SECOND EDITION

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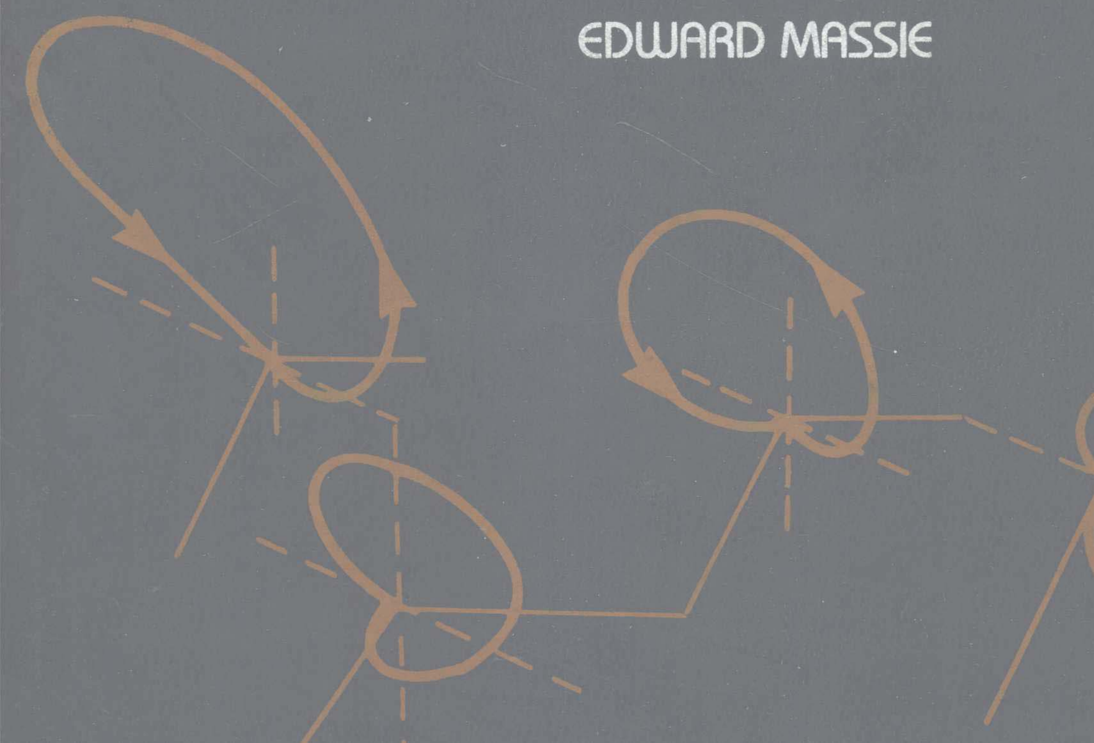

CLINICAL VECTORCARDIOGRAPHY AND ELECTROCARDIOGRAPHY

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It is with considerable pleasure, esteem and love that we dedicate this book to our wives and children.

To my wife, NANCY, *and* JON, STUART *and* ANNE
To my wife, MAUREEN, *and* JONATHAN *and* MARILYN
To my wife, FELICE, *and* HENRY *and* BARRY

Preface to the Second Edition

CLINICAL VECTORCARDIOGRAPHY AND ELECTROCARDIOGRAPHY was first published in 1960. Since that time electrovectorcardiography has changed considerably. For example, the orthogonal lead system of Frank is now more widely used than the Grishman cube system; the new knowledge of the conduction system has altered concepts of axis deviation, bundle branch block and heart block; the correlation of hemodynamic data with vectorcardiographic data has provided a noninvasive method of assessing hemodynamic change; the analysis of preoperative and postoperative vectorcardiograms has altered some of our concepts of ventricular hypertrophy; and new knowledge of ischemic heart disease has been obtained from the correlation of coronary arteriographic studies and coronary bypass surgery, which has extended our understanding of myocardial injury and infarction. These changes in cardiology and vectorcardiography certainly justify a new textbook of vectorcardiography.

