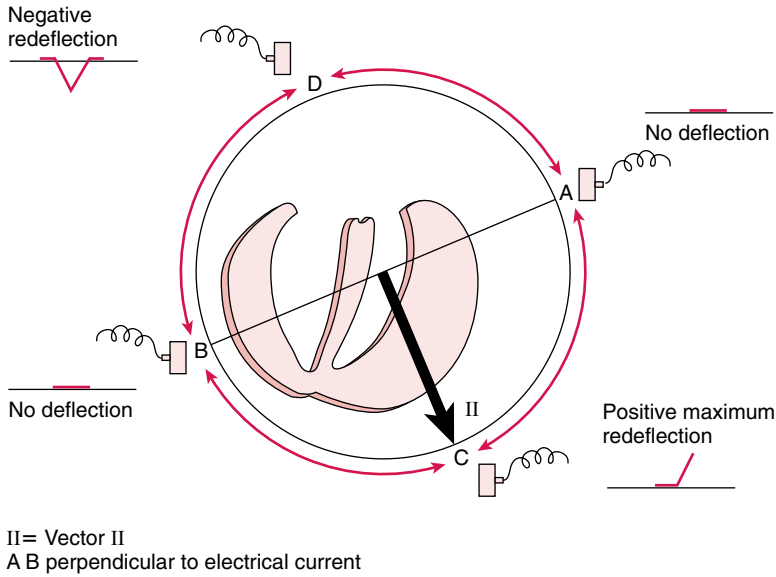

Fig. 1-10. Effect of varied electrode positions on the amplitude and direction of deflections recorded: Leads between C and A or C and B give positive deflection less than at C. Leads at D and A or D and B record negative deflection of varying size. The line AB is perpendicular to the electrical current.

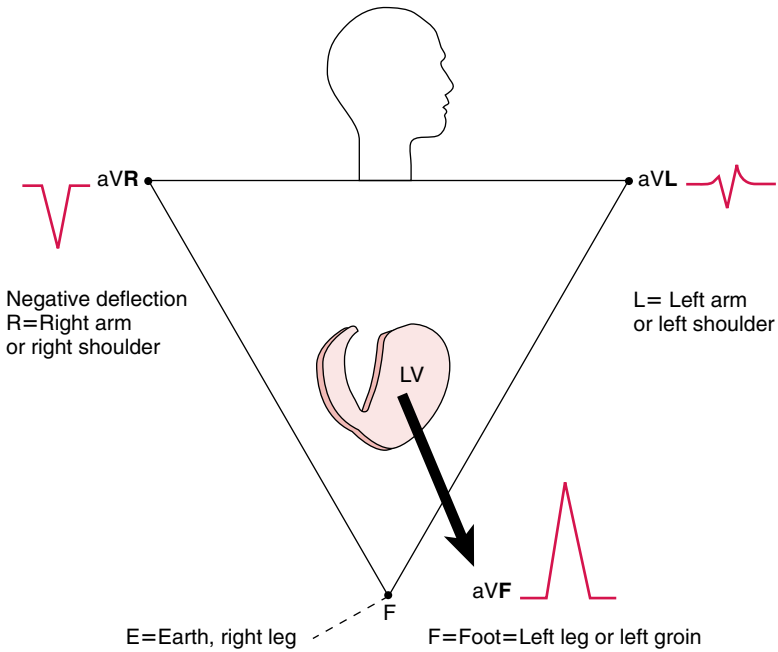

Fig. 1-11. The heart depicted as a three-muscle mass that lies in the center of an equilateral triangle. The two shoulders and left groin are sensing positions.

- R = right arm lead
- L = left arm lead
- F = foot = left leg lead

These leads lie along the frontal plane of the body and display action potential only in the frontal plane. (See discussion of frontal plane axis in Step 9 in Chapter 2 and in Chapter 9.)

