

# **PRACTICAL CARDIOLOGY**

**William J. French, M.D. and J. Michael C. Iley, M.D.  
editors**



# PRACTICAL CARDIOLOGY

## Ischemic and Valvular Heart Disease



Edited by  
**William J. French, M.D.**  
**J. Michael Criley, M.D.**

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W.J.F.  
J.M.C.

## Preface

Physicians are frequently confronted by the many clinical manifestations of ischemic and valvular heart disease in their patients. The clinician must choose from a wealth of diagnostic methods to assess the patient's problem and select the most appropriate therapy. However, the burgeoning amount of information available makes it difficult to assimilate and keep in perspective the changes taking place in medical practice, while still providing the best care.

*Practical Cardiology: Ischemic and Valvular Heart Disease* evolved from a continuing medical education course sponsored by UCLA School of Medicine and Harbor-UCLA Medical Center; it was written mainly for the primary care physician. It has been the aim of the authors and editors that this book be both practical and up-to-date in its approach to the recognition, assessment, and management of patients with ischemic and valvular heart disease, while providing a physiologic foundation on which clinical decisions can be based.

Their aim has been to highlight the state of the art in the care of patients with various ischemic and valvular heart problems.

The editors would like to thank the authors who gave their time and knowledge to this project. We would also like to thank Michael Davies, formerly with Houghton Mifflin Co., Linda Olt, UCLA coordinator of this series of books, the many secondary and primary reviewers, the artistic efforts of Deanna Hockett and Maggie Chacon, and, especially, Ginger Braden and Ann Frick, for their secretarial expertise. Without the concerted efforts of these people, this book would not have been possible.

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There has been a need to highlight the state of the art in the care of patients with ischemic and valvular heart problems.

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PART I

ISCHEMIC  
HEART  
DISEASE

PART I

ISCHEMIC

HEART

DISEASE

# 1

KENNETH I. SHINE

PATHOGENESIS OF  
ATHEROSCLEROSIS

The fundamental lesion in atherosclerosis is the formation of atherosclerotic plaques in the arterial wall, including the heart. The process begins in the early stages of life, with the deposition of cholesterol and other lipids in the arterial wall. In the early stages, the process is reversible, but as the disease progresses, the plaques become more extensive and the process becomes irreversible. The plaques are composed of a core of lipid-rich material, surrounded by a fibrous cap. The fibrous cap is formed by the migration of smooth muscle cells from the media into the intima, where they proliferate and produce extracellular matrix. The plaques can rupture, leading to the formation of a thrombus, which can cause a heart attack or stroke. The process of atherosclerosis is a complex one, involving many factors, including genetics, diet, and lifestyle. The ultimate goal of research in this field is to develop effective therapies to prevent and treat atherosclerosis.

## Overview

The epidemic of ischemic heart disease in the United States continues to rage, though there have been signs during the last decade that it may be on the wane. In spite of this, diseases of the heart and blood vessels still kill almost one million Americans every year, more than all other causes of death combined. The challenge to prevent these diseases in an increasing proportion of our population, as well as to improve the therapy for those who develop its more advanced stages, still exists. In the overview of ischemic heart disease I would like to address the following categories: (1) progress in primary prevention and health enhancement; (2) current notions about sudden death and the pathophysiology of myocardial infarction; (3) the chronic management of angina pectoris; (4) the role of surgery in the management of patients with coronary artery disease. Finally, I would like to speculate regarding the potential implications of recent developments in the diagnostic area including the role of positron imaging as a potential tool for what one might describe as "intermediate prevention."



## **PATHOGENESIS OF ATHEROSCLEROSIS**

The fundamental lesion in diseases of the blood vessels and heart is that of atherosclerosis. Deposition of fatty material, including substantial amounts of cholesterol and saturated fats, begins in our society within the second decade of life. From animal models we know that this process in its early stages is likely to be reversible; however, when profound proliferation of smooth muscle and connective tissue develops, and when calcium deposition occurs on dystrophic or damaged tissue, the likelihood of reversibility is decreased. A great deal has been learned in recent years about the factors that predispose a person to the atherosclerotic process. Goldstein and Brown at Sothwestern and Fogelman and Edwards at UCLA have been able to define a series of cellular defects that are shared by fibroblasts and white cells as well as other tissues within the body. These genetically determined disorders prevent the cells in many individuals from regulating cholesterol levels satisfactorily. Thus far, specific therapies directed toward the cellular defects have not been developed but are among the promises of the near future.

## **LDL AND HDL CHOLESTEROL**

At the same time, transport functions of serum lipoproteins have provided some additional important clues. The low-density lipoprotein (LDL) fraction of these proteins appears to be responsible for the transport of lipid from the liver to distant organs. Elevations of this fraction are associated with increased rate of transport of lipids to the periphery, including the vascular smooth muscle and endothelium where it is deposited. Recent recognition that an increased level of high-density lipoprotein (HDL) cholesterol confers some protection against atherosclerosis and that this lipoprotein appears to play a crucial role in the transport of lipid from the periphery back to the liver suggests a crucial balance in the preservation of homeostasis. Some preliminary studies suggest that extended exercise of a conditioned type might indeed change the balance such that the ratio of HDL to LDL lipoprotein becomes more favorable. These preliminary studies remain to be confirmed, but,