

HEART DISEASE DIAGNOSIS AND THERAPY

CONTEMPORARY CARDIOLOGY

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HEART DISEASE DIAGNOSIS AND THERAPY

A Practical Approach

Second Edition

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HUMANA PRESS
TOTOWA, NEW JERSEY

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999 Riverview Drive, Suite 208
Totowa, New Jersey 07512

www.humanapress.com

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Production Editor: Robin B. Weisberg
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Printed in the United States of America. 10 9 8 7 6 5 4 3 2 1

eISBN: 1-59259-868-4

Library of Congress Cataloging-in-Publication Data

Khan, M. I. Gabriel.

Heart disease diagnosis and therapy : a practical approach / M. Gabriel
Khan.-- 2nd ed.

p. ; cm. -- (Contemporary cardiology)

Includes bibliographical references and index.

ISBN 1-58829-439-0 (alk. paper)

1. Heart--Diseases.

[DNLM: 1. Heart Diseases--diagnosis. 2. Heart Diseases--therapy. 3. Cardiovascular Agents--therapeutic use. WG 210 K45h 2005] I. Title. II. Contemporary cardiology (Totowa, N.J. : Unnumbered)

RC681.K47 2005

616.1'2--dc22

2004015846

DEDICATION

Dedicated to my wife Brigid

FOREWORD

Dr. Khan has done it again. For the last several years he has produced books at a rate usually achieved only by writers of romantic novels. With seemingly little effort he has authored more than a book a year packed with eminently usable information, and he has now capped his series of authored volumes with another masterpiece. *Heart Disease Diagnosis and Therapy: A Practical Approach* is a unique assemblage of what a physician dealing with cardiac patients needs to have at his or her fingertips—or at least within easy reach—when confronted with a challenging cardiovascular problem.

Unique? Yes, because Dr. Khan has disregarded tradition and convention to produce a reader-friendly and practical, yet up-to-date and comprehensive, treatise. By omitting unnecessary anatomy and physiology, for instance, he has made room for more detailed coverage of pharmacology, which is important to the wielder of the deadly drugs in the field. By avoiding long discussions of matters of little interest to the physician in the front line, he has managed to keep his book to a reasonable, handy size; in so doing, he has, in some important sections, managed to exceed the coverage of even the monumental Hurst and Braunwald tomes. His thrust is essentially clinical; his aim is to aid the clinician in his hour of need.

The electrocardiogram, often considered somewhat passé, is still the most often ordered, the most often diagnostic, the most cost-effective and yet, by cardiologist and computer alike, the most often misinterpreted of all cardiologic tests. Well aware of this, Dr. Khan has included a large number of illustrative electrocardiograms.

Two vitally important aspects of cardiology are the diagnosis and management of myocardial infarction and the recognition and treatment of cardiac arrhythmias. Accordingly, the practical aspects of these two challenges are handled in lavish detail. I would like to have seen emphasis on the reciprocal changes in the early diagnosis of acute inferior myocardial infarction, and details of the morphologic clues in the diagnosis of ventricular tachycardia could have been usefully expanded.

But no one could thumb through these pages without being impressed with the infinite amount of work that must have gone into their preparations and the author's breadth of knowledge. Whenever I read Khan, I am affected as the rustics were by Oliver Goldsmith's parson:

And still they gaz'd, and still the wonder grew
That one small head could carry all he knew.

Khan's knowledge is truly encyclopedic and, for his fortunate readers, he translates it into easily read prose.

Henry J. L. Marriott, MD, FACP, FACC

PREFACE

In preparing this second edition of *Heart Disease Diagnosis and Therapy: A Practical Approach*, I was determined that this book must remain clinically focused because of the changing role of clinical cardiologists. The majority of cardiologists prefer to be invasive cardiologists; those who practice noninvasive cardiology spend much of their time performing and interpreting noninvasive cardiac tests. Across North America and worldwide a large number of interns go into internal medicine programs. More than half go on to become general internists, and these internists render care to more than 60% of patients with cardiac problems. Thus, these physicians must acquire a sound knowledge base in clinical cardiology in order to render competent care to this large pool of patients that is not serviced by cardiologists. In particular, these trainees must have at their fingertips the basis for the clinical diagnosis and pharmacologic therapy of cardiac disorders. They do not require much information on invasive and noninvasive cardiac testing, neither of which they are called on to perform.

I believe that most trainees have difficulty extracting essential information from available large volume textbooks that are aimed at nonseasoned cardiologists. These are good reference books, but they are not study books. What type of textbook must the internal medicine resident use as a study book to improve clinical acumen and cram prior to the board examination? I strongly believe that such a text should:

- Have greater depth of coverage in clinical cardiology than the available medium-sized textbooks that range from 700 to 1000 pages; none of these books are suitable study books.
- Be thorough in its coverage. Most available texts are compressed; the tightly run lines make it difficult to use them as study books.

The format and printing style, therefore, should display the material so that the information can rapidly reach the visual cortex and be relayed to the storage area for memory in the brain. It is still necessary for students and senior trainees to commit the essentials to memory, and to rework these facts through patient's problem formulation and plan of management. The assessment of the factual information from a computer may suffice but this cannot replace the human touch at the bedside, where a sound knowledge base and clinical judgment can outsmart the computer program.

Many advances have been made in cardiology since publication of the first edition in 1996. The colossal amount of new scientific information necessitated the expansion of virtually all chapters in the preparation of this new edition. Results of recent randomized clinical trials are put in a special section in each chapter. An extensive current and relevant bibliography has been provided. The chapter on hypertension criticizes the national guidelines for the management of hypertension. The first edition warned that the World Health Organization, the International Society of Hypertension, and the JNC

should reexamine their logic for the recommendation of alpha blockers as initial therapy. The American College of Cardiology issued a warning in 2000, and the ALLHAT study that showed the detrimental effects of these agents was published in 2002.

We believe that a niche exists for a succinct user-friendly text that gives in-depth coverage of common cardiologic problems with emphasis on practical aspects of diagnosis, cardiovascular pharmacology, and other therapeutic strategies.

Our book is aimed at internists; clinical cardiologists; physicians in emergency rooms, intensive care units, and coronary care units; residents in cardiology, internal medicine, and family medicine; generalists; family physicians; and critical care nurses.

We did not intend to produce a comprehensive textbook of cardiology and intentionally did not discuss the following:

- Anatomy and physiology. A 20-page overview of this topic is not relevant to clinical practice. Clinicians and trainees have been sufficiently afflicted in their preclinical years with anatomy and physiology. Although we agree that physicians must be conversant with normal structure and function, a short coverage of the topic is irrelevant to the reader.
- Radiology of the heart. This is now used mainly to detect congestive heart failure, which is covered in our chapter on heart failure. The echocardiogram is superior for most other conditions. Thus, a discussion of radiology of the heart was omitted.
- Echocardiography. A superficial overview of this important diagnostic tool does not assist the intended audience. There are many excellent books on this subject.
- Congenital heart disease is adequately covered in pediatric cardiology texts.

The space saved by the omission of the aforementioned topics has made room in our text for expansion of areas that we believe are requirements for physicians and trainees who render care to cardiac patients. Thus, our text gives considerably more coverage than the available competing texts in the following areas:

- Coronary artery disease. Because coronary artery disease is the most common form of heart disease and manifests as acute myocardial infarction, angina, arrhythmias, heart failure, and sudden cardiac death, chapters on these topics are extensive.
- ECG. The ECG is the most commonly requested cardiac diagnostic test. Although there are sophisticated and extensive investigations available to cardiologists, the ECG is the main diagnostic test for the early diagnosis of acute myocardial infarction. To reap the benefit of saving lives, percutaneous intervention or thrombolytic therapy must be instituted at the earliest moment after the onset of symptoms; therefore, a rapid diagnosis is imperative. Early diagnosis cannot be made by evaluation of serum creatine kinase (CK) or troponin. The ECG, however, is subject to many errors in interpretation; many conditions mimic the electrocardiographic diagnosis of infarction. Our text, therefore, has in-depth coverage of the electrocardiographic diagnosis of myocardial infarction.
- Valvular heart disease is a common problem. Diagnostic pearls are bulleted; management is covered succinctly and with appropriate depth.
- Drug therapy of heart diseases. Practical cardiovascular pharmacology is a strong point of this book because it is the final prescription given to a patient after a consultation that ameliorates symptoms and saves lives. The prescription may, however, cause adverse effects and inadvertently increase the risk of death. Inappropriate prescribing of cardiovascular drugs is not an uncommon occurrence. Our book aims to strengthen the physicians' expertise in this vast area of relevant cardiovascular therapeutics. The old query, "What harm have you done today, Doctor?" still holds.