Two important concepts must be reemphasized:

- If the excitatory depolarization head of the current (vector force) flows toward a unipolar electrode, a galvanometer will record an upward or positive deflection (*see* Fig. 1-3).
- When an excitatory depolarization process is directed away from the electrode, a downward or negative deflection is recorded (*see* Fig. 1-3).

Figure 1-11 displays deflections that can be recorded by limb leads R, L, and F. The main electrical current of activation flows toward the F (left leg) electrode and records an upward or positive deflection of large amplitude. The current flows away from the right shoulder (R) electrode and records a downward deflection. The right shoulder lead (R) looks into the interior of the heart toward the endocardium, and as mentioned previously, the current of activation flows from the endocardium and traverses the myocardium toward the pericardium and thus displays a negative deflection. The student should notice that aVR is always relatively negative and aVF is always relatively positive.

- Lead L at the left shoulder or left arm usually displays a small positive or equiphasic deflection, but the heart hangs in the chest and is subject to rotational changes, and the main current direction may be altered; thus, this lead may show a large-amplitude positive deflection in some individuals, and a negative deflection if the heart's position is vertical.

Why Augmented Leads?

- Why is a V added to the R, L, and F? These leads are termed unipolar limb leads, but voltage measurements are virtually never unipolar. The connection formed by attaching the R, L, and F electrodes together acts as a reference connection, and the lead formed is termed a V lead (V = voltage); thus the convention VR, VL, and VF, and the V is also used for the leads positioned on the chest, V_1 to V_6.
- Goldberger (1942) augmented Wilson's unipolar extremity leads that gave low-amplitude records; Goldberger's strategy increased the amplitude of the deflections by 50%. Thus, the letter a is used to denote the

augmented lead (e.g., aVL = augmented-voltage left arm lead [V = voltage]).

Standard Bipolar Limb Leads I, II, and III

Figure 1-12 shows the views of the heart obtained by leads I, II, and III.

- Lead I connects the two arms and is formed by connecting L to the positive terminal and R to the negative terminal of the galvanometer; thus, I = aVL = aVR. Lead I looks at the heart from the left, inferior to lead aVL, the lead of the left shoulder (arm), and displays the electrical tracing produced by a combination of the right arm and left arm electrodes. The right leg electrode is an earth (or ground) and minimizes interference.
- Lead II looks at the heart from a position to the left of the left groin, foot lead F (*see* Fig. 1-12).
- Lead III looks at the heart from a position to the right of the left groin, foot lead F. Thus, leads II, III, and aVF look at the inferior surface of the heart from different angles, and they usually show some similarities. Lead III is the most unreliable of the leads II, III, and aVF. Thus, many errors are made from the observation of the QRS and T wave in lead

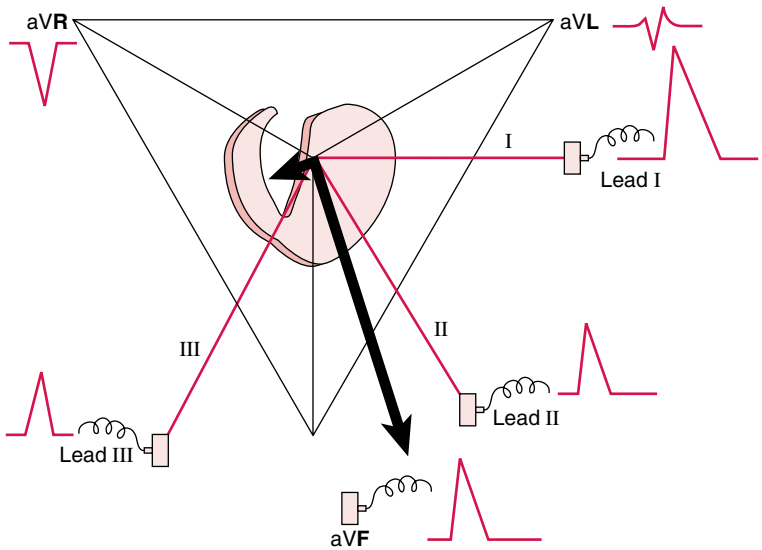

Fig. 1-12. Standard limb leads I, II, and III. Note that aVF leads II and III look at the inferior surface of the heart and deflections show minor variation. Leads I and aVL look at the anterolateral aspect of the heart.

