UNDERSTANDING ELECTROCARDIOGRAPHY

Arrhythmias and the 12-lead ECG

MARY BOUDREAU CONOVER,

FOURTH EDITION

UNDERSTANDING ELECTROCARDIOGRAPHY

Arrhythmias and the 12-lead ECG

MARY BOUDREAU CONOVER, R.N., B.S.

Instructor and Education Consultant,
California Hospital Medical Center,
Los Angeles, California; Instructor of Intermediate and
Advanced Arrhythmia Workshops,
West Hills Hospital, Canoga Park, California;
Education Consultant, Holy Cross Hospital Cardiac
Arrhythmia Center, Mission Hills, California

FOURTH EDITION

with 814 illustrations

The C. V. Mosby Company

ST. LOUIS • TORONTO • PRINCETON 1984

MOSBY

A TRADITION OF PUBLISHING EXCELLENCE

Editors: Michael R. Riley, Barbara Norwitz-Nathan
Assistant editor: Bess Arends
Manuscript editor: Stephen C. Hetager
Cover design: Suzanne Oberholtzer
Book design: Jeanne Bush
Production: Barbara Merritt, Susan Trail

FOURTH EDITION

Printed in the United States of America

The C.V. Mosby Company
11830 Westline Industrial Drive, St. Louis, Missouri 63146

Library of Congress Cataloging in Publication Data

Conover, Mary Boudreau
Understanding electrocardiography.

Includes bibliographical references and index.
1. Electrocardiography. I. Title. [DNLM:
1. Electrocardiography. WG 140 C753u]
RC683.5.E5C65 1984 616.1'207547 83-21944
ISBN 0-8016-1123-7

C/VH/VH 9 8 7 6 5 4 3 2 1 03/D/341

CONSULTING BOARD

Henry J.L. Marriott, M.D., F.A.C.P., F.A.C.C.

Director of Clinical Research, Rogers Heart Foundation; Director of Coronary Care Center and Director of Electrocardiography Department, St. Anthony's Hospital, St. Petersburg, Florida; Clinical Professor of Medicine (Cardiology), Emory University School of Medicine, Atlanta, Georgia; Clinical Professor of Pediatrics (Cardiology), University of Florida, Gainesville, Florida

Ara G. Tilkian, M.D., F.A.C.C.

Assistant Clinical Professor of Medicine, University of California, Los Angeles; Associate Director of Cardiology, Holy Cross Hospital and Valley Presbyterian Hospital, San Fernando Valley, California

Sarko M. Tilkian, M.D.

Director of Medical Education, Northridge Hospital; Staff Physician, Northridge Hospital Foundation, Northridge, California, and Tarzana Medical Center, Tarzana, California

John H.K. Vogel, M.D., F.A.C.P., F.A.C.C., F.C.C.P.

Director of Cardiology, Goleta Valley Community Hospital, Santa Barbara, California; Director of Santa Barbara Heart and Chest Institute, Santa Barbara, California; Clinical Professor of Medicine, University of Southern California School of Medicine, Los Angeles, California

Hein J.J. Wellens, M.D.

Professor of Medicine and Chief of Cardiology, University of Limburg, Maastricht, The Netherlands

PREFACE

Rapid, accurate arrhythmia detection and identification involve an understanding of electrocardiography solidly based on anatomy, physiology, electrophysiology, and arrhythmogenesis. This book, intended for both the beginner and the advanced student of electrocardiography, presents such information, with emphasis on mechanisms and causes, in the firm belief that the approach facilitates a correct diagnosis.

This edition has been updated in every detail and contains the most current information on the 12-lead electrocardiogram, arrhythmias, and arrhythmogenic mechanisms. For example, torsades de pointes is fully explained and illustrated, as are sinoatrial nodal reentry tachycardia and the latest findings on aberrancy versus ectopy. A completely new chapter deals with antiarrhythmic drugs, including calcium channel blockers, beta blockers, and other new antiarrhythmic drugs.