In the preparation of the text, we insisted that the discussion of appropriate therapy should be based on sound pathophysiologic principles to further strengthen the physician's ability to formulate a reasonable plan of management. Appropriate management and decision-making strategies require integration and orchestration of the following:

- Accurate diagnosis
- Pathophysiological implications
- Prediction of outcome or risk stratification
- Knowledge of the action of pharmacological agents and their correct indications
- Advantages and disadvantages of interventional therapy

To cover this wealth of clinical information, we prepared a succinct and straightforward text, highlighted by bullets to allow rapid retrieval of information. Chapters are formatted as follows: diagnosis and then therapy.

This clinically focused text should find a place in the hands of all residents in internal medicine and all clinicians.

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

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Acute Myocardial Infarction

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SIZE OF THE PROBLEM

More than 1.1 million patients have an acute myocardial infarction (MI) in the United States (US) annually, and more than 50% of these patients die within the first hour, caused mainly by arrhythmias, particularly ventricular fibrillation (VF). Of those admitted to a hospital, approximately 15% die during hospitalization. In addition, more than 1 million patients with symptoms suggestive of acute MI are admitted annually to coronary care units (CCUs).

From: *Contemporary Cardiology: Heart Disease Diagnosis and Therapy:
A Practical Approach, Second Edition*
Edited by: M. Gabriel Khan © Humana Press Inc., Totowa, NJ

In the year 2000, more than 12 million people worldwide died because of cardiovascular disorders mainly caused by the disease atheroma and subsequent thrombosis (atherothrombosis). It is estimated that by the year 2020, more than 24 million people will die annually from this disease in a world population of approximately 7.4 billion.

Intensive research is required to prevent atherothrombosis rather than the management of its complications, which include fatal and nonfatal heart attack, angina, heart failure (HF), abdominal aortic aneurysm, stroke, kidney failure, peripheral vascular disease causing intermittent claudication, and gangrene of the lower limb. Most research done in major institutions in the US and in developed countries is directed at the management of complications of atherosclerotic coronary artery disease (CAD). The worldwide advent of CCUs in the early 1970s, thrombolytic agents in the late 1980s, coronary angioplasty in the 1980s and 1990s, and stents during the past decade have improved survival but this can be considered as modest. Most importantly, the development of left ventricular (LV) assist devices (that are clearly a bridge to transplantation and not artificial hearts) requires considerable financial support for their development. They will save less than 1000 lives annually worldwide because these devices are not artificial hearts and their success depends on the ability to obtain donor hearts, of which, there are presently less than 4000 worldwide.

Obstructive atherosclerotic CAD leads to the following:

- Stable or unstable angina.
- Fatal or nonfatal MI.
- Sudden death.
- HF.
- Arrhythmias, atrial fibrillation, and thromboembolism that may cause stroke.

Approximately 20 million Americans have coronary heart disease, close to 7 million of whom have angina and more than 10 million have had a heart attack. The approximate economic cost of CAD and stroke in North America is approximately \$350 billion, with roughly \$120 billion for CAD. Although there has been a mild decrease in the incidence of CAD in North America during the past decade, the disease process and its complications are expected to increase because of an aging population.

Unfortunately in developing countries, the prevalence of CAD and its complications have increased in the past decade, and it is estimated that by 2020 cardiovascular disease (CVD) will reach epidemic proportions worldwide. It is relevant that developing countries constitute more than 80% of the world's population and in these regions, particularly in India and other Asian countries, the incidence of CAD is on the increase.

The high incidence of communicable, maternal, perinatal and nutritional diseases in these countries will fall from roughly 41 to near 17% but CVDs will increase from about 20% to more than 33% over the next 20 years. Japan is unique among the developed countries in that although the stroke rates were the highest in the world during the 1960s, the incidence did not rise as sharply as in other developed countries and have remained lower. In Japan, CVD rates have fallen more than 60% since the 1960s, largely because of a decrease in stroke rates. Life expectancies for men and women are the highest in the world and reach 77 years for men and 83 years for women.

The CAD mortality per 100,000 men and women respectively in countries where data is available is as follows:

- Russian Federation: 767 and 288.
- Ukraine: 749 and 342, yet in the neighboring Slovenia the mortality rate is low.

- Scotland: 655 and 273.
- Finland: 631 and 587.
- Portugal: 207 and 73.
- Spain: 181 and 52.
- France: enjoys the lowest cardiovascular mortality of all the developed countries; also, the CAD mortality is low: 142 and 36.

CVD mortality rates in Canada, New Zealand, and Australia are similar to those in the US.

Prevention

Yusuf et al. reported on an extensive case-control study in 52 countries. Nine modifiable risk factors were all significantly related to acute myocardial infarction:

- abnormal lipids
- smoking
- hypertension
- diabetes
- abdominal obesity
- psychosocial factors
- lack of daily consumption of fruits and vegetables
- regular physical activity
- regular alcohol consumption

CLINICALLY RELEVANT PATHOPHYSIOLOGY

Acute MI is nearly always caused by occlusion of a coronary artery by thrombus overlying a fissured or ruptured atheromatous plaque. The ruptured plaque, by direct release of tissue factor (TF) and exposure of the subintima, is highly thrombogenic. Exposed collagen provokes platelet aggregation. The extrinsic coagulation cascade is activated through the interaction between vascular TF and the circulating blood, causing *in vivo* generation of thrombin, which converts fibrinogen to fibrin.

Fibrin interacts with activated platelets to form a mesh structure that stabilizes the mural thrombus. Thus, atherothrombosis completes the occlusion of the artery. Lipid-rich atheromatous plaques contain TF associated with macrophages within the lesion that may enhance the thrombogenicity of these plaques. In coronary angiography performed during the early hours of ST elevation, MI has confirmed the presence of total occlusion of the infarct-related artery in more than 90% of patients. It is not surprising that aspirin, through inhibition of platelet aggregation, reduces the incidence of coronary thrombosis and is especially useful in prevention of the progression of unstable angina to thrombosis and MI. Chewable aspirin (160 to 320 mg) is particularly useful when given at the onset of chest pain produced by infarction. Patients must be informed that the use of chewable aspirin can prevent fatal and nonfatal infarction but that nitroglycerin does not. This advice should serve to motivate individuals to carry chewable aspirins for emergency use.

However, aspirin does not block all pathways that relate to platelet aggregation and does not nullify the intensely thrombogenic constituents of atheromatous plaques. In addition, aspirin does not decrease the incidence of sudden death in patients with acute MI. Aspirin does however reduce the incidence of MI in patients postinfarction and in those with unstable and stable angina. Thus, chewable aspirin administration plays a key role in the prevention and management of acute MI.

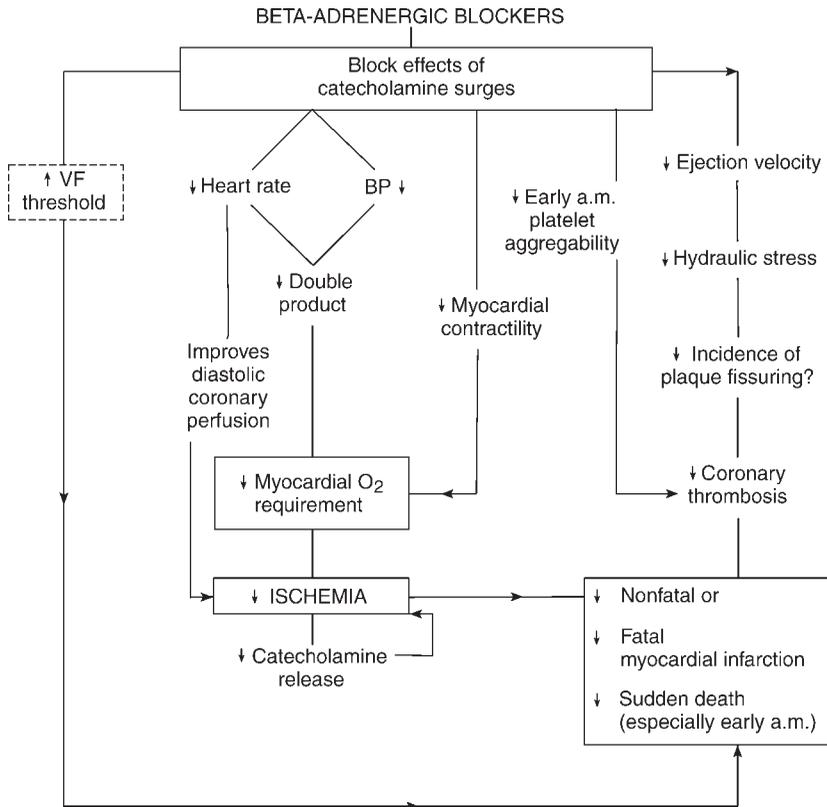


Fig. 1.1. Salutary effects of β -adrenergic blockade. \uparrow , increase; \downarrow , decrease. From Khan M. Gabriel, *Cardiac Drug Therapy*, Philadelphia, WB Saunders 2003, with permission from Elsevier.