III. Normal yet pathologic-appearing Q waves and T wave inversion may be observed frequently in lead III as a normal variant (*see* Chapters 6 and 8).

- The six leads display six photographs of the heart's electrical activity taken from six angles (one every 30 degrees). The six leads can be visualized as traversing a flat plane over the chest of the patient (i.e., the frontal plane). Importantly, if only two of the six leads are recorded, the most informative pair are I and aVF.

Vertical Versus Horizontal Heart Position

Figure 1-13 shows the changes in QRS waveform caused by alteration of the position of the heart:

- Both aVR and aVL face the ventricular cavity and show a QS complex.
- A qR complex in lead aVL indicates a horizontal heart position, and the QRS morphology in aVL resembles that in V_5.
- A qR complex in aVF and a QS complex in aVL indicate a vertical heart position, and the QRS morphologies in leads aVF and V_5 resemble each other.
- The position of the heart varies between horizontal and vertical.

Chest Leads/Precordial or V Leads

The six chest leads give six more views of the heart's electrical activity and vector forces; they are positioned around the anterior and left chest wall in a horizontal plane. Figures 1-14 and 1-15 indicate the position of the precordial chest leads that overlie the right and left ventricles.

V_1 and V_2 face and lie close to the wall of the right ventricle. V_2 and V_3 lie near the intraventricular septum. V_4 and V_3 look at the anterior parts of the left ventricle, with V_4 close to the apex. V_5 and V_6 (leads I and aVL) view the anterolateral region of the left ventricle and often appear similar to each other. The recording in lead aVL, however, varies depending on a horizontal or vertical heart position. If V_7 is taken, it is positioned in the posterior axillary line.

The precordial electrodes V_1 to V_6 are so close to the electrical currents of the heart that no augmentation is necessary. Lead V_6 is far around (in the axilla) and is separated from the free wall of the left ventricle by a significant distance. Figure 1-15 indicates the approximate relationship of the ventricular myocardium and the precordial chest leads V_1 to V_6.