This book is designed to build up your understanding of the 12-lead ECG gradually, beginning with the important chapters on anatomy and physiology, electrophysiology and mechanisms of arrhythmias, normal electrical activation of the heart, axis determination, and the normal 12-lead ECG. These chapters give you the background to understand the abnormal conditions that can be diagnosed from the 12 diagnostic leads, such as bundle-branch block, myocardial infarction, hypertrophy and chamber enlargement, and Wolff-Parkinson-White syndrome.

The chapter on atrioventricular block places emphasis on the difference between nonconduction and pathological block and explains the pathophysiological basis for the differential diagnosis between types I and II AV block.

With each new edition of this book, Wolff-Parkinson-White syndrome assumes more and more importance clinically as we learn more about the degrees of preexcitation, concealed WPW syndrome, and the incidence and types of arrhythmias associated with preexcitation. The chapter on this important subject has been updated and expanded.

The chapter on the electrical axis retains its two clinically useful methods of axis determination—the easy two-step method and the more precise hexaxial figure.

A chapter on electrograms has been added, in view of the fact that more and more patients are returning from surgery with atrial and ventricular wires in place. For the same reason the subject of accidental electrocution of the electrically sensitive patient has taken on greater significance; the chapter on this important topic has been revised by its original author, Edward L. Conover.

Mary Boudreau Conover

CONTENTS

1 **Anatomy and physiology of the heart,** 1

Heart chambers, valves, and vessels, 2
The cardiac cycle, 2
Papillary muscles and the chordae tendineae, 4
Surfaces of the heart, 4
Conductive system of the heart, 6
Properties of cardiac muscle, 9
Sinus node as pacemaker of the heart, 11
Autonomic nervous system and control of the heart, 11
Coronary circulation, 12
Summary, 14

2 **Electrophysiology of the heart,** 15

Current flow in the heart: its cable properties, 16
Microscopic structure of cardiac cells, 16
Resting membrane potential, 19
Maintenance of the resting membrane potential, 19
Depolarization, 20
Threshold potential, 20
Repolarization, 20
Recording the cardiac action potential, 20
The fast sodium channels and their gates, 21
Fast-response action potential, 22
Slow calcium-sodium channels, 23
The action potential and conduction velocity, 24
Refractory period, 26
Supernormal period, 27
Automaticity, 27
Action potentials of different types of pacemaker cells, 29
Membrane responsiveness, 29
Summary, 29

3 **Arrhythmogenesis,** 31

Circus reentry, 33
Focal reentry, 36
Altered automaticity, 36
Afterdepolarization and triggered activity, 38
Summary, 39

4 **Determination of the electrical axis,** 41

Vectors, 42
Instant-to-instant cardiac vectors, 42
Axis of the heart, 42
Current flow related to axis of a lead, 42
The three bipolar limb leads—Einthoven's triangle, 45
Unipolar limb leads, 47
An easy two-step method of axis determination, 48
The hexaxial figure: a more precise method of axis determination, 52
Extreme axis deviation, 55

5 **The 12-lead electrocardiogram,** 57

Frontal plane leads compared to horizontal plane leads, 58
Bipolar leads compared to unipolar leads, 58
The precordial leads, 59
Sequence of ventricular activation, 60
Ventricular complex as seen in the limb leads, 60
Ventricular complex as seen in the precordial leads, 62
Additional helpful leads, 64
The normal electrocardiogram, 65
Artifacts, 70

6 **Measurement of rate and intervals,** 73

ECG paper, 74
Calculation of heart rate, 74
The PR interval, 74
The QRS complex, 76
The ST segment, 78
The QT interval, 78

7 **Arrhythmias originating in the sinus node,** 81

Normal atrial depolarization, 82
Sinus tachycardia, 82
Sinus tachycardia in the setting of myocardial infarction, 84
Sinus bradycardia, 84
Sinus arrhythmia, 85
Sinoatrial block, 85
Sinus arrest, 86

