MYOCARDIAL INFARCTION

electrocardiographic differential diagnosis

ARY L. GOLDBERGER, M. D.

THIRD EDITION

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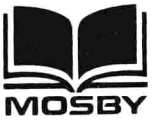

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Foreword

The diagnosis of myocardial infarction cannot always be readily made, as the electrocardiogram is not always diagnostic even in the presence of an established infarction. Similarly, the electrocardiogram may be misleading in that it may suggest the presence of an infarction when in fact one has not occurred either in the remote or recent past. Knowledge of the pitfalls in the electrocardiographic interpretation of infarction will inevitably help physicians to provide better care for their patients. The emotional and economic savings to a patient whose condition is not misdiagnosed as a myocardial infarction are at the least satisfying and at the most capable of changing the course of his life.

Dr. Goldberger has written a comprehensive, authoritative, and scholarly text that will be the definitive work in this field for decades to come. He provides examples and explanations of virtually all of the simulators of myocardial infarction. The book is written in a clear style and is richly illustrated with examples of each aberration described. I predict that it will be a classic in its own time. It was my privilege to be able to be associated with him during the preparation of this book.

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Preface

The art of clinical medicine is in large measure the art of differential diagnosis. Each facet of clinical evaluation—patient history, physical examination, and laboratory testing—entails its own, unfortunately often large, list of differential possibilities.

Perhaps nowhere are these problems so well illustrated as in the diagnosis of myocardial infarction. The so-called pathognomonic electrocardiographic changes of Q waves, ST segment deviations, T wave inversions, and tall, positive T waves are often as elusive and misleading as the crushing chest pains that prove to be acute cholecystitis or cardiac neurosis.

The main purpose of this book is to help refine the electrocardiographic diagnosis of myocardial infarction. The clinician who misinterprets the Q waves of the Wolff-Parkinson-White pattern or hypertrophic cardiomyopathy and assigns the diagnosis of coronary artery disease commits one error. However, his regrets are certainly no less than those of a colleague who overlooks the subtle, "hyperacute" T wave changes of an incipient myocardial infarction and sends the patient home with antacids.

The third edition of this book includes new material on numerous pseudoinfarct patterns, cardiomyopathies, ambulatory monitoring, and exercise testing, as well as revised discussions of the pathophysiology of the QRS and ST-T changes of myocardial infarction. The distinction between "subendocardial" and "transmural" infarct patterns is critically reassessed. In addition, new chapters have been added discussing the limited sensitivity of the electrocardiogram (ECG) in diagnosing coronary artery disease, the utility of the ECG in recognizing certain complications of infarction, and the integration of the ECG with other noninvasive tests in evaluating patients with possible acute infarcts.

In writing this book, I assumed a familiarity with basic electrocardiography on the part of the reader and attempted to continue where most textbook discussions end. As the clinician involved in the daily evaluation of patients soon discovers, it is the exceptions to the rule and the variations on classic themes that make up a large, and by far the most interesting, part of clinical practice.

A number of people contributed to the three editions of this book. In particular I wish to thank Dr. Emanuel Goldberger, my father, for his encouragement and

special interest and Dr. Lawrence S. Cohen for his guidance and advice. In addition I would like to thank the trustees of the Anna H. and Louis B. Goldberger Memorial Foundation for Medical Research for their support. Finally, I thank my wife Ellen for again abiding my absence and presence during the preparation of this new edition.

Ary L. Goldberger

Contents

1 Introductory concepts

Over the past two decades major new diagnostic technologies have been introduced into clinical cardiology, particularly in the areas of electrophysiology, radiology, ultrasonography, and nuclear medicine. The next decade promises even more exotic additions, including nuclear magnetic resonance. Given these rapid advances, the future role of the conventional electrocardiogram (ECG) might be questioned. Undoubtedly these new technologies will permit greater resolution in diagnosing biochemical and structural cardiac abnormalities. However, in the daily assessment of patients and the emergency evaluation of the critically ill, the ECG will retain its prominent and unique role as a noninvasive, immediately available, inexpensive, and highly versatile test. Familiarity with the uses and limitations of this test is therefore essential to all clinicians.

ECG PATTERNS OF MYOCARDIAL ISCHEMIA AND INFARCTION

One of the pivotal aspects of electrocardiography centers on the recognition and differential diagnosis of myocardial ischemia and infarction. Unfortunately for clinicians, there is no single ECG pattern associated with myocardial ischemia. Rather, as depicted in Fig. 1-1, the ECG may show a variety of depolarization (QRS) and repolarization (ST-T) changes, including Q waves, ST segment elevations, ST segment depressions, tall (positive) T waves, and deeply inverted (negative) T waves.

Three major factors account for this variability: the extent of ischemic injury ("transmural" versus "subendocardial"), the locus of ischemia (anterior versus inferoposterior part of the left ventricle), and the duration of ischemia (hyperacute or acute versus evolving or chronic).