The increased morning incidence of acute MI, documented in several studies of the diurnal variation of infarction, is related to the early-morning catecholamine surges, which induce platelet aggregation, and an increase in blood pressure and hydraulic stress, which may lead to plaque rupture (Fig. 1.1). β -adrenergic blockers have been shown to decrease the early-morning peak incidence of acute infarction and sudden death. It is important for clinicians to recognize that calcium antagonists and nitrates do not have these lifesaving effects and that these agents are overprescribed and β -adrenergic blocking agents are underused.

Unfortunately, when an atheromatous plaque ruptures, the thrombogenic effect of plaque contents cannot be completely nullified by the inhibition of all aspects of platelet aggregation, and chemical agents that can arrest the effects of these thrombogenic substances deserve intensive study. Preliminary studies in patients suggest that direct thrombin inhibitors such as hirudin, administered with aspirin, are effective in the prevention of coronary thrombosis. These studies may pave the way for further research that may uncover newer types of antithrombotic agents that are superior to available agents in preventing coronary thrombosis.

Coronary artery spasm appears to play a lesser role in the pathogenesis of coronary occlusion leading to infarction. Evidence of coronary vasoconstriction was found when angiography was performed shortly after infarction, and intermittent occlusion, presum-

ably on a vasomotor basis, has been apparent in some cases. Vasoconstriction appears to be a secondary factor.

The first gene linked directly to acute MI has been isolated from an extended Iowa family that has been plagued for generations with CAD. The gene, *MEF2A*, appears to protect the artery walls from building up atheroma. Individuals who have this gene mutation are destined to have the disease.

Vulnerable (High-Risk) Atheromatous Plaques

Plaque disruption is associated with physical forces, and occurs more frequently with the fibrous cap that is weakest, that is, when it is thinnest and most heavily infiltrated by foam cells. For eccentric plaques, this is often the shoulder or between the plaque and adjacent vessel wall. The shoulder regions of plaques often have a thinner fibrous cap that is highly infiltrated with macrophages and is prone to rupture. In sudden coronary death, often only a superficial erosion of a markedly stenotic and fibrotic plaque is observed

Thrombosis occurs over plaques because of the following:

- Denudation and erosion of the endothelial surface.
- Disruption or tear in the cap of a lipid-rich plaque; blood from the lumen enters the lipid core of the plaque, where thrombus is formed. Plaque disruption appears to be three times more common than the more superficial process of endothelial denudation. Sudden death as a result of CAD in relatively young subjects, however, has put the ratio of thrombi owing to plaque rupture compared with endothelial erosion as 1.3:1.

In sudden death in younger patients, plaque rupture is more commonly caused by endothelial erosion. Acute MI with thrombosis caused by endothelial erosion is reportedly more common at a younger age and particularly in women.

Three major factors determine the vulnerability of the fibrous cap:

1. Circumferential wall stress or cap fatigue.
2. Location, size, and consistency of atheromatous core.
3. Blood flow characteristics, particularly the impact of flow on the configuration and angulation of the plaque.

Plaque Rupture

Uneven thinning and fracture or fissuring of the plaque's fibrous cap leads to rupture. The porridge-like substances exposed to the flowing blood is highly thrombogenic and trigger thrombosis that blocks the lumen of the artery. This is the main cause underlying an MI. Fracture of the fibrous cap occurs often at the shoulders of a lipid-rich plaque where macrophages enter. The fibrous cap is believed to become thin because of the depletion of matrix components through the activation of enzymes such as matrix-degrading proteinases and cysteine and aspartate proteases, and through the reduction in the number of smooth muscle cells. Endothelial-cell desquamation through activation of basement membrane degrading metalloproteinases appears to be involved, but the mechanisms are unclear.

Activated T cells may also inhibit matrix synthesis through the production of interferon- γ . Evidence of superficial erosion of the intimal lining has been observed in approximately 25% of patients who had sustained an MI and died within a few hours.

- The provision of durable collagenous tissue processed by smooth muscle cells is important in maintaining the existence of the plaque's fibrous cap. Collagen provides most of the biomechanical resistance to disruption of the fibrous cap. Substances found in

degranulating platelets appear to increase smooth-muscle cell collagen synthesis that may reinforce the strength and viability of the fibrous cap. In addition, in some lesions there is a marked decrease in the presence of smooth muscle cells or increased smooth-muscle cell death within the plaque occurs and reduces collagen production.

- It is possible that the new capillaries and vessels within the plaque may be important for the survival of smooth muscle cells.

Platelets play an important role in initiating clotting in arteries and arterioles. Platelets form an initial plug of clot and are followed by the deposit of a fibrin mesh that forms a firm clot. Platelets are trapped by the material exposed by the fractured plaque, and the first phase of thrombosis is initiated. Aspirin or platelet glycoprotein (GP) IIa/IIIb receptor blockers are used to prevent this deleterious platelet aggregation.

Hemorrhage Into the Plaque

New capillaries and small vessels grow into the plaque and provide a useful function in that they may provide nutrient material for smooth muscle cells that form collagen necessary to strengthen the fibrous cap. However, these new vessels are fragile and may burst, causing a minute hemorrhage within the plaque. The pressure within the plaque may cause disruption of the fibrous cap, and thrombosis completes the occlusion of the artery. In approximately 5% of patients with acute MI, the initiating cause is hemorrhage into a plaque of atheroma rather than erosion–rupture followed by thrombosis. Angiogenesis and gene therapy may promote hemorrhage into plaques, and caution is required.

Myocardial Necrosis

In about 20 minutes, occlusion of a coronary artery leads to death of cells in areas of severely ischemic tissue, which will usually become necrotic over 4 to 6 hours. Because early and late mortality are directly related to the size of the infarct, limitation of infarct size (or even prevention of necrosis) initiated at the earliest possible moment is of the utmost importance. The ischemic zone surrounding the necrotic tissue provides electrophysiologic inhomogeneity that predisposes the occurrence of lethal arrhythmias. These arrhythmias are most common during the early hours after onset and contribute to one of the major mechanisms of sudden death.

Extensive myocardial necrosis is the major determinant of HF; papillary, septal, and freewall rupture; and cardiogenic shock in which more than 35% of the myocardium is usually infarcted. The most effective means of reducing the extent of myocardial necrosis is the administration of chewable aspirin and a β -blocking agent (metoprolol or carvedilol) and establishment of patency of the infarct-related artery by thrombolytic therapy or percutaneous coronary intervention (PCI) within 1 hour of the onset of symptoms of coronary thrombosis.

CLINICAL STUDIES THAT RELATE TO PLAQUE VULNERABILITY

Maehara et al.

Study question: What are the clinical and angiographic correlates of plaque rupture detected by intravascular ultrasound?

Methods: Three-hundred plaque ruptures in 254 patients were assessed by angiographic and intravascular ultrasound.

Results: The plaque rupture occurred in 46% of patients with unstable angina and 33% of patients with MI but was also observed in 11% of patients with stable angina. The tear

in the fibrous cap (63%) occurred at the shoulder and 37% in the center of the plaque. Thrombi were common in patients with unstable angina. The plaque rupture site contained the minimum lumen area site in only 28% of patients; rupture sites had larger arterial and lumen areas and more positive remodeling than minimum lumen area sites.