Sick sinus syndrome, 89
Paroxysmal supraventricular tachycardia resulting from SA nodal reentry, 89
Summary, 91

8 **Supraventricular ectopics,** 93

Atrial ectopic mechanisms, 95
Junctional ectopic mechanisms, 113
AV nodal reentry, 121
Overdrive suppression, 126
Summary, 128

9 **Ventricular ectopics,** 131

Mechanisms—a quick review, 133
Premature ventricular complexes, 133
Ventricular tachycardia, 150
Bidirectional ventricular tachycardia, 150
Ventricular fibrillation, 152
Torsades de pointes, 154
Accelerated idioventricular rhythm, 159
Ventricular escape, 163

10 **Atrioventricular block,** 167

Pathology, 168
First-degree AV block, 169
Second-degree AV block, 170
Complete (third-degree) AV block, 179
Complete AV block in atrial fibrillation, 181

11 **Atrioventricular dissociation,** 183

Sinus bradycardia and AV dissociation, 184
Accelerated idiojunctional rhythm with AV dissociation, 185
Accelerated idioventricular rhythm with AV dissociation, 187
AV block and AV dissociation, 190

12 **Laddergrams,** 193

How to begin, 195
Ventricular ectopics, 196
Premature atrial complexes, 196
Retrograde conduction, 196
Junctional rhythms, 196
Conduction blocks and escape beats, 198
Fusion, 199
Wenckebach periods, 199
Reciprocal beats and rhythms, 200

13 **Bundle-branch block,** 203

Normal ventricular activation time (intrinsicoid deflection), 204
T-wave changes in BBB, 204
Right bundle-branch block, 206
Left bundle-branch block, 210
Rate-dependent (critical-rate) BBB, 214
BBB complicating acute anteroseptal infarction, 214
Hemiblock, 216
Monitoring for axis shifts and for RBBB, 223
Trifascicular block, 223

14 **Aberrant ventricular conduction versus ventricular ectopy,** 227

RBBB aberration versus left-ventricular ectopy, 228
LBBB aberration versus right-ventricular ectopy, 230
Summary of Wellens' findings, 231
Situations in which aberrancy may occur, 234
Other clues, 238
Summary, 240

15 **Accessory pathways,** 241

Embryology, 242
Classification of accessory pathways, 243
Accessory AV connection: Wolff-Parkinson-White syndrome, 244
AV nodal fast pathway: short PR–normal QRS syndrome, 258
Nodoventricular or fasciculoventricular tracts: Mahaim fibers, 260

16 **Fusion complexes,** 265

Mechanisms, 266
ECG changes, 266
Ventricular fusion, 266
Atrial fusion, 270
Summary, 270

17 **Parasystole,** 271

ECG criteria for classical parasystole, 272
Entrance block with exit conduction, 274

18 **Myocardial infarction,** 277

Terminology: ischemia and anoxia, 278
Pathophysiology of the evolving myocardial infarction, 278
ECG signs of myocardial infarction, 280
Transmural versus subendocardial injury patterns, 288
Reciprocal changes, 289
Locating the infarct, 291

Inferior-wall myocardial infarction, 292
Inferolateral myocardial infarction, 294
Anterior-wall myocardial infarction, 295
Lateral-wall myocardial infarction, 299
Posterior-wall myocardial infarction, 299
Right-ventricular myocardial infarction, 301
Stages of recovery, 301

19 **Chamber enlargement,** 303

Clinical implications of myocardial hypertrophy, 304
Left-ventricular hypertrophy, 304
Right-ventricular hypertrophy, 309
Left-atrial enlargement, 311
Right-atrial enlargement, 312

20 **Electrograms,** 313

Protecting the patient from electrocution, 314
Unipolar AEG compared to bipolar AEG, 314
Multichannel recordings, 316
Single-channel recordings, 318
Troubleshooting, 319
The His-bundle electrogram, 321

