American College of Emergency Physicians®
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The Textbook of Emergency Cardiovascular Care and CPR

EDITOR-IN-CHIEF
John M. Field

AHA EDITORS
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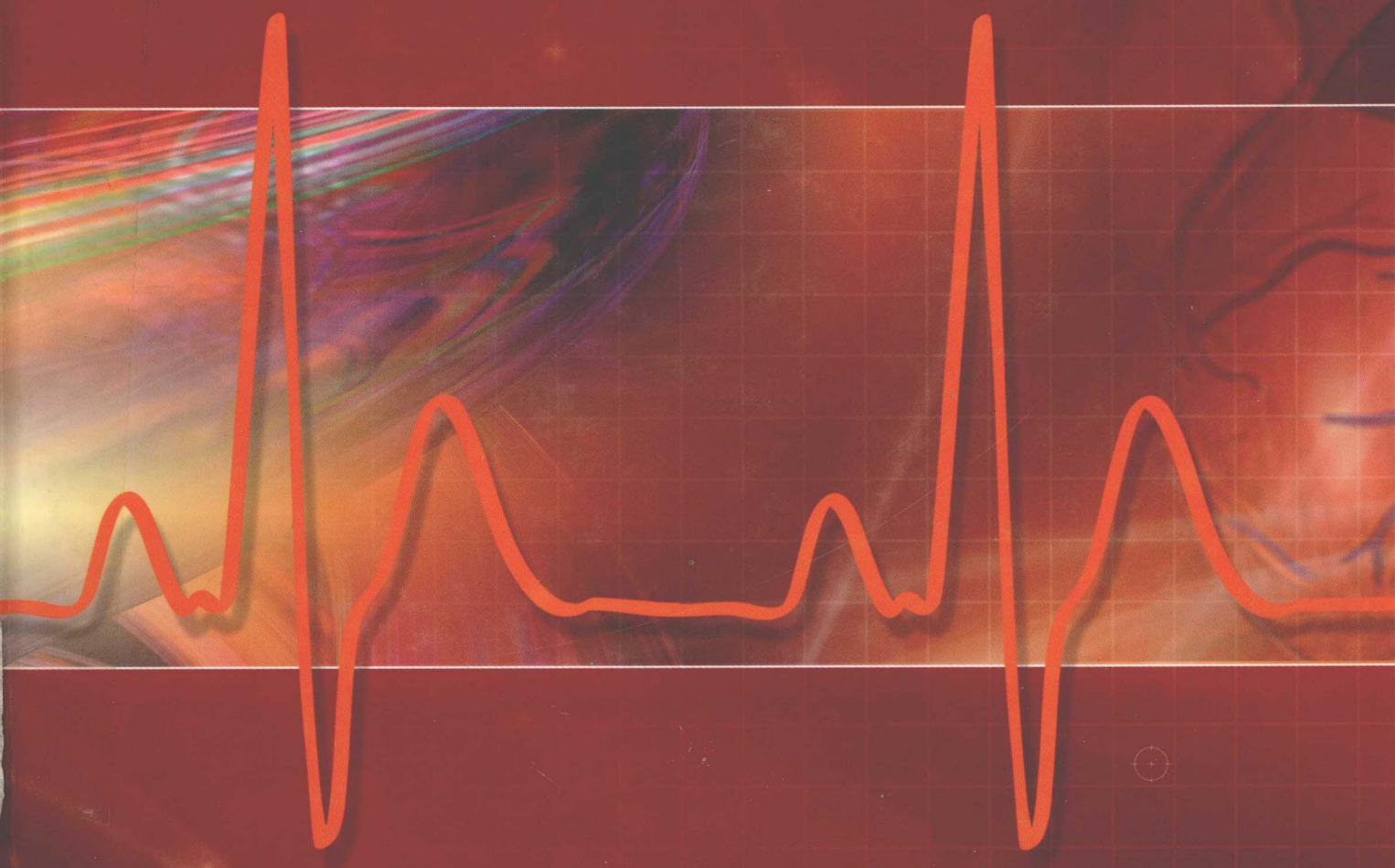