Extent of ischemic injury: "transmural" versus "subendocardial"

Traditionally, myocardial infarctions have been divided on the basis of ECG-pathologic correlations into two groups: *transmural* (Q wave) infarcts, associated with a full or nearly full thickness of subepicardial and subendocardial necrosis, and *nontransmural*, or *subendocardial*, infarcts (without Q waves), in which the zone of necrosis is limited to the inner layer of the ventricle. However, as critics have appropriately pointed out,[29,30,33] this pathologic stratification based on ECG criteria is oversimplified and often misleading. Q waves may appear with sub-

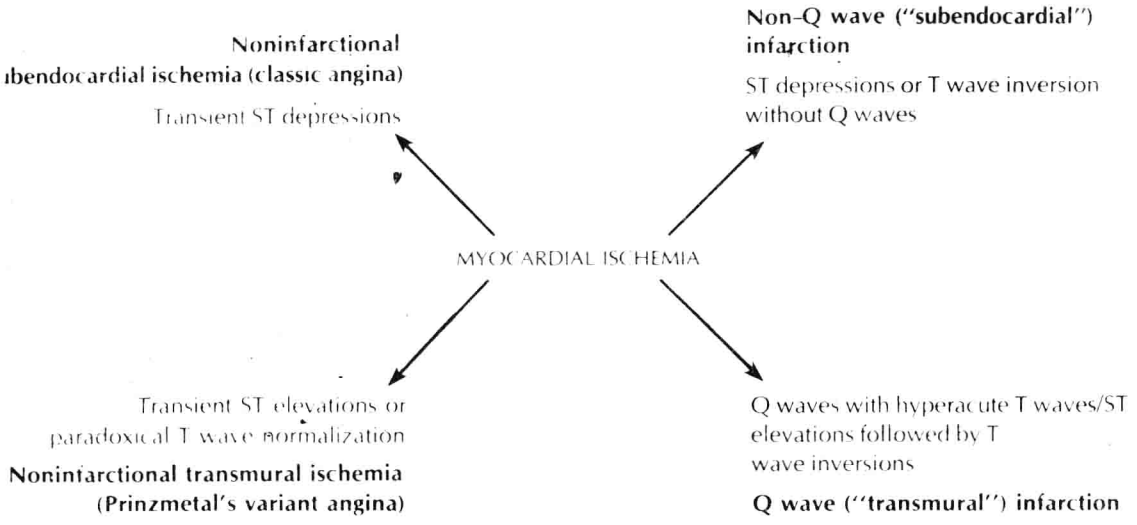

FIG. 1-1. Variability of ECG patterns associated with myocardial ischemia. As discussed in text, ECG may also be normal or nonspecifically abnormal.

endocardial infarcts, whereas not all transmural infarcts are associated with Q waves. Therefore in this book the terms *Q wave infarct* and *non–Q wave infarct*[30] have been adopted in preference to the usual "transmural" and "nontransmural" appellations. A detailed review of this somewhat controversial subject is provided in Chapter 2.

A related question is whether ST segment elevation is a reliable indicator of transmural ischemia and whether ST segment depression is a reliable indicator of subendocardial ischemia. This classic distinction, based on the polarity of the ST segment, has also been challenged; the debate over the pathogenesis of ischemic repolarization changes is reviewed in Chapters 8 and 11. The weight of evidence seems to support the association of ST segment elevation with transmural ischemia and ST segment depression with subendocardial ischemia, although the mechanism of these changes is unresolved. ST segment depression associated with predominant subendocardial ischemia may occur with typical anginal attacks, as shown in Fig. 1-1, or during exercise stress testing (Chapters 11 and 13). In contrast, transmural ischemia without infarction may be associated with transient ST segment elevation (Prinzmetal's variant angina, Chapter 8) or with paradoxical normalization of T waves (Chapter 18).

Locus of ischemia

Q wave infarcts tend to be localized to a region of the anterior or inferoposterior wall of the left ventricle. This topographic localization, discussed in detail in

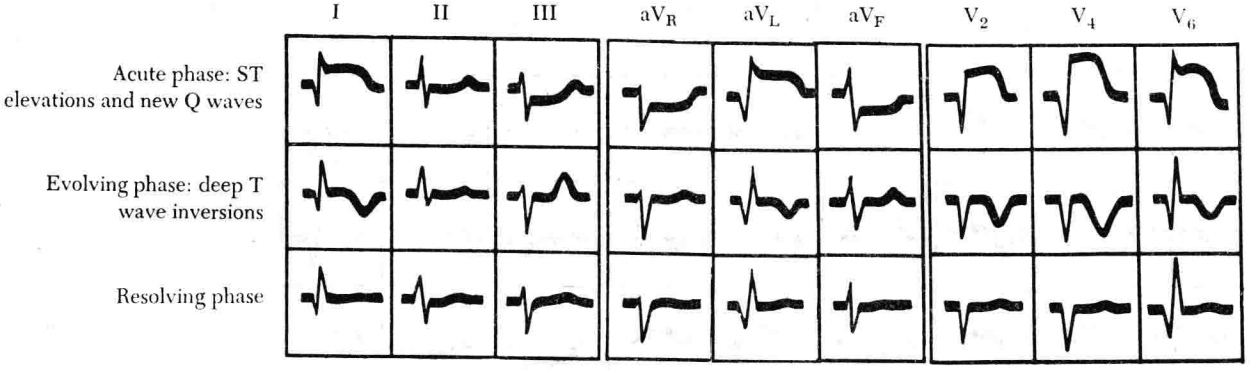

FIG. 1-2. Sequential QRS and ST-T changes seen with anterior wall Q wave infarction. Note reciprocal ST-T changes in inferior leads (II, III, and aV$_F$). (From Goldberger, A.L., and Goldberger, E.: Clinical electrocardiography, ed. 2, St. Louis, 1981, The C.V. Mosby Co.)