AM Varnava et al.

Study question: Is there a relationship between the morphologic characteristics of coronary plaque vulnerability, lipid core size and macrophage count and coronary artery positive remodeling (no lumen narrowing), or increased constrictive adventitial fibrosis and thickening with negative remodeling (lumen narrowing)?

Methods: The hearts of 88 male patients with sudden cardiac death were assessed.

Results: One-hundred-eight plaques were studied, 59% had positive remodeling and 40% had negative remodeling. Plaques with positive remodeling had a larger lipid core (39% vs 22%, $p < 0.001$) and a higher macrophage count. Plaques with negative remodeling were associated with greater thinning of the medial and adventitial wall opposite the plaque.

Conclusions: Plaques with positive remodeling have a high-lipid content and macrophage count. This may explain why plaque rupture often occurs at sites with only modest lumen stenosis.

The processes and mechanisms that underlie thinning, erosions, fracture, and rupture of plaques are unclear and are presently a subject of extensive research. Also, investigative strategies to define high-risk plaques must be sought.

Hydrodynamic Stress and Catecholamine Surge

Use of a β -blocking agent may inhibit plaque rupture perhaps by its ability to decrease cardiac ejection velocity. This action reduces hydraulic stress on the arterial wall that might be critical at the arterial site where the atheromatous plaque is predisposed to rupture or erosion (Fig. 1.1.).

- Catecholamine-dependent activity could explain not only the increase in the incidence of sudden death and acute coronary syndromes (ACS) after emotional and physical stress, but also the circadian distribution of these events.
- Only some β -blockers, however, have been proven to prevent fatal or nonfatal coronary events as well as HF; carvedilol and metoprolol are the preferred agents to be administered. **Atenolol a commonly used agent is not advisable (see later discussion of which β -blocking agent to choose).**

DIAGNOSIS

Chest Pain

- Usually lasts more than 20 minutes and often persists for several hours. The pain of infarction, however, can last for only 15 minutes, and, occasionally, fatal infarction is ushered in by only a few minutes of severe pain or even unheralded cardiac arrest. Infarction may be relatively silent, particularly in diabetic patients and in the elderly.
- Typically retrosternal and across the chest.
- Variations of a crushing, vice-like, heavy weight on the chest and pressure, tightness, strangling, aching.
- At times, only a discomfort with an oppression and burning or indigestion-like feeling.

- May radiate to the throat, jaws, neck, shoulders, arms, scapulae, or the epigastrium. At times, pain is centered at any one of these areas (e.g., the epigastrium, left wrist, or shoulder, without radiation).
- Upper epigastric and lower chest pain believed to be gastroesophageal in origin without feelings of indigestion is not uncommonly caused by a heart attack.
- Usually builds up over minutes or hours, as opposed to aortic dissection, in which pain has an abrupt onset like a gunshot.

Associated symptoms and factors include the following:

- Diaphoresis, cold clammy skin, and apprehension (however, all of these symptoms may be absent).
- Shortness of breath, nausea, vomiting, dizziness.
- Women with acute MI often reveal atypical symptoms with low levels of chest pain or absence of pain. In one study, acute chest pain was absent in 43%; acute symptoms were shortness of breath (57.9%), weakness (54.8%), and fatigue (42.9%).
- Presyncope and, rarely, syncope may occur owing to bradyarrhythmias, especially in inferior MI.
- Occasionally there is no pain. A marked decrease in blood pressure with associated symptoms, along with electrocardiogram (ECG) findings, should suffice in making the diagnosis.
- Painless infarcts (in about 10% of patients), especially in diabetics or the elderly. In these patients, associated symptoms are often prominent and serve as clues to diagnosis.
- More than 30% of patients have a history of angina or prior infarction.
- Approximately 33% of patients with acute infarction have no major risk factors, which include death of a parent or sibling younger than age 55, cigarette smoking, hypertension, or diabetes; and more than 25% have cholesterol levels less than 5.2 mmol/L (200 mg/dL). Importantly, absence of these factors should not influence the diagnosis.

Physical Signs

- Patient appears apprehensive, anxious, cold, clammy.
- Area of chest pain may be indicated with a clenched fist.
- Tachycardia 100–120 per minute. An increase in blood pressure owing to increased sympathetic tone is observed in approximately 50% of patients with anterior infarction.
- Bradycardia less than 60 beats per minute (BPM) and a decrease in blood pressure in about two-thirds of inferior infarcts; many of these patients become hypotensive, sometimes profoundly.
- S₄ gallop is common; S₃ and S₄ if in HF or cardiogenic shock.
- Murmur of mitral regurgitation as a result of papillary muscle dysfunction.
- Crepitations, more prominent over the lower third of the lung fields, may be present.
- Elevated jugular venous pressure owing to left and right HF or a very high venous pressure in the presence of right ventricular infarction or cardiac tamponade.
- Frequently, there are no abnormal physical signs, and this finding in a patient with suggestive symptoms should not decrease the level of suspicion that the patient may have an MI.

Although sophisticated tests have evolved to improve diagnostic accuracy, they are of limited value in the era of thrombolysis and aggressive PCI. Thus, a relevant history and correct interpretation of the ECG are of paramount importance in the implementation of early thrombolytic therapy, or PCI, which will be of greatest benefit if instituted within 2 hours of symptom onset.

THE ELECTROCARDIOGRAM

Despite varied criticisms and the advent of new and expensive diagnostic technologies, the ECG has retained its prominent and vital role as an irreplaceable noninvasive and inexpensive test for diagnosis of acute MI.

DIAGNOSTIC ECG FEATURES OF ST SEGMENT ELEVATION

Acute MI:

- ST segment elevation of at least 1 mm in two or more contiguous limb leads (Fig. 1.2).
or
- At least 1 mm ST elevation in two or more contiguous precordial leads (Fig. 1.3).

The above criteria, which have been used in most clinical trials of thrombolytic therapy, have become internationally standard and are considered diagnostic in patients with symptoms suggestive of acute MI. Where symptoms are not typical, the response to nitroglycerin is ascertained. Also, minimal ST segment elevation in black patients must be reassessed to exclude the occasional normal variant. There is clear recognition that Q-waves may evolve early or late and cannot be relied on for early diagnosis. Thus, the terms “transmural” and “nontransmural” have been abandoned and Q-wave or non-Q-wave infarction cannot be categorized in the early phase. The best differentiating feature is ST segment elevation, which is present in more than 90% of patients with acute coronary thrombotic occlusion.

In addition, later ECG signs of infarction include:

- Diminution of R waves (poor R wave progression).
- Evolving Q-waves.
- The simultaneous presence of reciprocal ST segment depression is not diagnostic of but provides major support to confirm the electrocardiographic diagnosis (Fig. 1.2).
- Patients who are developing non-ST elevation (non-Q-wave infarction) often manifest ST depression, or T-wave change (see later discussion of non-Q-wave infarction and ACS).

In patients with ischemic-type chest discomfort, ST segment elevation greater than 1 mm in two contiguous leads reportedly has a specificity of 91% and sensitivity of approximately 50% for diagnosing acute MI. The sensitivity increases to more than 85% with serial ECG done every 30 minutes for 6 hours or more in those in whom the initial ECG reveals no ST segment elevation. *See* later discussion of ST depression and non-Q-wave infarction.

Because the ECG is a vital yet nonspecific tool, it is necessary to correlate the ECG findings with the clinical presentation. In this regard, it is wise to recall Marriott’s “warnings”:

- An “abnormal” ECG does not necessarily mean an abnormal heart.
- Exclude normal variants (*see* later discussion of mimics).
- Consider causes of heart disease other than coronary.