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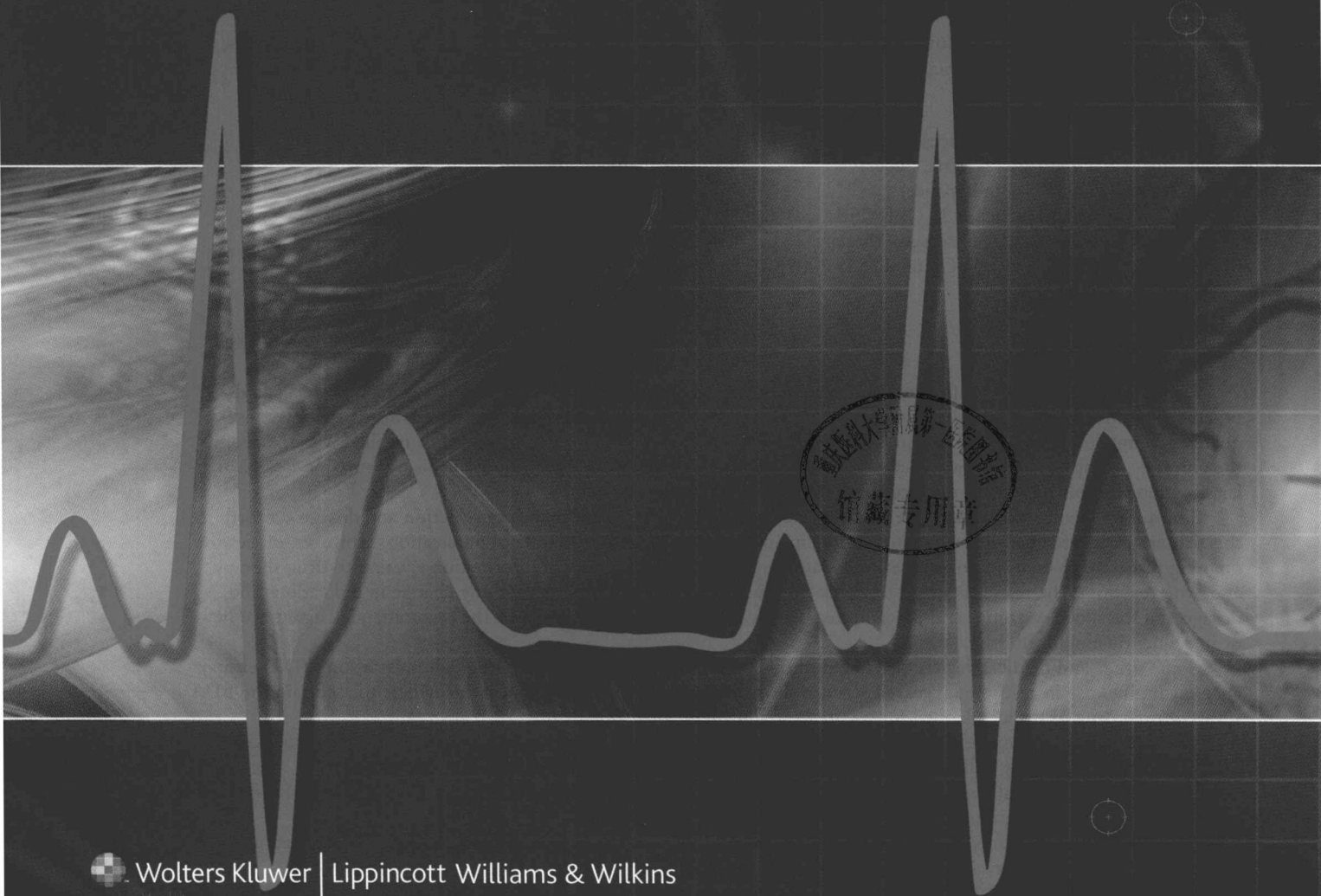

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Foreword

In the 1950s and 1960s anesthesiologists, engineers, and cardiologists ushered in the modern era of resuscitation, combining mouth-to-mouth ventilation, closed-chest compressions, and electrical defibrillation of the heart. Early pioneers summarized their science and treatment recommendations in the *First Conference on CPR* sponsored by the National Academy of Science in 1966. Over the following half century, emergency cardiovascular care and resuscitation science have continued to provide a bench-to-bedside approach for improving timely emergency care to patients suffering from an acute cardiovascular event, such as sudden cardiac death.