21 **Antiarrhythmic drugs,** 323

Arrhythmogenic mechanisms, 324
Pharmacokinetics, 324
Quinidine and quinidine-like drugs, 325
Lidocaine and lidocaine-like drugs, 331
Beta blockers, 334
Slow channel–blocking agents, 338
Other antiarrhythmics, 341

22 **The effects of potassium and calcium levels on the electrocardiogram,** 349

Potassium, 350
Calcium, 354

23 **Accidental electrocution,** 355

Edward L. Conover

The electrically sensitive patient, 356
Electrical current flow, 356
Ohm's law, 357
Typical conductors found in a CCU or an ICU, 357
Electrical shock, 358

Sources of currents, 359
Typical hazardous situations, 362
What can be done to avoid accidental electrocution, 364

24 Pacemakers, 369

John H.K. Vogel
Anita Powe
Mary Boudreau Conover
Victor A. Morant
Michael Janssen

The pacing system, 370
Complexes produced by the pacemaker spike, 373
The sensing circuit, 373
Pacemaker refractory period, 374
Hysteresis, 374
Methods of insertion, 375
Complications, 376
Troubleshooting DDD electrocardiograms, 381

Glossary, 395

Appendix A Beta blockers—a comparison, 400
B Calcium antagonists—a comparison, 402

1

ANATOMY AND PHYSIOLOGY OF THE HEART

- ☐ **Heart's valves, chambers, and cycle**
- ☐ **Papillary muscles and the chordae tendineae**
- ☐ **Surfaces of the heart**
- ☐ **Conductive system**
- ☐ **Atrioventricular junction**
- ☐ **Properties of cardiac muscle**
- ☐ **Sinus node**
- ☐ **Autonomic nervous system**
- ☐ **Coronary circulation**

HEART CHAMBERS, VALVES, AND VESSELS

Fig. 1-1 shows the heart's chambers, vessels, and valves, and the flow of blood through them. The four chambers of the heart function as a double pump; the two ventricles do the actual pumping, with the two atria serving as their primers. The right ventricle pumps venous blood to the lungs for oxygenation, while the left ventricle pumps oxygenated blood into the systemic circulation.

Any pump needs valves so that the fluid will flow in the right direction. There are four valves in the heart: the *mitral valve,* separating the left atrium from the left ventricle; the *tricuspid valve,* separating the right atrium from the right ventricle; the *aortic valve,* separating the left ventricle from the aorta; and the *pulmonic valve,* separating the right ventricle from the pulmonary artery. These valves are diagramatically illustrated in Fig. 1-1. The two "inflow tract" valves (mitral and tricuspid) are sometimes referred to as the atrioventricular (AV) valves. The two "outflow tract" valves (pulmonic and aortic) are sometimes referred to as semilunar valves.

The heart valves are mechanically very simple—they are cusps that open with pressure on the convex side (downstream) and close with pressure on the concave side (upstream). The mitral valve is the only valve with two cusps (like a bishop's miter); all the rest have three.

THE CARDIAC CYCLE

Diastole

During diastole, when the whole heart is relaxed, blood enters the atria through unguarded (no valves) conduits—the *superior and inferior vena cavae* on the right and the four *pulmonary veins* on the left (Fig. 1-2, *A*). The valves from the atria into the ventricles are open (the two AV valves, mitral on the right and tricuspid on the left); blood fills the atria and passively dumps into the two ventricles. The amount of blood accumulated in the two ventricles depends, of course, on the length of diastole. If diastole is short, as with a fast heart rate, there will be less blood to pump to the lungs and the systemic circulation; this is also true if the ventricles contract without the benefit of a preceding atrial systole, as with a premature ventricular beat.