If the first ECG is not diagnostic of acute injury or infarction but the patient is strongly suspected of having an ACS, the ECG is repeated every 30 minutes until diagnostic changes are observed and until troponin or creatine kinase MB (CK-MB) results are reported. If the ECG is equivocal and there is a strong clinical impression that acute MI is present, valuable clues may be obtained from an echocardiogram; a magnetic resonance imaging (MRI), if available, can assist in this clinical setting with the diagnosis of

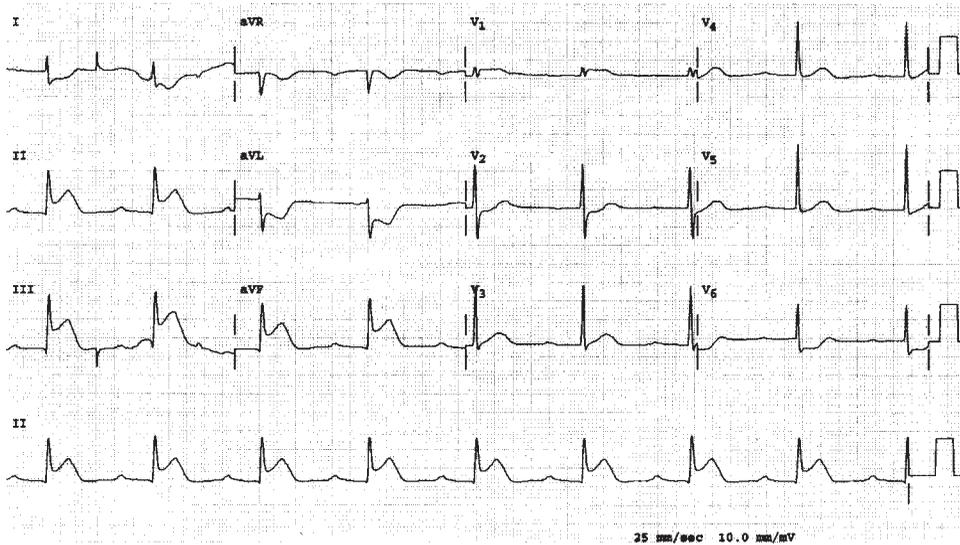


Fig. 1.2. ST segment elevation leads II, III, and aVF indicate acute injury, acute inferior infarction; note reciprocal ST segment depression.

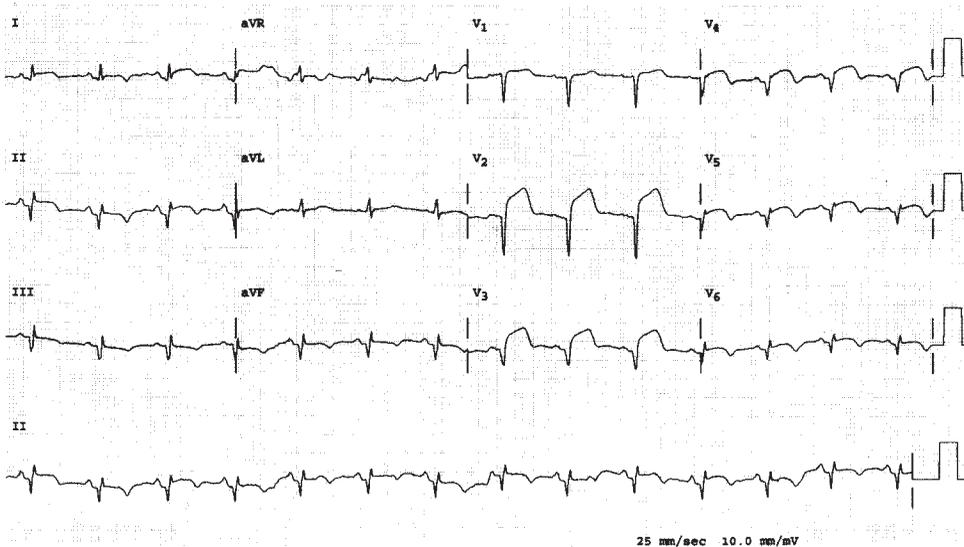


Fig. 1.3. ST segment elevation and Q waves V_2 – V_6 ; acute anterior infarction. Q waves leads II, III, and aVF: inferior infarct, age indeterminate.

acute MI. In patients presenting with ACS, an expensive MRI is not justifiable when the inexpensive and time-honored ECG reveals diagnostic ST segment elevation with reciprocal depression.

Because the initial ECG abnormality may not be fully diagnostic in up to 40% of cases, it is imperative to correlate the findings with accurate historical details. In patients with

chest pain, new or presumably new Q waves in two contiguous leads with ST elevation are diagnostic in more than 90% of cases:

- Q-waves are fully developed in 4 to 12 hours and may manifest as early as 2 to 4 hours from onset of chest discomfort or associated symptoms.
- Evolutionary ST-T changes occur during 12 to 24 hours but may be delayed up to 30 hours.
- Inferior MI: ST elevation in leads 2 and 3 and aVF with evolving Q-waves and reciprocal depression in leads 1, aVL, V₁–V₃. The latter depression may be the result of reciprocal changes, but there is evidence to suggest that in some patients it is owing to left anterior descending (LAD) artery disease. The evolutionary changes in repolarization that occur with inferior infarction evolve more rapidly than with anterior infarcts.
- Tachycardia may increase ischemic injury, causing elevation of the ST segment that must be differentiated from extension of infarction or pericarditis.

In pericarditis however, reciprocal ST depression occurs in aVR minimally in V₁, and does not occur in other leads. Also PR depression is a common feature of acute pericarditis (*see* Chapter 13).

VALUE OF LEAD aVR IN DIAGNOSIS OF ACUTE MI

- aVR is a lead that is often ignored, but recently has gained importance in the diagnosis of left main coronary artery (LMCA) occlusion.
- [Figures 1.4](#) and [1.5](#) show ST elevation in aVR that is greater than the elevation in lead V₁, marker of LMCA obstruction. This criterion is nonspecific: specificity is 80% and sensitivity is 81%. Circumflex branch occlusion also may cause ST elevation in aVR, both with no elevation in lead V₁. In addition, right ventricular overload may reveal ST elevation in aVR, but the clinical scenario is easily differentiated. Subendocardial infarction with marked ST segment depression in V₄ through V₆ that is not caused by LMCA occlusion may reveal ST segment and elevation in aVR, but the elevation is less than that observed in lead V₁.
- Because LMCA occlusion is a highly serious condition, any noninvasive diagnostic clue represents a valuable addition for the clinical assessment of acute MI.

NONDIAGNOSTIC ECG

Acute MI may be present with ECG changes that are nonspecific in 10–20% of cases and may result from the following:

- Slow evolution of ECG changes. The tracing may remain normal for several hours.
- Old infarction masking the ECG effect of a new infarct.
- Inferior MI associated with left anterior hemiblock in which R waves are expected to be small or minute in lead 3 and aVF.
- Left bundle branch block (LBBB).
- Apical infarction.
- Posterior infarction not associated with ST elevation or Q waves.

ECG and Location of Infarction Sites

- Anteroseptal: ST elevation V₁, V₂, V₃, may involve V₄ ([Fig. 1.6](#)). [Figure 1.7](#) shows the evolutionary changes in the same patient 10 hours later.
- Anterior: ST segment elevation V₃–V₄, may involve V₂ and V₅ ([Fig. 1.3](#)).
- Extensive anterior: V₁–V₆, I aVL ([Fig. 1.8](#)).
- Anterolateral: V₅–V₆, I aVL, may involve V₄ ([Fig. 1.9](#)).
- Inferior: II, III, aVF ([Fig. 1.2](#), [1.10](#), [1.11](#)); inferolateral II, III, aVF, V₆, may involve V₅, aVL.