As recognized by those early pioneers, improving patient outcomes requires dedicated effort from many: multiple specialties and sub-specialties, national and international scientists and clinicians, and millions of individuals trained and ready to act. Collaboration, cooperation, and participation of these many dedicated groups and individuals are required for the successful translation of science into clinical practice, including the effective transmission of both information and skills to prepare individuals who are not only skilled, but also willing and able to take appropriate action.

The American Heart Association and the American College of Emergency Physicians have assumed leadership roles in emergency cardiovascular care, recognizing that successful patient outcomes depend on not only effective action in the first minutes to hours of the acute event, but also the decisions made over the next days to weeks. The placement of a stent for an acute ST-segment elevation myocardial infarction (STEMI) or an automated implantable cardioverter/defibrillator (AICD) for survivors of sudden cardiac death begins within the community and is facilitated by knowledgeable healthcare providers, EMS systems, and skilled emergency physicians.

This textbook represents the recognition and sponsorship of these principles and educational efforts. The textbook is the first major cooperative collaboration in this area. Experts in clinical emergency cardiovascular care summarize state-of-the art strategies to promote interdisciplinary excellence in resuscitation and emergency medicine, and scientists and educators provide insight into implementation strategies and careers. Key AHA and ACEP guidelines and clinical policies are immediately available and referenced and provide science and consensus recommendations in critical areas.

Continuing collaboration will have one objective: to provide the best care and the most favorable outcomes for victims of sudden cardiac arrest and other cardiovascular emergencies by optimizing what is done in the first minutes and hours and days, when critical interventions often make the difference between survival and death, between functional recovery and disability.

Timothy J. Gardner, MD, FAHA, President
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Preface

This first edition of *The Textbook of Emergency Cardiovascular Care and CPR* is a humble effort by the editors to bring together the work of many others in the field of emergency cardiovascular care. Special emphasis is placed on the practicing physician and other healthcare providers who strive to intervene on behalf of patients with an acute cardiovascular emergency within the first several hours after onset—often the most critical moments that determine short-term survival and long-term prognosis.

The core content of the book has been written and edited by experts in the field of resuscitation and emergency cardiovascular care, but more broadly, it brings together the collective efforts of many others who have committed their time and effort to this field. As such, the components of this book and its electronic version are intended to provide expeditious and efficient access to evidence-based materials, clinical guidelines, and scientific treatises in the relevant topics.

Each core chapter is an up-to-date summary of current research and clinical practice. Within each chapter, links to AHA ECC Guidelines, ACEP Clinical Policies, International Liaison Committee on Resuscitation (ILCOR) and AHA Scientific Statements and worksheets, and ACC AHA Guidelines are available so the reader can reference and review the evidence in context. Where applicable, audiovisual aids are also included to integrate the material. Readers can access AVI clips online that demonstrate performance of a technique or diagnostic procedure.

This format recognizes that the *individual practitioner* and *healthcare providers* must ultimately apply group data and consensus recommendations to each patient in an emergent situation in a timely and comprehensive manner. In this regard, the editors dedicate this text to healthcare providers who strive on a daily basis to treat cardiovascular emergencies, preventing or reversing cardiac arrest when possible, optimizing recovery and functional disability, and supporting the patient and family humanely when death is imminent.