Atrial systole

Atrial systole (contraction) occurs in response to an impulse from the sinus node (the P wave of the electrocardiogram). It is the first event in the cardiac cycle. Note in Fig. 1-2, *B*, that when this occurs the atrial blood is pushed into the already full ventricles. This extra contribution to the end-diastolic pressure in the ventricles is often picturesquely called the "atrial

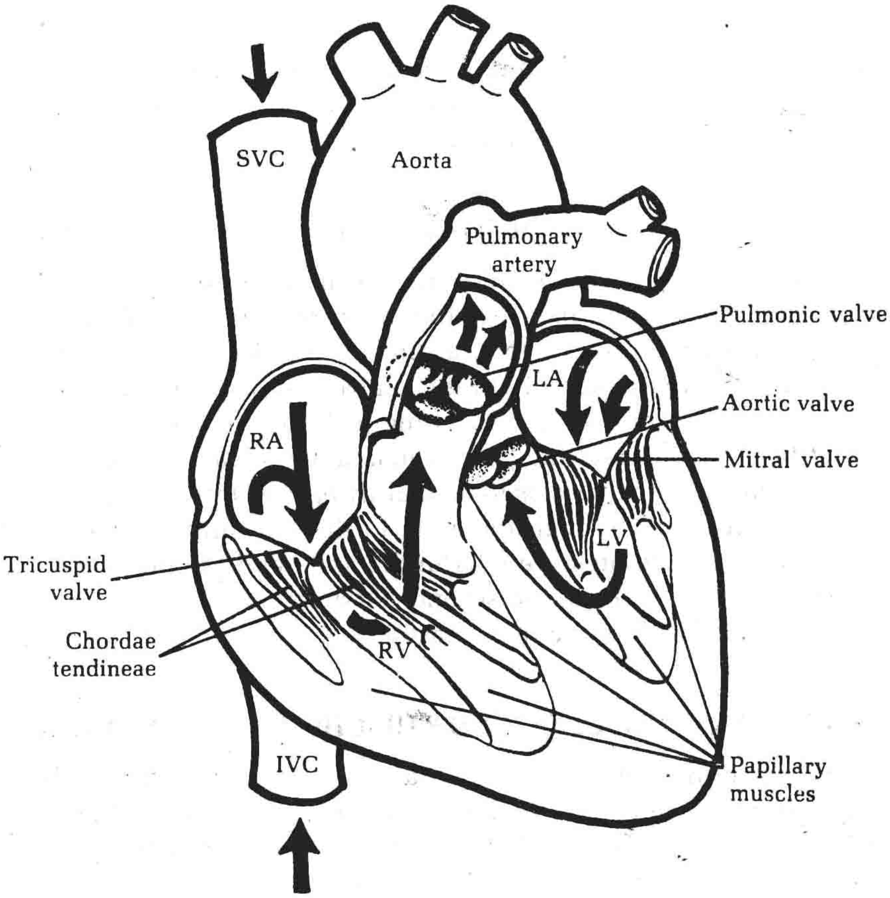

Fig. 1-1. Course of the blood through the chambers, valves, and vessels of the heart. *SVC*, Superior vena cava; *IVC*, inferior vena cava. The papillary muscles and the chordae tendineae are also shown. (From Tilkian, A.G., and Conover, M.H.: Understanding heart sounds and murmurs, ed. 2, Philadelphia, 1984, W.B. Saunders Co.)