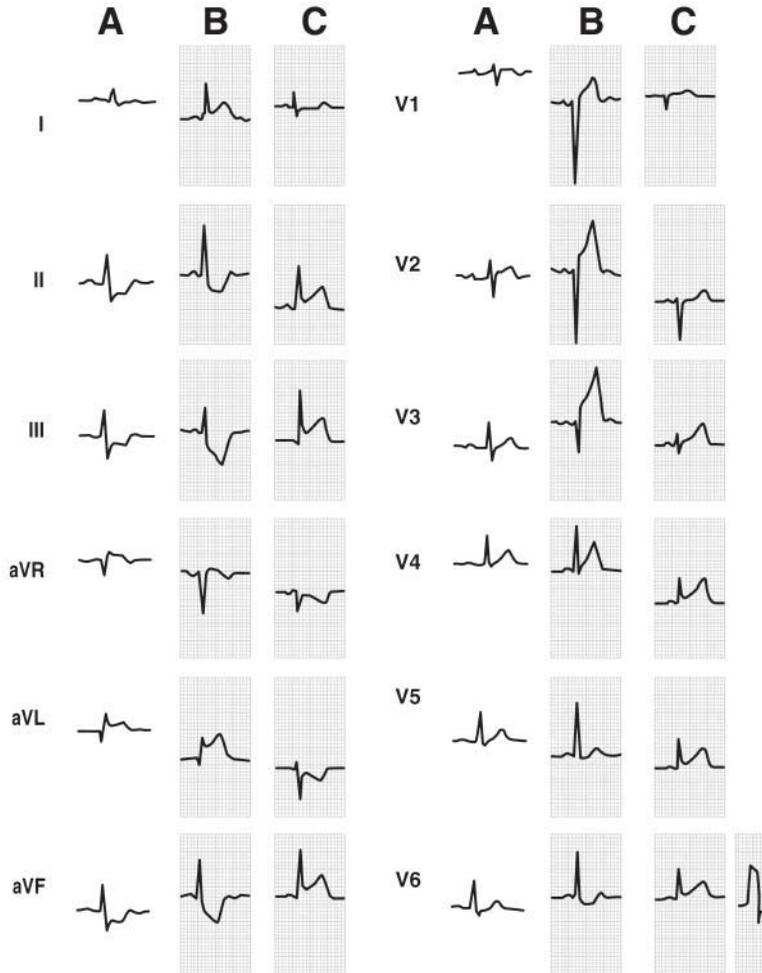


Fig. 1.4. Representative 12 lead ECG tracings at admission in a patient in the left main coronary artery (LMCA) group (A), the left anterior descending (LAD) coronary artery group (B), and the right coronary artery (RCA) group (C). In the patient in the LMCA group, ST segment elevation is apparent in lead aVR. In the patient in the LAD group, significant ST segment elevation in the precordial leads is seen, whereas ST segment shift in lead aVR is negligible. In the patient in the RCA group, ST segment elevation in the inferior leads is marked. From Yamaji H et al: J Am Coll Cardiol 58:1351, 2001, with permission from American College of Cardiology Foundation.

- Posterior infarction: Tall R waves and upright T waves in V_1, V_2 (Fig. 1.12.); occasionally ST depression V_1-V_2 , and often inferior or inferolateral infarct signs.
- Right ventricular infarction: ST segment elevation V_3R, V_4R , associated with inferior infarction (Fig. 1.13.).
Localization of infarction from the ECG is, however, not precise.
- LMCA occlusion: ST segment elevation in lead aVR is greater than elevation in lead V_1 associated with ST depression in lead aVL, V_4 to V_6 (Figs. 1.4., 1.5.).

ECG and Size of Infarction

The extent of ST segment elevation gives clues to infarct size, but the correlation is not close. The site of infarction influences mortality but is not as paramount as the size of

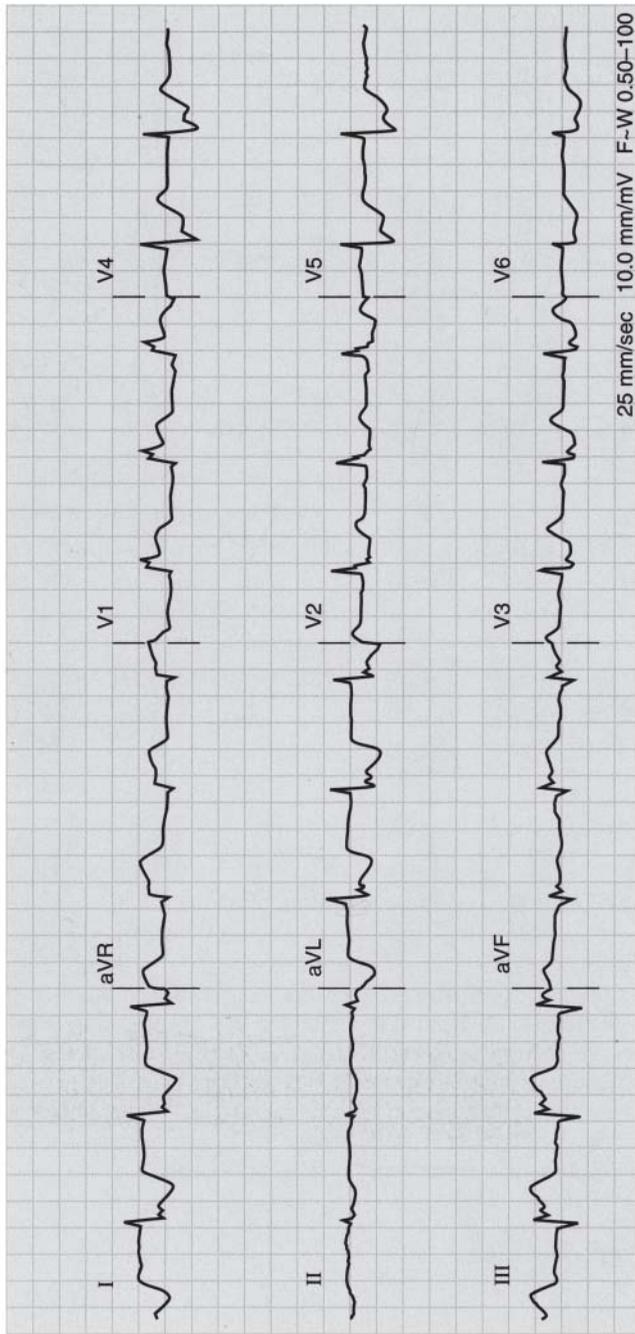


Fig. 1.5. Patient with chest pain for 3 hours. Inferior myocardial infarction and ST elevation in aVR and a V₁ indicates left main occlusion. From Khan M Gabriel. Rapid ECG interpretation, Second edition, Philadelphia 2003. WB Saunders, with permission from Elsevier.

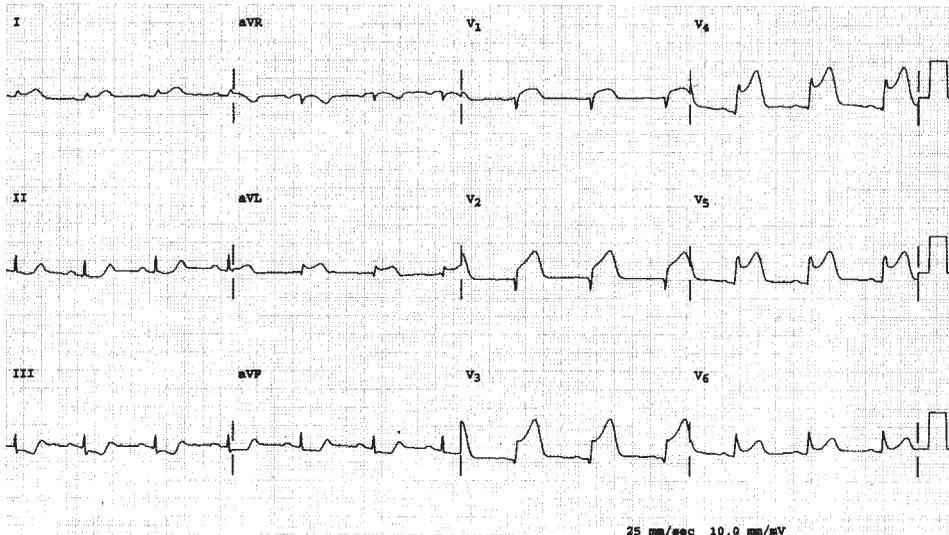


Fig. 1.8. Marked ST segment elevation in eight leads: I, aVL, V₁–V₆: extensive anterior infarction; note reciprocal depression in inferior leads.