Acknowledgments

The original framework for this textbook was developed by the AHA ACLS Subcommittees over many years and expertly edited by Richard Cummins, MD, MPH, MSc, in its most recent version. This core was expanded by the editors and focused on evidence-based medicine. In this context, it now summarizes and includes the work of many too numerous to include here: the writing groups, task forces, contributors and editors of the contained guidelines, clinical policies, and statements referenced in the links or reproduced by the authors and editors. Their expertise, original work, and material are recognized and were invaluable to this work.

The leadership and foresight of the American Heart Association and the American College of Emergency Physicians are summarized in the Foreword. In particular the editors would like to recognize three individuals. Mary Ann McNeely, PhD, AHA Director of ECC Product Development during inception, had the insight and provided the foundation for the text, and Mr. Stephen Prudhomme, AHA Vice President, Healthcare Quality, directed and facilitated its execution and publication. Ms. Marta Foster, Director and Senior Editor, ACEP Educational and Professional Publications, was invaluable in coordinating the ACEP leadership and selection of the dedicated ACEP editors.

Special Contributors

Owing to the extensive evidence-based nature of the text, the complexity of the material, and the coordination of media involved, many additional people were instrumental in executing the final version of this text. We would like to especially recognize Kara Robinson, ECC Publications Editor, for her writing, editing, and coordination of graphics; Julie A. Linick, ELS, for her writing and editing; and Erik Soderberg, Senior Product Manager, AHA ACLS Products, for his coordination and editing of audiovisual material.

Note on Drugs and Medication Dosing

Emergency cardiovascular care is a dynamic and fluid topic. At the time of this publication, work is in progress for the evidence evaluation leading to the publication of updated guidelines in many areas discussed in this text. Every effort has been made to ensure the correct and most accurate dosing of ECC medications. In this effort, we would like to recognize the detailed review and contribution of Anthony Busti, PharmD, BCPS. However, readers are advised to check carefully for changes in drugs, dosing and indications.

Contents

Part

One

Acute Coronary Syndromes

Chapter 1
Pathophysiology and Initial Triage of Acute Coronary Syndromes

John M. Field

In patients with symptoms of possible ACS, some of the traditional risk factors for CAD (e.g., hypertension, hypercholesterolemia, cigarette smoking, family history) are only weakly predictive of acute ischemia. Therefore, the presence or absence of these traditional risk factors ordinarily should not be used to determine whether an individual should be admitted or treated for ACS. Patients who present with ischemic symptoms should undergo early risk stratification using a risk-stratification model.[1,2]

- A Disrupted Plaque as the Proximate Cause of ACS
- Usually a Nonocclusive Plaque in the Infarct-Related Artery
- Triage Priorities for ACS—Not Only the Traditional Risk Factors
- A Systematic Approach—Key to Selecting a Strategy

Introduction to Acute Coronary Syndromes

An acute coronary syndrome (ACS) due to a disrupted plaque is present in the majority of patients with adult cardiac arrest.[3,4] Alarmingly, the first prolonged episode of ischemic discomfort has a 34% fatality rate. More than half the patients with sudden cardiac death (SCD) have no prior symptoms; in 17%, SCD is the first, last, and only symptom.[5–7] Additionally, the consequences of coronary atherosclerosis and ACS are responsible for the epidemiologic pool of patients at risk for SCD and those with significant cardiovascular morbidity. A conservative estimate for the number of discharges from patients with ACS from hospitals from 2005 is 772,000.[7]

It is, therefore, appropriate for a textbook on emergency cardiovascular care to begin with a section on acute coronary syndromes. The following chapters by Drs. Nichol and

Mitchell, Hollander, Ornato, Pollock and Summers, Peacock, Dean, and Stahmer set the stage for the broad spectrum of patients who present with cardiovascular emergencies. Currently, there are approximately 15.8 million individuals in the United States with coronary artery disease (CAD). There will be at least 1.4 million new, recurrent, or silent episodes of ACS. About 325,000 of these patients will experience out-of-hospital or emergency department SCD.[7]

This introductory chapter provides a broad general overview of ACS, focusing on an understanding of the pathophysiology necessary to apply triage and risk-stratification principles. In the following chapters, the authors mentioned above review these principles in current detail for health care providers who are charged with managing patients with ACS during the first several hours after presentation.

