CARDIAC EMERGENCY CARE

EDWARD K. CHUNG

SECOND EDITION

Cardiac Emergency Care

Edited by EDWARD K. CHUNG, M.D.

Professor of Medicine
Jefferson Medical College of
Thomas Jefferson University
and
Director of the Heart Station
Thomas Jefferson University Hospital
Philadelphia

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Cardiac Emergency Care

To My Wife, Lisa,
and to My Children,
Linda and Christopher

PREFACE TO THE FIRST EDITION

It is not the purpose of this book to discuss in depth various subjects in medicine or to describe in detail all possible emergency medical events. The primary intention is to describe common cardiac emergencies which are frequently encountered in our daily practice.

The contents are intended to be clinical, concise, and practical. It is hoped that this book will provide all physicians with up-to-date materials that will assist them directly in the daily care of their patients with cardiac emergency problems.

The book will be particularly valuable to house staff, cardiology fellows, practicing internists, cardiologists, family physicians, and emergency room physicians, as well as coronary care unit nurses. In addition, medical students will derive great benefit from reading this book in learning a general approach to various cardiac emergencies.

I am grateful to all authors for their valuable contributions to *Cardiac Emergency Care*. I also wish to thank my personal secretary, Miss Theresa McAnally, for her devoted and cheerful secretarial assistance. She has been most helpful in handling correspondence with the contributors, as well as in typing several chapters of mine for this book. It has been my pleasure to work with the staff of Lea & Febiger Publishers. In particular, I would like to express my thanks to Mr. R. Kenneth Bussy, Executive Editor, for his indispensable assistance.

Philadelphia EDWARD K. CHUNG, M.D.

PREFACE

Since 1975, when the first edition of this book was published, there have been significant changes in the therapeutic approach to cardiac emergencies. The best example of those changes is that at present the most common indication for artificial cardiac pacing is the sick sinus syndrome and the brady-tachyarrhythmia syndrome—*not* the Adams-Stokes syndrome due to complete AV block. Thus a new chapter, "The Sick Sinus Syndrome and the Brady-Tachyarrhythmia Syndrome," has been added in this edition.

Another good example of the changes is that the nursing aspects of cardiac emergency care have become extremely important. Thus in-depth knowledge of cardiac emergency care is now an essential part of the training of nurses who deal with cardiac patients, especially in the intensive coronary care and the intermediate coronary care units and in connection with the rehabilitation programs for the various cardiac conditions. Consequently, another chapter, "The Nursing Aspect of Cardiac Emergency Care," has also been added in this edition.

The chapter "Radiologic Diagnosis in Cardiopulmonary Emergencies" has been added because the roentgenographic recognition of various cardiac emergencies is invaluable for cardiac emergency care.

The whole text has been revised considerably, although the basic aims and designs of the book are essentially unchanged. The unique feature of

the book is its practical approach, but the book format has been changed somewhat—to a cookbook style—so that busy primary physicians and house officers may be able to use the book as a quick reference source.

Many illustrative ECGs have been replaced, and many new ones have been added. In addition, clinically pertinent tables have been included.

The secretarial and the editorial burdens were borne cheerfully by Miss Theresa McAnally, my secretary and editorial assistant. Her able assistance and effort have been most valuable in the completion of the book. The endless cooperation of the staff of Lea & Febiger—and particularly of Mr. R. Kenneth Bussy, Executive Editor—is greatly appreciated.

Lake Naomi, Mount Pocono, Pa. EDWARD K. CHUNG, M.D.

CONTRIBUTORS

A.A.J. Adgey, M.D., F.R.C.P., F.A.C.C.
Cardiologist, Department of Cardiology
Royal Victoria Hospital
Belfast, Northern Ireland

Theodore L. Biddle, M.D., F.A.C.P., F.A.C.C.
Associate Professor of Medicine
University of Rochester
School of Medicine and Dentistry
Director, Coronary Care Unit
Strong Memorial Hospital
Rochester, New York

Albert N. Brest, M.D., F.A.C.P., F.A.C.C.
James C. Wilson Professor of Medicine
Director, Division of Cardiology
Thomas Jefferson University
Philadelphia, Pennsylvania

Stanley K. Brockman, M.D., F.A.C.S., F.A.C.C.
Professor of Surgery and
Director, Division of Cardiothoracic Surgery
Thomas Jefferson University
Philadelphia, Pennsylvania