This book reflects the latest information in electrovectorcardiography as it applies to arteriosclerotic disease, rheumatic heart disease, congenital heart disease and pulmonary heart disease, and it provides the reader with an up-to-date bibliography of pertinent contributions in the field. We have stressed the underlying physiologic mechanism(s) of these abnormalities as a means of demonstrating that the interpretation of the electrovectorcardiogram need not be an empirical, isolated exercise. We hope that this textbook will give the clinician a more complete understanding of heart disease and ultimately help to provide better care for cardiac patients.

In preparing this manuscript, we sought to construct a book that could serve as a reference for the experts in the field as well as a textbook for the novice. We hope to have accomplished this by providing an initial section on basic electrophysiology, followed by a discussion of the normal vectorcardiogram, and then by a presentation of pathologic data. We have also included a section on cardiac arrhythmias so the book can serve as a complete electrocardiology reference. In order to provide complete information regarding source materials, we have included a bibliography with each chapter.

We wish to extend our thanks to Dr. David Goldring, Dr. Antonio Hernandez and Dr. Alex Hartman, Jr., of the St. Louis Children's Hospital, who allowed us to review their clinical, electrocardiographic and hemodynamic data on children with congenital heart disease. Over fifty illustrations of electrocardiograms and vectorcardiograms were kindly provided from their extensive files for inclusion in Chapter 12, "Congenital Heart Disease." Our colleagues, Dr. Om Bahl and Dr. Howard Bomze, reviewed sections of the manuscript and made many worthwhile suggestions. We would also like to express our sincere thanks to numerous clinicians and investigators who have allowed us to use illustrations from their published articles and books.

Mrs. Bev Gestring has been instrumental in the preparation of this book since its inception in late 1971 with typing, mounting figures, checking references and so forth. We are also grateful to Miss Emili Sharon Stout from the University of Kansas Medical Center for reviewing manuscripts, checking references and correcting copy. Their help will always be remembered. We appreciate the help of Mrs. Karen Schanuel for obtaining and typing numerous patient charts and records. Mrs. Helen W. Armstead and Ms. Emma Routt, technicians of the Barnes Hospital Heart Station, deserve our thanks for their work in enabling us to obtain the vectorcardiograms and electrocardiograms, as does Mrs. Doris Larson, Chief Technician at the University of Kansas Electrocardiography and Vectorcardiography Laboratory, for obtaining and mounting many of the materials used for illustrations. We would also like to thank members of the Design and Illustration Section of the University of Kansas Medical Center, headed by Mrs. Beverly Sherrell, and the Departments of Surgical Illustration and Medical Illustration of Washington University. We want to express our deep gratitude to Mr. Alan Neider for his most skillful and able handling of the illustrations, which are so important in a publication such as this. For the consideration and cooperation of Year Book Medical Publishers, we wish to express our thanks. Finally, we would like to pay tribute to the late Thomas Walsh, M.D., whose enormous contribution to the original work is acknowledged by all of us.

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Table of Contents

PART III: THE CARDIAC ARRHYTHMIAS

PART IV: OTHER CONDITIONS AFFECTING THE ECTROCARDIOGRAM

APPENDIX: ILLUSTRATIVE VECTORCARDIOGRAMS AND ELECTROCARDIOGRAMS

PART I

The Normal Electrocardiogram and Vectorcardiogram

CHAPTER 1

Basic Considerations

THE ELECTRON MICROSCOPE and the microelectrode technique have added a great deal to our knowledge of the structure and function of the myocardium. A better understanding of various cellular phenomena has provided scientific explanations for many empirical observations in clinical electrovectorcardiography. Knowledge of the electrophysiology of various cardiac tissues (atrial and ventricular myocardium, Purkinje fibers, sinoatrial and atrioventricular nodes and internodal tracts) is essential for logical comprehension and interpretation of arrhythmias, hypertrophy, ischemia, infarction and conduction disturbance. With this knowledge of electrophysiology, one can better correlate electrocardiograms and vectorcardiograms with clinical abnormalities.