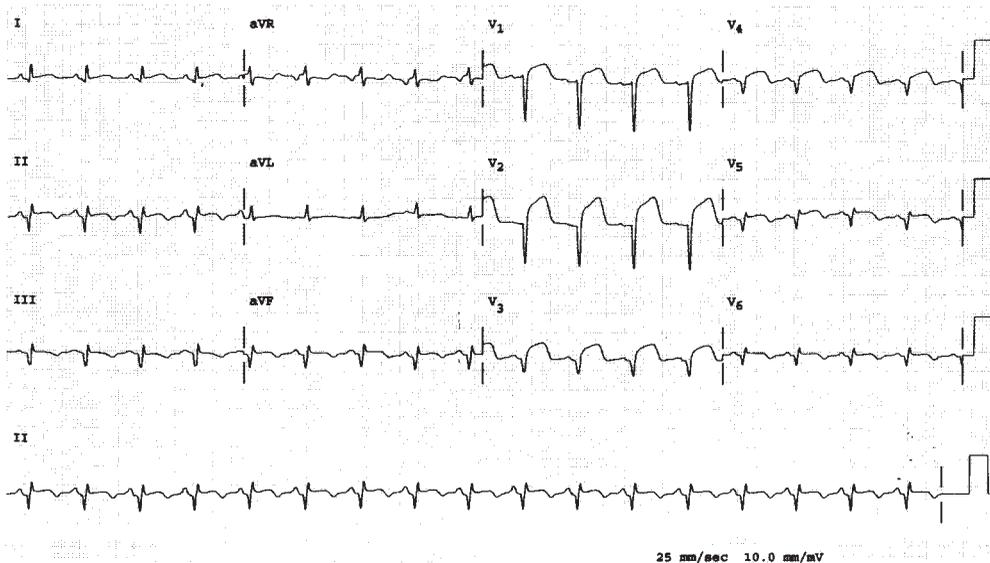


Fig. 1.9. Acute anterolateral infarction; inferior infarct age indeterminate.

ECG MIMICS OF MYOCARDIAL INFARCTION

- So-called early repolarization pattern: If early repolarization pattern involves limb leads, the ST segment is more elevated in lead II than in lead III. Early repolarization of atrial tissue is also present, resulting in PR-segment depression, but the PR-segment depression is not as marked as that in patients with acute pericarditis in which there is reciprocal ST segment depression in lead aVR but not in aVL, whereas in most patients with inferior

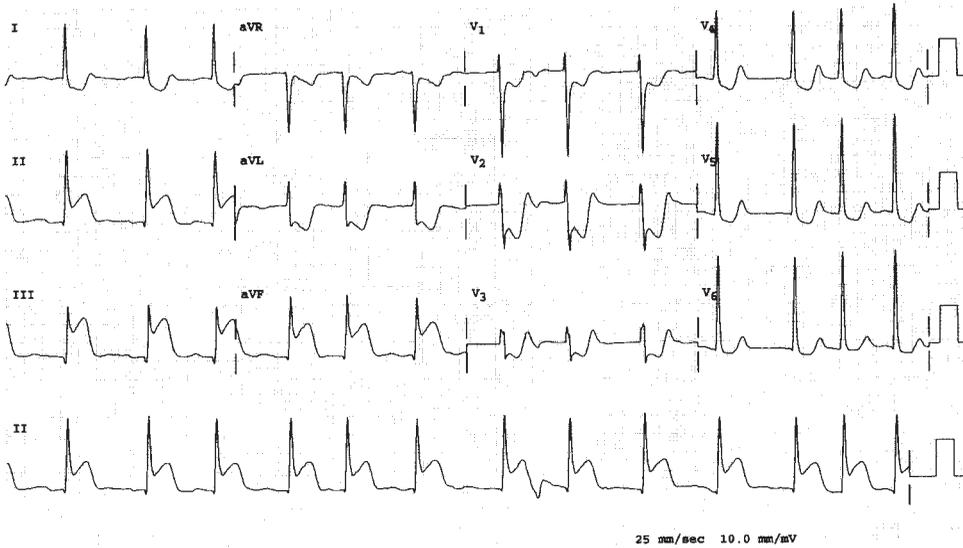


Fig. 1.10. ST elevation leads II, III, and aVF and marked reciprocal depression anterior and lateral leads: acute inferior infarction; also, acute atrial fibrillation.

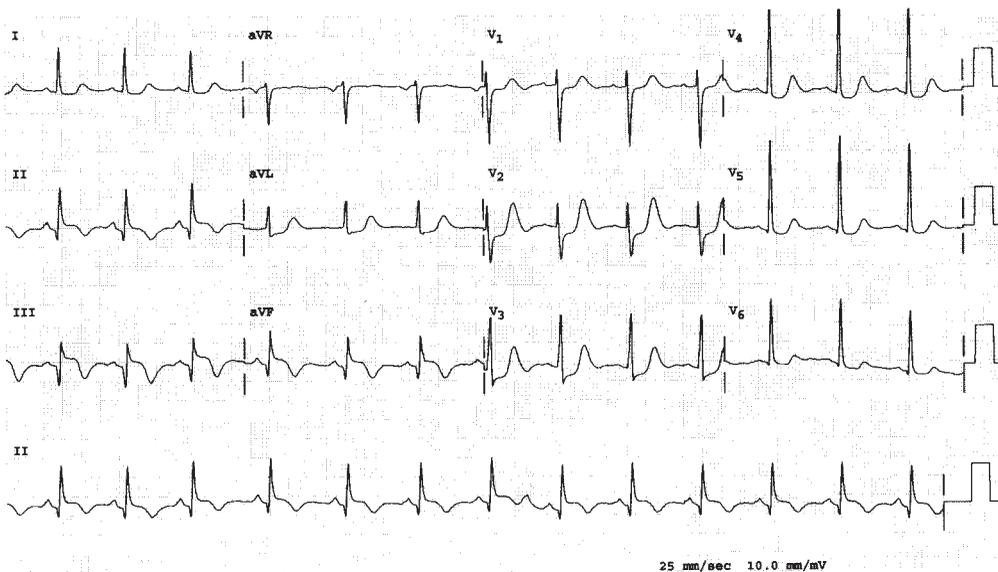


Fig. 1.11. ECG from the same patient as in Fig. 1.8., 24 hours later, indicates evolutionary changes.

infarctions, the ST segment is often more elevated in lead 3 than in lead 2 and there is reciprocal ST-segment depression in lead aVL (Fig. 1.10.).

- In some young individuals, especially African-Americans, the ST segment is elevated in V₃ to V₅ associated with minor T-wave inversion as a normal variant; the ST segment tends to be slightly coved and may mimic acute MI, and caution is required to assess the clinical findings (see pp. 20 and 21).

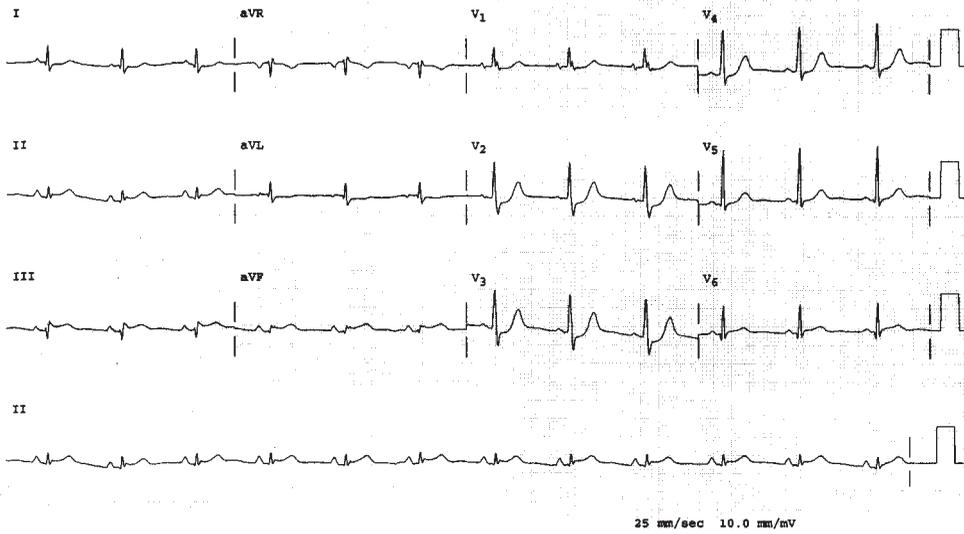


Fig. 1.12. Acute inferior infarct. Note tall R waves V₁-V₂ in the absence of right ventricular hypertrophy, WPW or RBBB and thus in keeping with posterior infarction; note upright T wave V₁, V₂.