Agustin Castellanos, M.D., F.A.C.C.
Professor of Medicine
University of Miami
School of Medicine
Miami, Florida

Edward K. Chung, M.D., F.A.C.P., F.A.C.C.
Professor of Medicine
Jefferson Medical College of
Thomas Jefferson University
Director of the Heart Station
Thomas Jefferson University Hospital
Philadelphia, Pennsylvania

Lisa S. Chung, M.D.
Chief Medical Officer and Medical Director
U.S. Public Health Service
Philadelphia, Pennsylvania

Jay N. Cohn, M.D., F.A.C.P., F.A.C.C.
Professor of Medicine
Head, Cardiovascular Section
University of Minnesota School of Medicine
Minneapolis, Minnesota

Edward Genton, M.D., F.A.C.P., F.A.C.C.
Professor of Medicine
Associate Dean for Health Services
McMaster University
Faculty of Health Sciences
Hamilton, Ontario, Canada

* William J. Grace, M.D., F.A.C.P., F.A.C.C.
Professor of Clinical Medicine
New York University–Bellevue Medical Center
Director, Department of Medicine
St. Vincent's Hospital
New York, New York

Michael J. Gullotti, M.D.
Fellow in Cardiology
Thomas Jefferson University
Philadelphia, Pennsylvania

* Deceased

George H. Khoury, M.D., F.A.A.P., F.A.C.C.
Professor of Pediatrics
Assistant Dean of Continuing Medical Education
University of Kansas
School of Medicine
Wichita, Kansas

Albert S. Klainer, M.D., F.A.C.P.
Professor of Medicine
Rutgers Medical School
Newark, New Jersey
Chairman, Department of Internal Medicine
Morristown Memorial Hospital
Morristown, New Jersey

Louis Lemberg, M.D., F.A.C.P., F.A.C.C.
Professor of Clinical Cardiology
University of Miami School of Medicine
Miami, Florida

J. F. Pantridge, M.D., F.R.C.P., F.A.C.C.
Professor of Cardiology
The Queen's University of Belfast
Belfast, Northern Ireland

Leon Resnekov, M.D., F.R.C.P., F.A.C.C.
Professor of Medicine
Joint Director, Section of Cardiology
University of Chicago School of Medicine
Chicago, Illinois

Martha I. Spence, R.N., M.N., C.C.R.N.
Instructor, University of Miami School of Nursing
Patient Educator Instructor
Staff Nurse
Baptist Hospital
Miami, Florida

David H. Spodick, M.D., F.A.C.P., F.A.C.C.
Professor of Cardiovascular Medicine
University of Massachusetts School of Medicine
Director, Division of Cardiology
St. Vincent's Hospital
Worcester, Mass.

Robert M. Steiner, M.D., F.A.C.R.
Associate Professor of Radiology
Director, Cardiopulmonary Radiology
Jefferson Medical College of
Thomas Jefferson University
Philadelphia, Pennsylvania

Paul Walinsky, M.D.
Associate Professor of Medicine
Co-director, Coronary Care Unit
Thomas Jefferson University
Philadelphia, Pennsylvania

Leslie Wiener, M.D., F.A.C.P., F.A.C.C.
Professor of Medicine
Thomas Jefferson University
Philadelphia, Pennsylvania

Paul N. Yu, M.D., F.A.C.P., F.A.C.C.
Sarah McCort Ward Professor of Medicine
University of Rochester
School of Medicine and Dentistry
Head, Cardiology Unit
Strong Memorial Hospital
Rochester, New York

CONTENTS

Chapter 1

ACUTE PULMONARY EDEMA*

THEODORE L. BIDDLE
PAUL N. YU

GENERAL CONSIDERATIONS

Acute pulmonary edema is a common medical emergency that demands prompt and effective treatment. In this chapter pulmonary edema of both cardiac and noncardiac origin is discussed, with particular emphasis on a practical overview for clinical management.

Four anatomic compartments of the pulmonary circuit have been delineated in an effort to improve our understanding of the pathophysiology of heart failure.[1] The *vascular compartment* consists of the pulmonary arteries, capillaries, and veins that participate in fluid exchange with the interstitital tissue of the lung. The *alveolar compartment* comprises the alveoli, whose walls are made up of epithelial cells with a lipoprotein layer called surfactant. That layer coats the inner alveolar surface and exerts an "anti-atelectasis effect," stabilizing the alveoli and preventing their collapse under conditions of low alveolar volume. The *interstitial space* is interposed between the small pulmonary vessels and the alveoli, and it also contains small lymphatics and conducting airways. The *lymphatic space* is the extensive network of pulmonary lymphatics that

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drain excess fluid from the alveolar and interstitial compartments. Endothelial cells of the pulmonary capillaries, alveoli, and lymphatics and other cell types have been discussed extensively elsewhere.[1-3] Their specific function, although of great importance, is not described in this review.