ANATOMY AND ELECTROPHYSIOLOGY OF THE HEART

The myocardium consists of columns of striated cardiac muscle cells approximately 100 μ long and 15 μ wide (Fig 1–1). These cells are entirely enclosed by a semipermeable membrane called the *sarcolemma*.[1] The myocardial cell has a central nucleus surrounded by columns of myofibrils. These myofibrils are embedded in the sarcoplasm, and mitochondria are interspersed between the individual myofibrils. Oxidative phosphorylation occurs in the mitochondria and produces high-energy phosphate bonds in the form of adenosine triphosphate (ATP), which provides the energy needed for contraction of the sarcomere.

The myofibrils are divided into sarcomeres, which are the basic contractile elements. A *sarcomere* is the unit of a myofibril between two consecutive Z bands and normally measures 2.2 μ in length (See Fig 1–1). This unit is delimited at either end by the dense Z band. From the Z bands at either end, the thin filaments (actin) pass centrally to interdigitate with the thick filaments (myosin). The latter make up the A band; the I band is the portion of the sarcomere where the thin filaments exist alone. The H zone is in the center of the sarcomere, where only myosin filaments are present. During muscle contraction, the H zone decreases in length. The myofibrils are arranged in transverse register so that their corresponding light and dark bands lie at the same level. The ends of myofibrils are inserted into intercalated disks, and the disks of adjacent cells appear to be fused in places called *tight junctions*. The low electrical resistance of the intercalated disks and the intimate relationship of cells at tight junctions would allow the transmission of a low-voltage electric impulse between myocardial cells, and this is self-propagating once initiated.[2] Cell-to-cell propagation of impulses is important in myocardial cells, since they lack the individual nerve axon-muscle cell relationship that is present in skeletal muscle.

The sarcolemma surrounding each myocardial fiber rests upon its basement membrane, which separates the interior of the cell from the interstitial fluid. This membrane functions to maintain the ionic and electric gradients that are present in the resting cell. There are transverse invaginations of the sarcolemma, called the *transverse* or *T tubular system*. These tubules encircle each myofibril at the