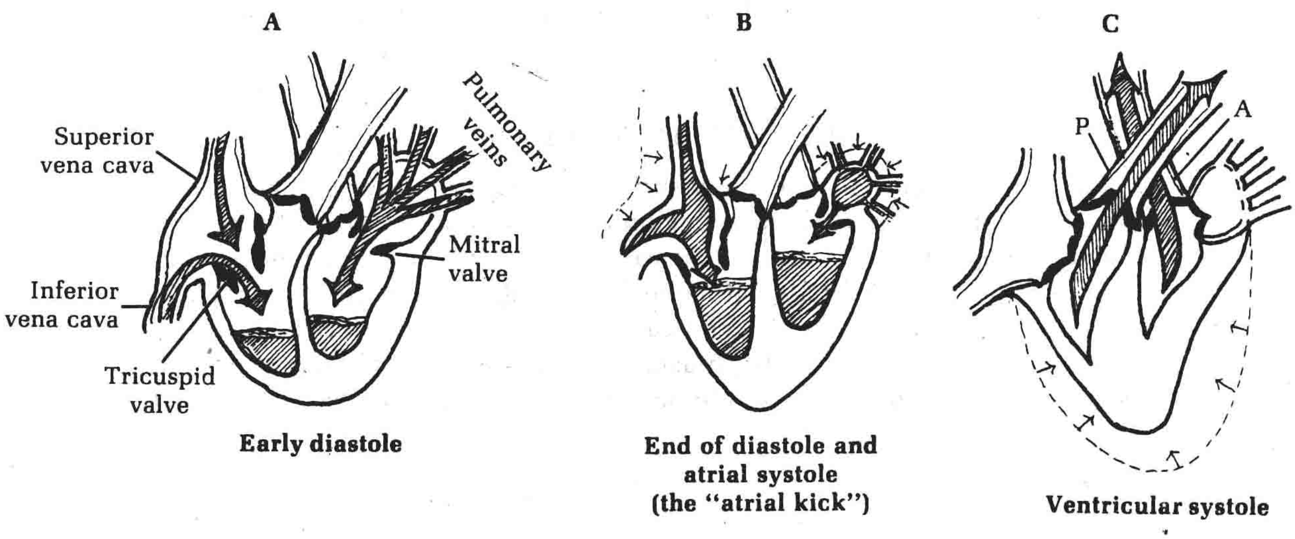

Fig. 1-2

kick." It acts as a primer for the ventricular pump according to *Starling's law*, which says that the more a muscle is stretched, the stronger it contracts—until it is stretched too much, and then if fails. The cardiac impulse is normally delayed in the AV node (PR interval) to allow for the important contribution of the atria to the ventricular blood volume.

Ventricular systole

Ventricular systole occurs in response to the impulses traveling through the conductive system and invading the myocardial tissue (QRS of the electrocardiogram). In Fig. 1-2, *C*, look at both ventricles at once and imagine what happens when pressure within the ventricular chambers suddenly rises. When the ventricles contract, the resulting pressure causes the AV valves to close (first heart sound) and the pulmonic and aortic valves to open, permitting the contents of the ventricles to be rapidly ejected through the pulmonary artery to the lungs and through the aorta to the systemic circulation. When the ventricles relax, the pressure falls in the aorta and the pulmonary artery, causing their valves to close (the second heart sound), and the cycle begins again with ventricular filling during diastole.

PAPILLARY MUSCLES AND THE CHORDAE TENDINEAE

The AV valves (mitral and tricuspid) are prevented from bulging back into the atria during ventricular systole by slender, strong cords (chordae tendineae). These cords extend from the borders of the valves to fingerlike projections on the ventricular walls (papillary muscles). When the ventricles contract, so do the papillary muscles, causing the chordae tendineae to restrain the valve leaflets from inverting into the atria.

Fig. 1-3 illustrates the chordae tendineae and the papillary muscles. Note that the left ventricle has two papillary muscles with chordae tendineae extending to the two cusps of the mitral valve. In the right ventricle there are three papillary muscles with chordae tendineae extending to the three cusps of the tricuspid valve.

SURFACES OF THE HEART

Fig. 1-4 illustrates all four heart surfaces—anterior, posterior, lateral, and inferior (diaphragmatic). The drawing on the left in Fig. 1-4 presents the simplified concept of heart surfaces that we will be using in the chapter on patterns in myocardial infarction (Chapter 18). That is, there are, for practical purposes, only two surfaces to the heart—superior and inferior. The inferior surface is composed of the diaphragmatic wall and the posterior wall, and because the left ventricle is a cylinder, the superior surface has an anterior aspect and a lateral aspect.