Two important cell-mediated responses in pulmonary edema involve the generation of early symptoms and late pathologic changes. The J receptors in the interstitial space act as stretch receptors stimulated by increases in interstitial pressure or fluid resulting in the characteristic tachypnea of acute congestive heart failure.[4] Also, edema fluid promotes the formation of collagen, reticulum, and elastic fibers in the interstitial space. That phenomenon may lead to the interstitial fibrosis common in people with chronic elevation of the pulmonary capillary pressure.

Traditionally, the study of the pathophysiologic aspects of pulmonary edema has been concerned with (1) increased hydrostatic pressure or (2) increased permeability of the alveolar-capillary "membrane." But those factors cannot account for every illness in the wide spectrum of illnesses characterized by pulmonary edema. In general, one or more of four factors are responsible for the production of acute pulmonary edema: (1) elevation of the pulmonary capillary pressure, (2) damage to the pulmonary capillary "membrane," (3) decrease in the plasma osmotic pressure,

Table 1–1. *Etiology of Acute Pulmonary Edema*

Cardiac causes
Left ventricular failure
Myocardial infarction
Acute decompensation of chronic left ventricular failure—aortic, hypertensive, or cardiomyopathic
Mitral valve disease
Volume overload
Noncardiac causes
Altered permeability of pulmonary capillary membrane
Inhalation of toxic agents
Adult respiratory distress syndrome
Bacteremic sepsis
Uremia
Radiation pneumonitis
Disseminated intravascular coagulation
Decreased plasma oncotic pressure—hypoalbuminemia
Lymphatic obstruction
Uncertain etiology
High-altitude pulmonary edema
Heroin overdose
Pulmonary embolism
Neurogenic causes
Postanesthesia

and (4) impairment of the lymphatic drainage. Our etiologic approach considers both cardiac and noncardiac forms of pulmonary edema (Table 1–1). The cardiac forms are discussed extensively in this chapter. And since a broad knowledge of the many noncardiac forms of pulmonary edema is also necessary for intelligent management, those forms are also considered.

ETIOLOGY OF ACUTE PULMONARY EDEMA

The most common cause of acute pulmonary edema is cardiac disease, whether atherosclerotic, valvular, hypertensive or myopathic in origin. (Pericardial disease is discussed in Chapter 14.) Acute or chronic myocardial "failure" occurs when the left ventricle is unable to eject the normal stroke volume. Thus the diastolic pressure in the left ventricle rises, causing an elevation of the left atrial and pulmonary venous pressures. Normally, the plasma oncotic pressure prevents a substantial diffusion of intravascular fluid across the normal capillary membrane to the interstitial space. With increasing hydrostatic pressure, however, interstitial and intra-alveolar edema may occur. We have measured the amount of lung water in patients with acute myocardial infarction complicated by heart failure.[5,6] The lung water measured by a double isotope technique increased according to the increasing severity of pulmonary congestion as determined by both clinical and radiographic criteria (Figs. 1–1 and 1–2). A significant correlation was found between the pulmonary capillary (wedge) pressure and lung water. In all patients with acute pulmonary edema, the pulmonary wedge pressure was elevated. With clinical improvement, both the pulmonary wedge pressure and the lung water decreased. Increasing arterial hypoxemia has been correlated with more severe pulmonary edema and elevation of lung water.[6]

In patients with left ventricular failure secondary to aortic valve disease, hypertensive cardiovascular disease, or cardiomyopathy, the elevation of the left ventricular end-diastolic pressure eventually leads to an increase in pulmonary capillary pressure and transudation of fluid into the interstitial or alveolar compartments of the lungs. Patients with rheumatic mitral stenosis develop an elevation of the left atrial and then of the pulmonary capillary pressure. The magnitude of mitral valvular obstruction and the increase in hydrostatic force determine in the main the severity of the pulmonary congestion.

Volume overload from excessive intravenous fluid therapy may precipitate acute pulmonary edema but usually only in patients with preexisting myocardial dysfunction or severe valvular disease.

Noncardiac types of pulmonary edema are usually related to alterations