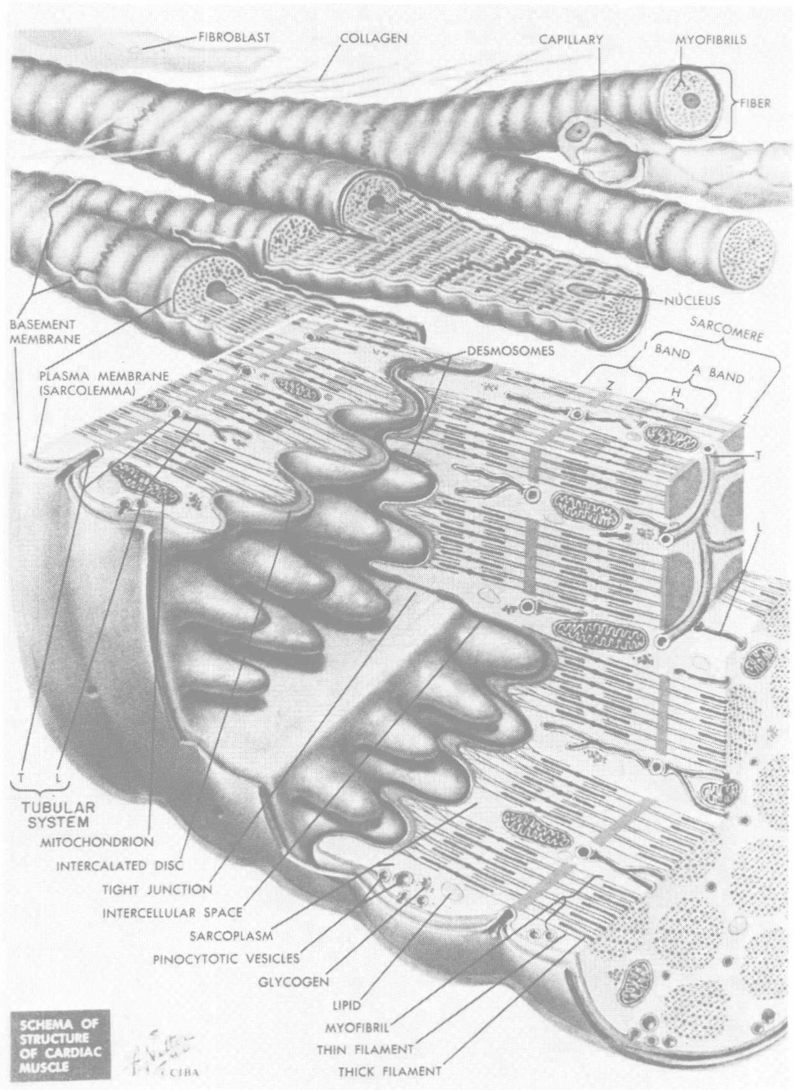

Fig 1–1.—A schematic drawing of the structure of cardiac muscle. The individual cell is composed of myofibrils, between which are interspersed various organelles such as mitochondria, the transverse tubular system and the sarcoplasmic reticulum. Note that the transverse tubular system, an extracellular structure, surrounds the myofibril at each Z band of the sarcomere. The sarcoplasmic reticulum, an intracellular structure, surrounds each sarcomere. (© Copyright 1969 CIBA Pharmaceutical Company, division of CIBA-GEIGY Corporation. Reproduced, with permission, from Netter, F. H.: *The CIBA Collection of Medical Illustrations* [Summit, N.J.: CIBA Pharmaceutical Company, 1969], p. 21. All rights reserved.)

level of Z bands (Figs 1–1 to 1–3) and are in direct continuity with the interstitial fluid that surrounds the cell.

Another tubular system, present entirely within the cell and consisting of an extensive network of longitudinal sarcotubules, is called the *sarcoplasmic reticulum* or *L* (longitudinal) *tubular system* (see Fig. 1–3). The individual units of the cell's sarcoplasmic reticulum each surround a myofibril and extend from Z band to Z band, thus being approximately 1 sarcomere in length (2.2 μ). The sarcoplasmic reticulum surrounding each sarcomere is