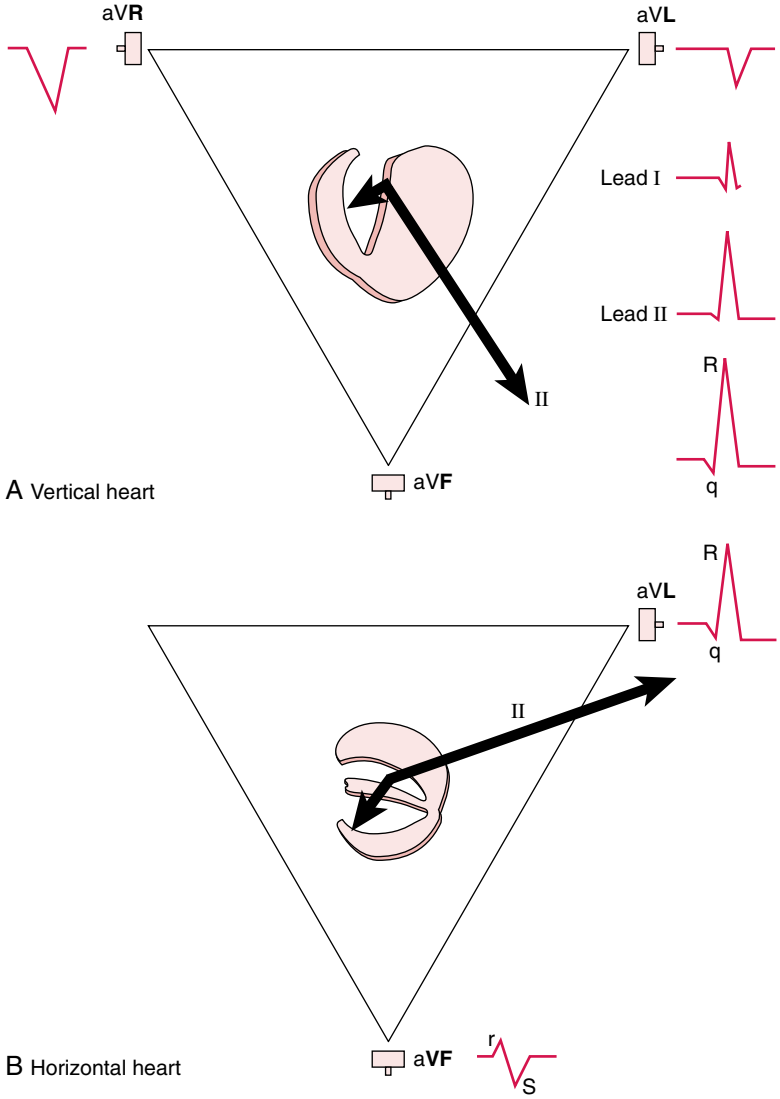

Fig. 1-13. Changes in deflections with the heart in a vertical (**A**) and a horizontal (**B**) position. In the vertical position (**A**), both the aVR and aVL face the cavity of the ventricles and record a QS complex. A QRS complex in aVF indicates a heart that is positioned close to vertical; qRS in aVL indicates a horizontal heart position (**B**).

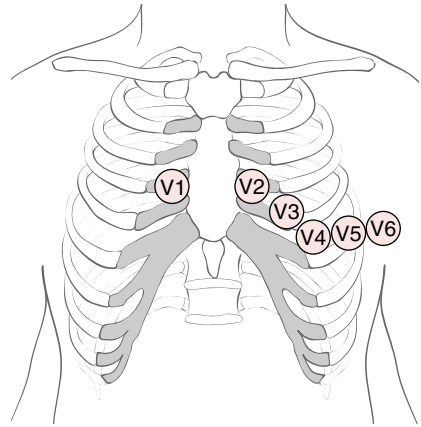

Fig. 1-14. Position of precordial chest leads.

Figure 1-16 reemphasizes that the position of leads aVL and aVF and other limb leads are in the same frontal plane. The chest leads V_1 to V_6 encircle the left thorax in a horizontal plane (*see* Fig. 1-15).

Caution: The entire chest, with the heart within it, acts as a volume conductor, and thus voltage varies appreciably at locations only a centimeter apart. Therefore, the leads placed on the chest wall V_1 to V_6

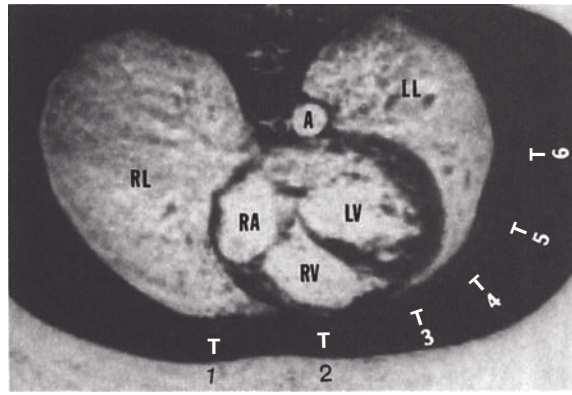

Fig. 1-15. Magnetic resonance image of heart to illustrate approximate relationship of chest electrodes to cardiac chambers. Points 1 to 6 represent sites of the six precordial electrodes V_1 to V_6. RA, right atrium; RV, right ventricle; LV, left ventricle; RL, right lung; LL, left lung; A, aorta. (From Marriott HJL: *Practical Electrocardiography*, 8th ed., Philadelphia, 1988, Williams & Wilkins.)