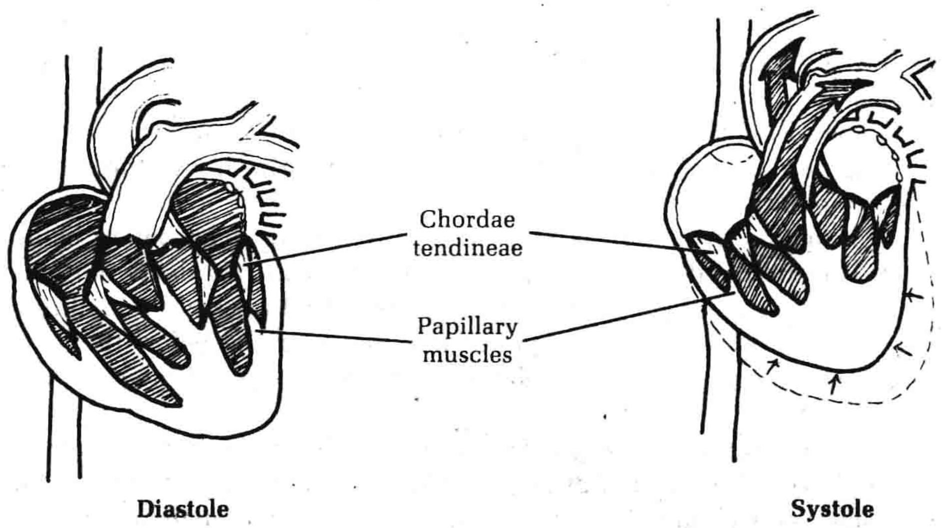

Fig. 1-3. The papillary muscles and chordae tendineae during diastole and systole.

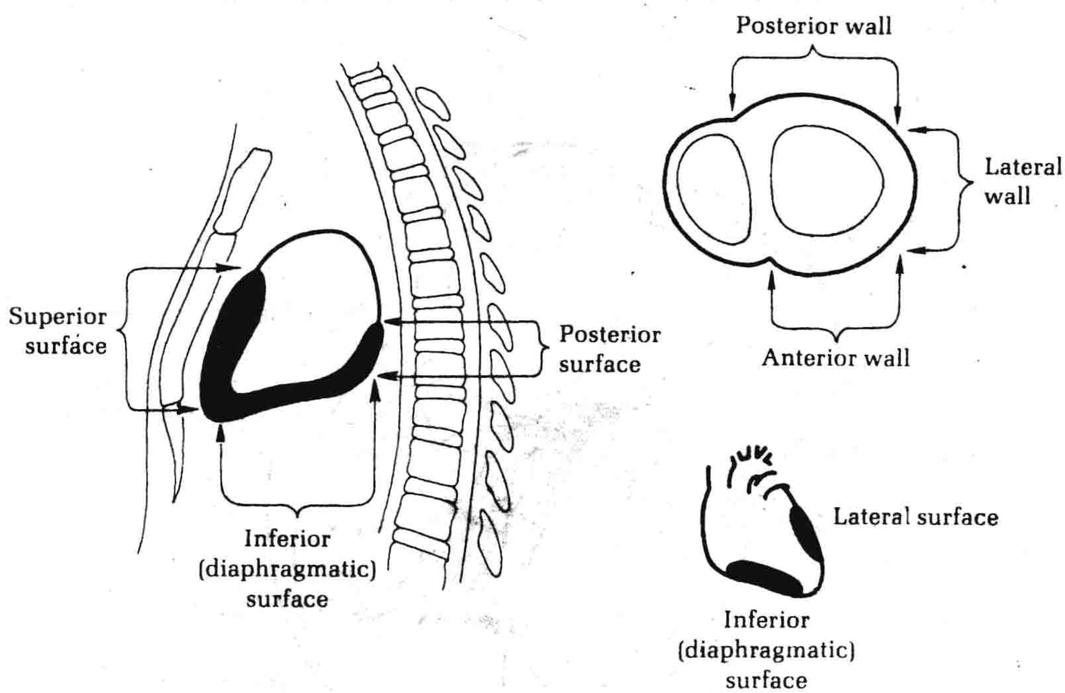

Fig. 1-4. Surfaces of the heart.