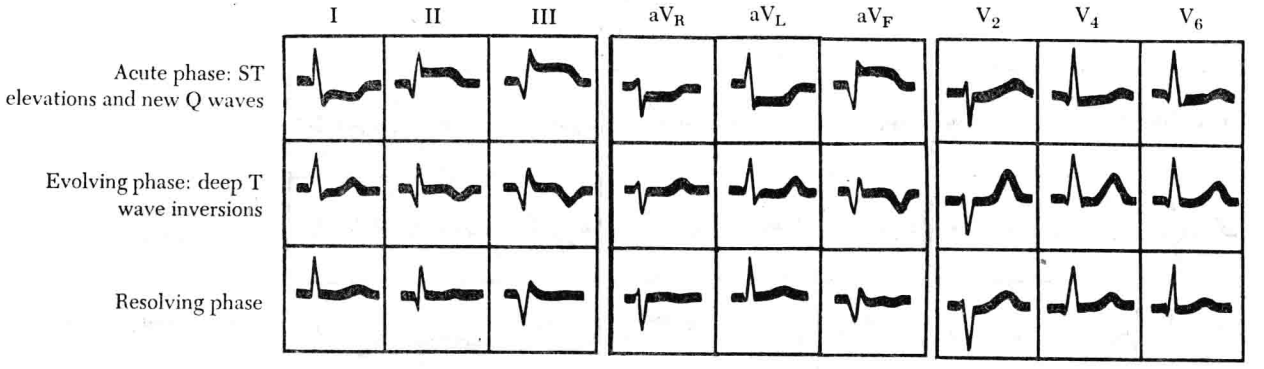

FIG. 1-3. Sequential QRS and ST-T changes with inferior wall Q wave infarction. Note reciprocal ST-T changes in anterior leads. (From Goldberger, A.L., and Goldberger, E.: Clinical electrocardiography, ed. 2, St. Louis, 1981, The C.V. Mosby Co.)

Chapter 2, reflects the regional distribution of the coronary arteries. Right ventricular infarction is discussed separately (Chapters 2 and 21).

Duration of ischemic injury

The timing of the ECG in relation to the onset of myocardial infarction is of major importance. For example, the evolution of a classic Q wave infarct is generally also marked by a progression of distinctive changes in the ST segment and T wave (Figs. 1-2 and 1-3). These changes can be temporally divided into *acute* and *evolving* (subacute, chronic) patterns. The hallmark of the acute phase is primary elevation of the ST segment ("current of injury") in one or more leads, usually with reciprocal ST depressions in other leads (p. 199). Occasionally this ST segment elevation is preceded or accompanied by tall, positive T waves, the so-called *hyperacute* T waves of infarction. These hyperacute T wave changes and acute ST segment alterations are then typically followed by inversion of the T wave, with the appearance of deep "cove plane" or "coronary" T waves in leads reflecting the area of infarction during the evolving (subacute or chronic) phase (Chapter 14). The electrophysiologic basis for these evolutionary repolarization changes is discussed in Part Two.

LIMITATIONS OF ECG IN DIAGNOSIS OF CORONARY ARTERY DISEASE

Most clinical tests produce both false positive and false negative results.[15] The ECG, despite its utility, is no exception. Normal ECGs *(false negatives)* may be found in the presence of significant underlying cardiac disease (Chapter 20). *False positive* ECG patterns may occur either when an entirely normal ECG variant is mistaken for an abnormal pattern or when a definitely abnormal pattern caused by one particular pathologic condition is mistaken for a similar pattern found in conjunction with a different abnormality.

The ECG is limited both in its *sensitivity* and *specificity* in diagnosing myocardial ischemia or infarction. The sensitivity of a test is a measure of the percentage of abnormal tests in patients with a particular disease. The more false negative results (for example, normal ECGs in patients with underlying infarction), the less sensitive the test. The specificity of a test is a measure of the percentage of normal (negative) results in patients without a particular disease. The more false positive results (for example, pseudoinfarct patterns), the less specific the test.

Sensitivity: the problem of false negatives

Limitations in sensitivity reflect the fact that chronic or even acute ischemia does not always produce diagnostic changes in the ECG.

Assessment of the sensitivity of the ECG in diagnosing myocardial infarction is also complicated by the timing of the ECG in relation to the onset of ischemia. For example, in the very earliest phase of an infarct the ECG may be normal or