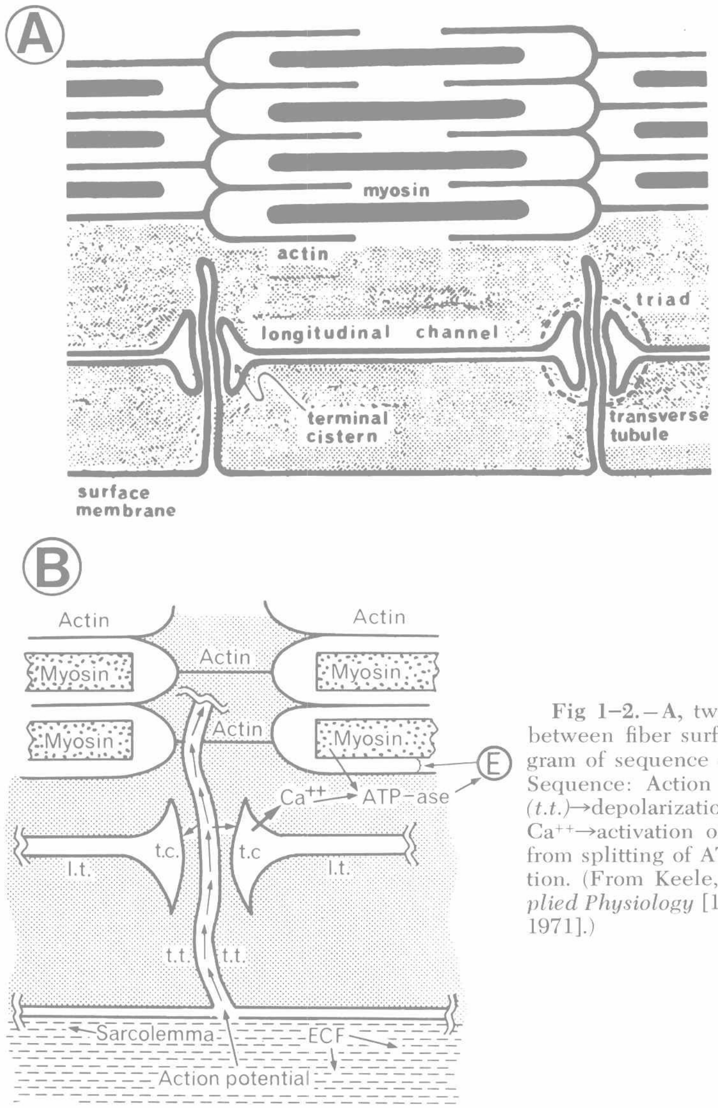

Fig 1–2.—**A**, two-dimensional diagram showing connection between fiber surface and the sarcoplasmic reticulum. **B**, diagram of sequence of events in excitation-contraction coupling. Sequence: Action potential conducted via transverse tubule *(t.t.)*→depolarization of terminal cistern *(t.c.)*→liberation of Ca^{++}→activation of myosin (= ATPase)→liberation of energy from splitting of ATP. Energy *(E)* causes actinomyosin contraction. (From Keele, C. A., and Neil, E.: *Samson Wright's Applied Physiology* [12th ed.; New York: Oxford University Press, 1971].)

not continuous with that of adjacent sarcomeres. The total volume of the sarcoplasmic reticulum, estimated on the basis of electron microscopic studies, is about 13% of the volume of the muscle fiber.[3] The sarcoplasmic reticulum does not contain a basement membrane. It appears to have large vesicles (called *lateral sacs* or *terminal cisterns*) that are in very close proximity to the T tubular system at the Z band but do not directly communicate with it. These areas of a cell, where the transverse tubular system and sarcoplasmic reticulum are apposed, are called *triads*.

The tubular system, consisting of transverse and longitudinal components, is involved in the transfer of excitation impulses to the contraction apparatus of the cell.[4] This is probably mediated by the transfer of calcium ions from the longitudinal tubular system or cell membrane (sarcolemma) to the myofibrils. The calcium ions initiate contraction by temporarily binding troponin, a protein which, in combination with tropomyosin, normally inhibits the interaction between actin and myosin.[5]

The energy for the interaction of actin and myosin is supplied by mitochondria-generated ATP,