Overview of the Acute Coronary Syndrome

Definition and Spectrum of Acute Coronary Syndromes

The formation and accumulation of lipid and oxidative byproducts in an arterial wall is called atherosclerosis. When this deposit involves the coronary arteries, it is called coronary atherosclerosis. This process is gradual (Fig. 1-1) and gives rise to no symptoms for the many years of pathologic progression.[8]

Almost all regional ACS are caused by disruption of an atherosclerotic plaque, either plaque rupture or plaque erosion. Many of these disruptions are subclinical events; but when symptoms occur, a spectrum of clinical syndromes can result in which the disrupted plaque is a common and proximate feature. These syndromes are unstable angina pectoris, non–ST-segment-elevation myocardial infarction (NSTEMI), and ST-segment-elevation MI (STEMI). SCD can occur with any of these syndromes (Fig. 1-2).

Stable and Unstable Plaques

Coronary atherosclerosis is a diffuse process with segmental lesions called *coronary plaques;* these gradually enlarge and extend, causing variable degrees of coronary artery luminal occlusion. Intravascular ultrasound of the coronary arteries has shown that the majority of the atheroma burden is subluminal and not visible by coronary angiography. Coronary arteries are usually closed about 70% (by angiography; 90% closed when viewed by a pathologist) before they cause symptoms and are considered for percutaneous coronary intervention or surgery[9] (Fig. 1-3, top).