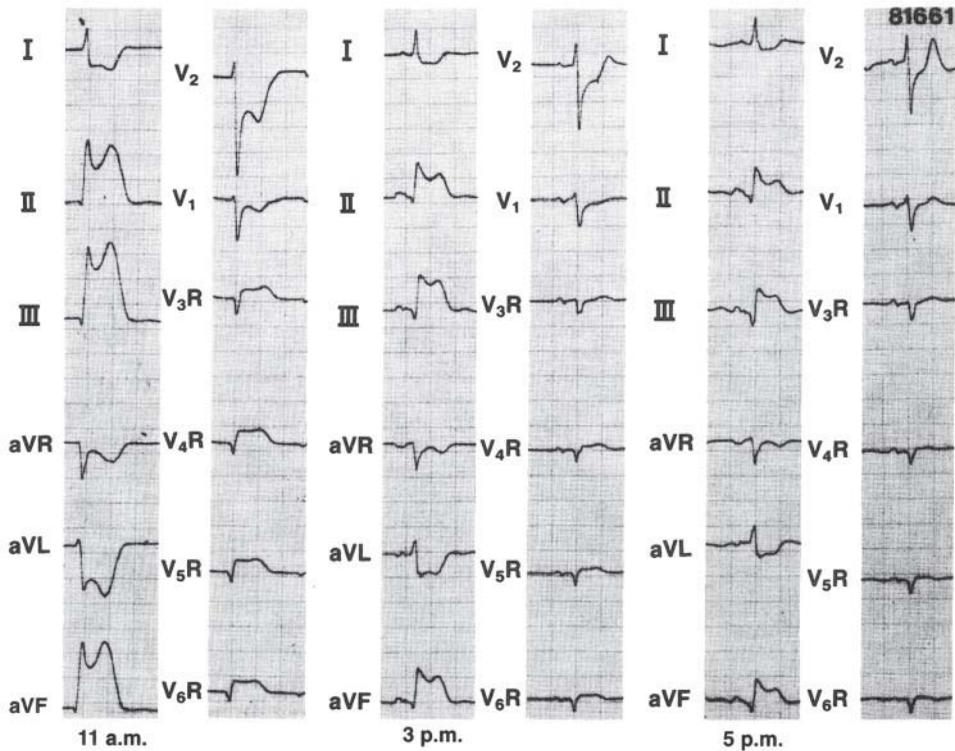


Fig. 1.13. Serial tracings from a patient with acute inferoposterior and right ventricular infarction. Note that the diagnostic changes for right ventricular infarction seen in lead V₄R have disappeared 7.5 hours after the onset of pain. From Wellens Hein JJ, Conover MB. The ECG in emergency decision making. Philadelphia: WB Saunders, 1992:92. Reprinted with permission from Elsevier.

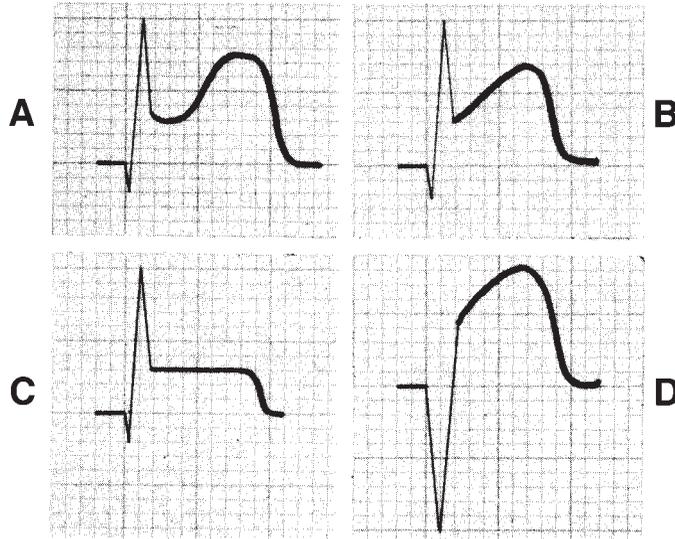


Fig. 1.14. Types of ST elevation seen with acute myocardial infarction (current of injury pattern). (A) Upwardly concave. The ST segment appears to have been lifted evenly off the baseline. A similar pattern occurs with benign early repolarization variant and acute pericarditis. (B) Obliquely straightened. (C) Plateau shaped. (D) Convex (similar elevations to this are sometimes seen in the right precordial leads with left bundle branch and left ventricular hypertrophy in the absence of infarction). From Goldberger AL. Myocardial infarction, electrocardiographic differential diagnosis. 4th ed. St. Louis: Mosby Year Book, 1991. Reprinted with permission from Elsevier.

These ST segment elevations meet the criterion for acute MI, according to the guidelines of the American College of Cardiology/American Heart Association (ACC/AHA): “ST elevation greater than 0.1 mV in two or more contiguous leads.” Because of this misleading criteria, The Clinical Policies Subcommittee of the American College of Emergency Physicians have added the qualifier “ST-segment elevations that are not characteristic of early repolarization or pericarditis, nor of a repolarization abnormality from LVH or bundle-branch block.”

Types of ST segment elevation caused by acute MI are illustrated in [Fig. 1.14](#).

Most important, ST elevation of infarction must be distinguished from the following:

Acute Pericarditis

- The ST segment is elevated diffusely in the limb leads as well as in the precordial leads and are not confined to leads referable to an anatomic segmental blood supply as occurs with acute MI.
- Elevation in lead I is accompanied by elevation in leads II, III, and aVF; the ST elevation is concave ([Fig. 1.15](#)), as opposed to convex upward with an injury current of infarction ([Fig 1.14](#)).
- Reciprocal ST depression and PR segment elevation in aVR is a typical finding with pericarditis ([Fig. 1.15](#)).
- The PR segment is diffusely depressed in pericarditis whereas, in acute infarction, the PR segment is not depressed except with the rare occurrence of atrial infarction.

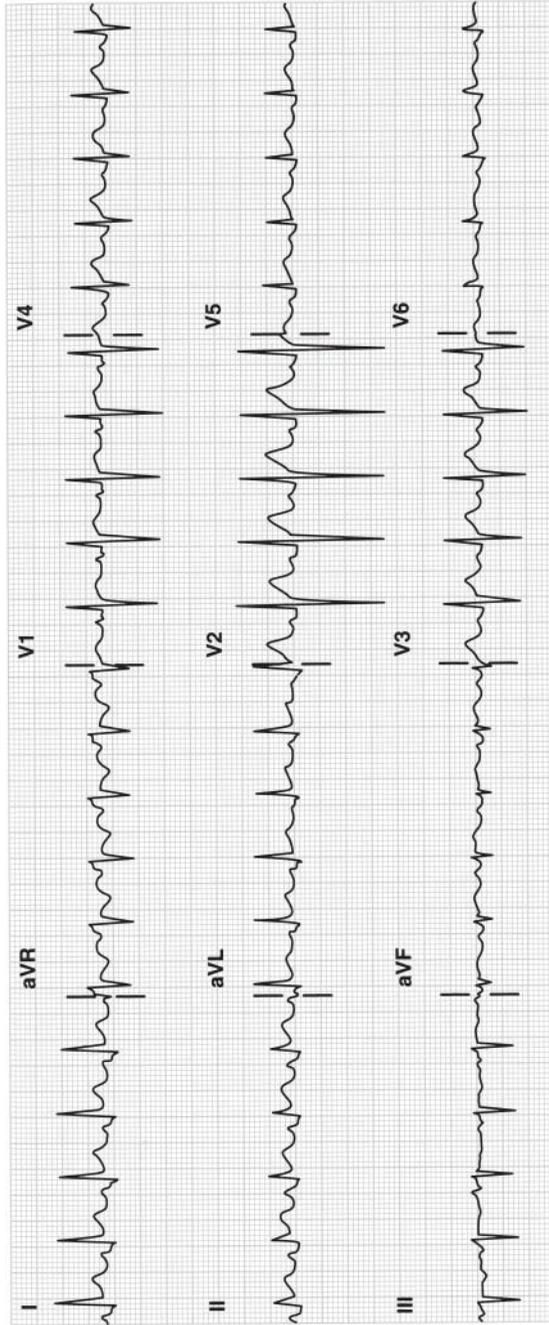


Fig. 1.15. Characteristic features of acute pericarditis: ST segment elevation in most leads; I, II, aVL, AVF, V₅ and V₆, with reciprocal ST depression and PR segment elevation in aVR. In addition note sinus tachycardia and prominent PR segment depression commonly seen with acute pericarditis. From Khan M Gabriel. Rapid ECG Interpretation, Second edition, Philadelphia 2003. WB Saunders, with permission from Elsevier.