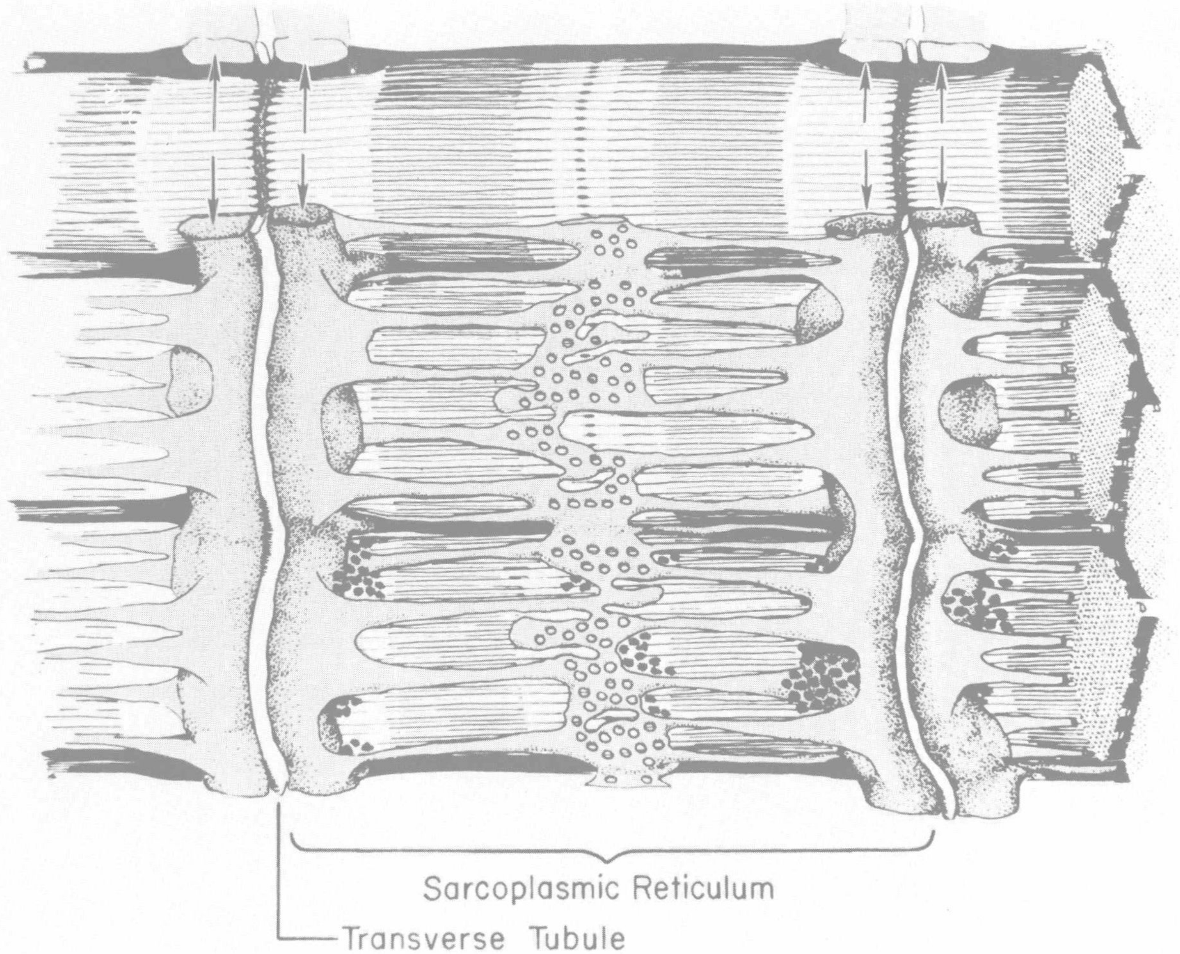

Fig 1–3. – Diagram showing the relationship of the sarcoplasmic reticulum and transverse tubular system to the myofibril. (From Peachey, L. D.: The sarcoplasmic reticulum and transverse tubules of the frog's sartorius, J. Cell. Biol. 25:209, 1965.)

which is readily available in the sarcoplasm around the sarcomere (see Fig 1–1). The interaction between actin and myosin is mediated via cross bridges between the head of the myosin molecule and actin, which allow the myosin filament to slip along the actin filament and thus decrease the length of the sarcomere.[6] With maximal contraction, the sarcomere is shortened by 20–50%. With passive stretching, the sarcomere may extend to about 120% of its normal length. It has been demonstrated, however, that the A bands, and thus the thick myosin filaments, always remain constant in length, whether the sarcomere is contracted, relaxed or stretched. Similarly, the distance between the Z line and the edge of the H zone remains constant at all stages of a normal contraction, indicating that the thin filaments likewise undergo no change in length (Fig 1–4). These findings indicate that muscle length must be due to sliding of the thick and thin filaments along one another, so that the degree of interpenetration or overlap of thick filaments by the thin filaments varies.[4] When muscle shortens maximally, the thin filaments may even slide past one another. These findings suggest that the cross bridges between the thick and thin filaments in the dense portion of the A band are rapidly formed and broken as the filaments slide along one another.

Following contraction of the sarcomere, calcium ions are actively taken up into the sarcoplasmic re-