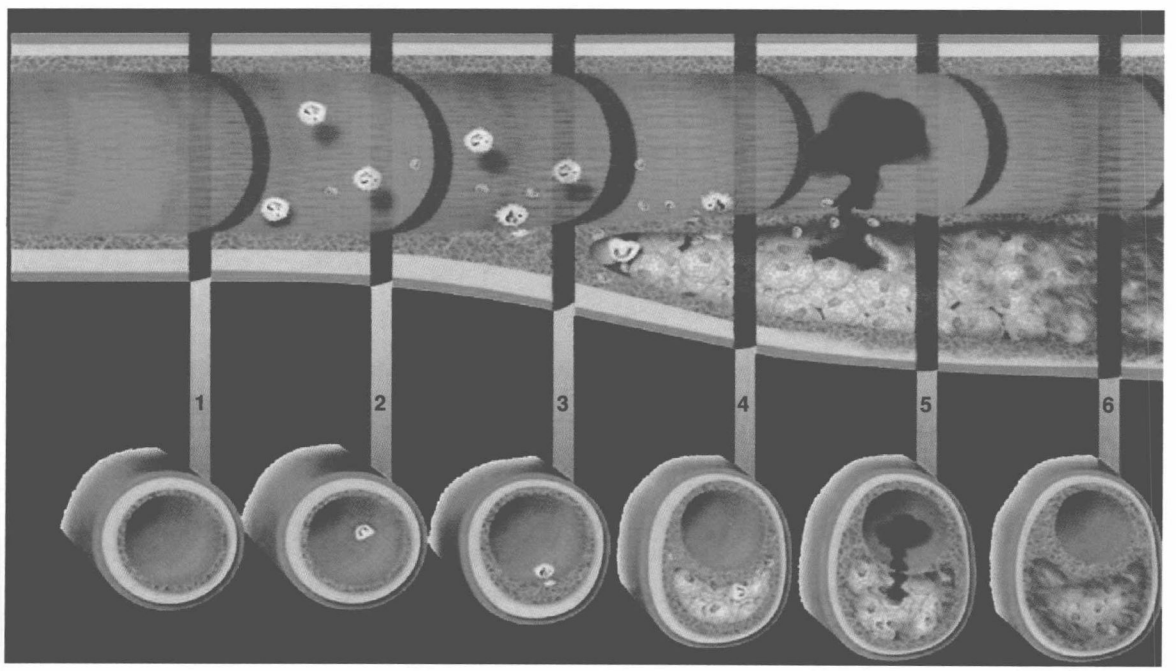

FIGURE 1-1 • Acute coronary syndromes (ACS). This figure illustrates the chronology of the progression of plaque formation and the onset and complications of STEMI, along with relevant management considerations at each stage. The longitudinal section of an artery depicts the "timeline" of atherogenesis from a normal artery (1) to (2) lesion initiation and accumulation of extracellular lipid in the intima, to (3) the evolution to the fibrofatty stage, to (4) lesion progression with procoagulant expression and weakening of the fibrous cap. An ACS develops when the vulnerable or high-risk plaque undergoes disruption of the fibrous cap (5); disruption of the plaque is the stimulus for thrombogenesis. Thrombus resorption may be followed by collagen accumulation and the growth of smooth muscle cells (6). (Modified from Libby P. Current concepts of the pathogenesis of the acute coronary syndromes. *Circulation* 2001;104[3]:365–372, with permission.)