CONDUCTIVE SYSTEM OF THE HEART

Before the heart can contract, it must be stimulated to do so, the stimulus being delivered quickly and efficiently to all areas of the myocardium. These two functions—self-excitation and rapid conduction velocity—are accomplished by a specialized conductive system. Automatically and at regular intervals an electrical stimulus arises in the sinus (or sinoatrial [SA]) node, a grouping of highly specialized tissue located in the right atrium adjacent to the superior vena cava. The atria are then depolarized, and the impulse invades the AV node.

Within the AV node the electrical current is delayed before being conducted to the bundle of His. This consistently occurring delay not only protects the ventricles from inappropriately high atrial rates but also allows time for the atrial contents to be propelled into the ventricles before the latter contract. The position of the AV node in the posterior floor of the right atrium is shown in Figs. 1-5 and 1-6.

After the impulse emerges from the AV node, its rapid propagation is resumed by way of the bundle of His. Rapid spread of the current within the ventricles is accomplished by the bundle branches and their ramifications. The entire interventricular conductive system is called the His-Purkinje system.

The bundle of His divides into two main branches, right and left. The right bundle branch (RBB) has terminal ramifications in the lower interventricular (IV) septum and the anterior wall of the right ventricle. This is dramatically illustrated in Figs. 1-6 and 1-7.

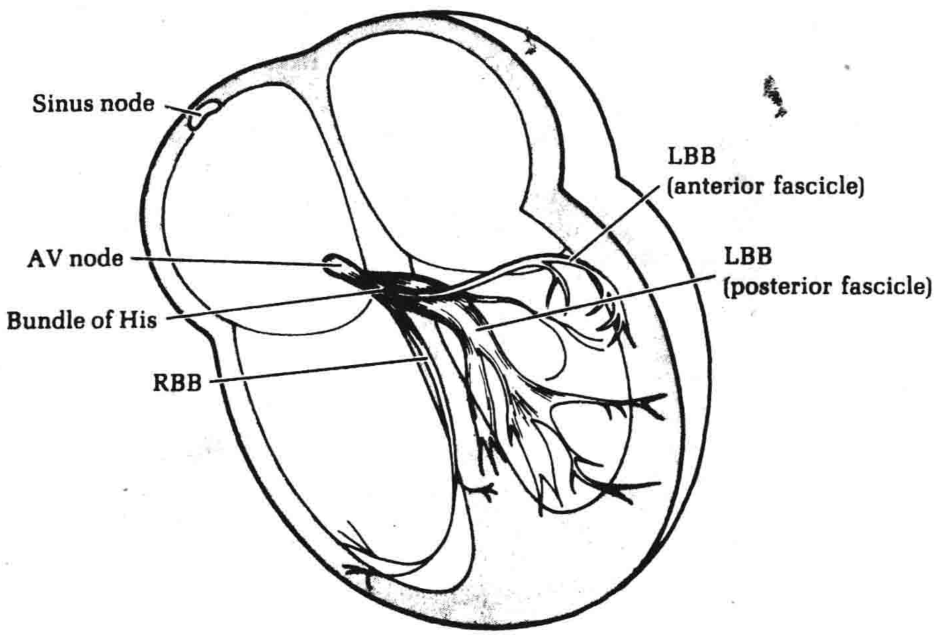

Fig. 1-5. Conductive system of the heart.