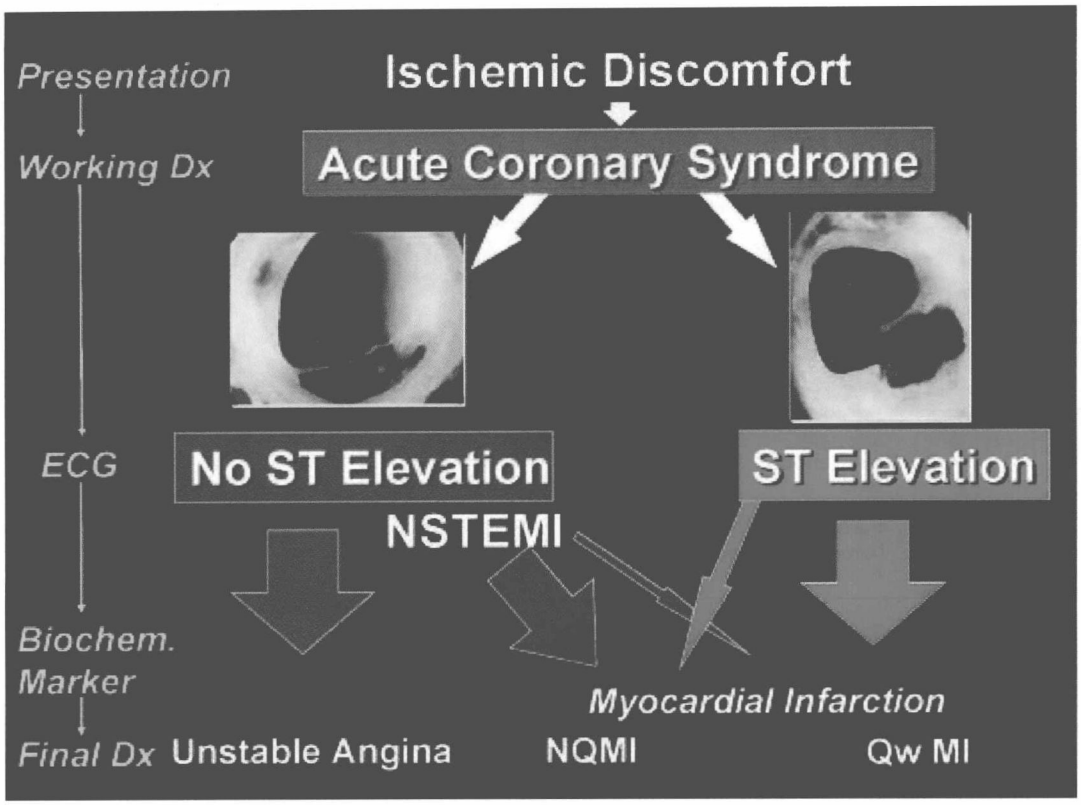

FIGURE 1-2 • Following disruption of a vulnerable or high-risk plaque, patients experience ischemic discomfort resulting from a reduction of flow through the affected epicardial coronary artery. This flow reduction may be caused by a completely occlusive thrombus (bottom half, right side) or subtotally occlusive thrombus (bottom half, left side). Patients with ischemic discomfort may present with or without ST-segment elevation on the ECG. Of patients with ST-segment elevation, most (large red arrow in bottom panel) ultimately develop a Q-wave MI (QwMI), while a few (small red arrow) develop a non–Q-wave MI (NQMI). Patients who present without ST-segment elevation are suffering from either unstable angina (UA) or a non–ST-segment elevation MI (NSTEMI) (large open arrows), a distinction that is ultimately made on the presence or absence of a serum cardiac marker such as CK-MB or a cardiac troponin detected in the blood. Most patients presenting with NSTEMI ultimately develop a NQMI on the ECG; a few may develop a QwMI. The spectrum of clinical presentations ranging from UA through NSTEMI and STEMI are referred to as the ACSs. (Modified from Libby P. Current concepts of the pathogenesis of the acute coronary syndromes. *Circulation* 2001;104[3]:365–372; Hamm CW, Bertrand M, Braunwald E. Acute coronary syndrome without ST elevation: implementation of new guidelines. *Lancet* 2001;358: 1533–1538; and Davies MJ. The pathophysiology of acute coronary syndromes. *Heart* 2000;83:361–366.)

Most plaques do not cause symptoms and are nonocclusive. But nonocclusive plaques are the ones most prone to cause ACS (Fig. 1-3, bottom). They have little if any hemodynamic effect before rupture, and stress testing and angiography cannot predict which ones will rupture and cause an ACS. Plaques can be classified as *stable* or *vulnerable* on the basis of their lipid content, the thickness of the cap that covers and separates them from the arterial lumen, and the degree of inflammation in the plaque itself.

1. A **"stable"** intracoronary plaque (Fig. 1-4A) has a lipid core that is separated from the arterial lumen by a thick fibrous cap. Stable plaques have less lipid, and the thick cap makes them resistant to fissuring and the formation of thrombi. Over time, the lumen of the vessel becomes progressively narrower, leading to flow limitations, supply-demand imbalance, and exertional angina. Stable plaques may progress to complete occlusion but do not usually cause STEMI because of the development over time of collateral supply to the myocardium at risk, thus preventing or limiting MI.
2. A **"vulnerable"** intracoronary plaque (Fig. 1-4B) has a lipid-rich core combined with an active inflammatory process that makes the plaque soft and prone to rupture. These plaques rarely restrict blood flow enough to cause clinical angina, and functional studies (e.g., stress tests) often yield negative results. Imaging techniques such as cardiac CT and MRI are being investigated as tools to identify unstable and inflamed plaques and may prove to be helpful in the future.
3. Inflammation is often found in the plaque. Inflammatory processes are concentrated in the leading edge affected by coronary blood flow. It is here that most plaque ruptures occur. A plaque that is inflamed and prone to rupture is called **unstable** (Fig. 1-4C).

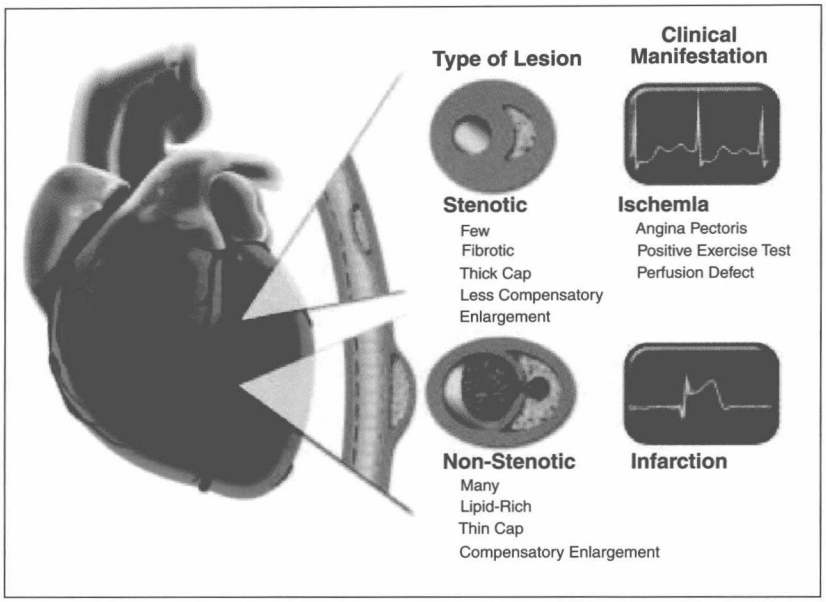

FIGURE 1-3 • Clinical manifestations of CAD in relation to degree of stenosis. Stenotic lesions tend to have smaller lipid cores, more fibrosis, and calcification as well as thick fibrous caps and less compensatory enlargement (positive remodeling). They typically produce exertional ischemia when, with exercise or emotion, demand for coronary blood flow exceeds supply. Nonstenotic lesions generally outnumber stenotic plaques and tend to have large lipid cores and thin fibrous caps susceptible to rupture and thrombosis. They often undergo substantial compensatory enlargement leading to underestimation of lesion size by angiography. Nonstenotic plaques may cause no symptoms for many years; when disrupted, however, they can provoke an episode of unstable angina or MI. (From Libby P, Theroux P. Pathophysiology of coronary artery disease. *Circulation* 2005;111[25]:3481–3488, with permission.)

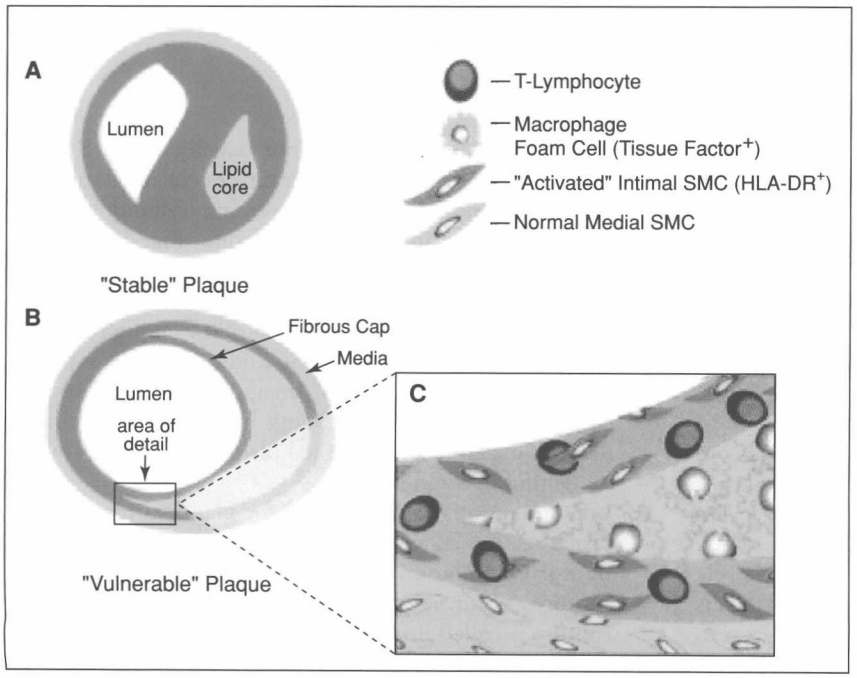

FIGURE 1-4 • Stable and vulnerable plaques. A. Stable plaque. B. Vulnerable plaque. C. Area of detail of vulnerable plaque showing infiltration of inflammatory cells. SMC, smooth muscle cell. (From Libby P. Molecular bases of the acute coronary syndromes. *Circulation* 1995;91:2844–2850, with permission. ©1995 American Heart Association.)