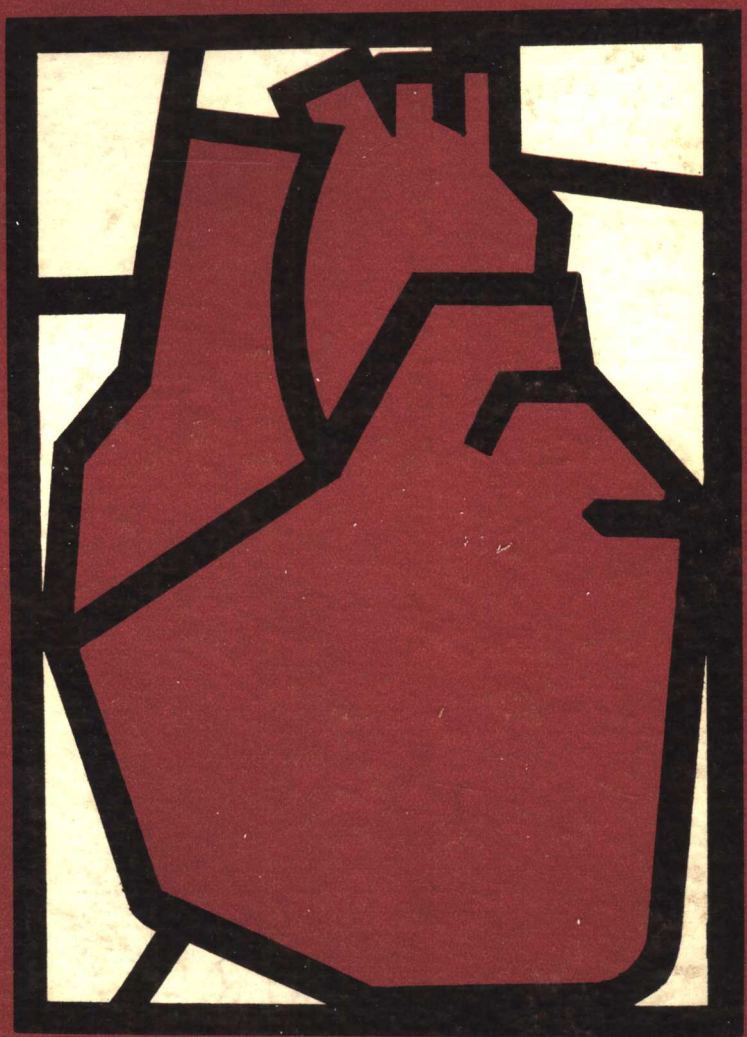
CARDIOLOGY



Cardiology:
a clinicophysiologic approach

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Dedication

This book, based on the practice of cardiology and cardiac surgery at Saint Vincent's Hospital and Medical Center is gratefully dedicated to the memory of

SISTER ANTHONY MARIE FITZMAURICE

Assistant Administrator from 1947 to 1961, and Executive Director of the Hospital until her death on November 17, 1970. She understood the need for the development of specialized areas of medical practice, emphasized the value of interdisciplinary approaches, and constantly encouraged the authors in their desire to improve patient care by blending the Art and Science of Medicine.

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Preface

The dramatic increase in deaths from heart disease which has marked the Twentieth Century has been paralleled by the growth and development of cardiology as a specialty. Almost every physician must be trained in some aspects of cardiology in order to deal with the large numbers of patients who present manifestations suggesting heart disease. At the same time, the technologic revolution has so invaded cardiology that many physicians shrink from seriously reviewing modern cardiologic concepts for fear of inundation by mathematical formulae and engineering jargon.

This book has been prepared for the internist without special training in cardiology, the general practitioner, the medical student or nurse interested in cardiology. We have attempted to blend basic physiology with clinical information in order to develop an orderly approach to clinical care, and our goal has been to present a workable framework enabling the reader to continually reevaluate his treatment by constant observation of his own efforts and by critical assessment of the growing literature in the field of heart disease.

All contributors have been associated with Saint Vincent's Hospital and Medical Center, and the patients described in this book were studied in the Cardiopulmonary Laboratory during the period 1963-1970. This period was one of great change for the catheterization laboratory as physiology left the ivory tower and joined the clinician at the bedside. This book is, in a sense, a personal account of the development of a program of physiologic cardiology.

The authors are deeply indebted to Dr. William J. Grace, Director of Medicine, whose imagination, vision, and direction have been responsible for the development of the Cardiopulmonary Laboratory at Saint Vincent's Hospital. We also thank Dr. Louis Rousselot, formerly Director of Surgery at Saint Vincent's and presently Assistant Secretary of Defense for Medical Manpower, for his parallel stewardship in the development of the section of Cardiac Surgery. Special thanks are directed towards Drs. Richard Kennedy, Albert Vitale, Matthew Ferguson, Philip Brickner and our many other colleagues who have graciously made their private patients available for study. Special thanks are due Dr. James Mazzara, Assistant Director of the Cardiopulmonary Labora-

tory, for his enthusiastic support and for his skillful supervision of the Fellowship Program. Much of the material collected in this book is due to the labor of Drs. Frank Flood, Philip Varriale, John Penny, Jin Kim and William Hardy who served as fellows in the Cardiopulmonary Laboratory.

Many people associated with the Laboratory have directly assisted in the care of our patients and in the accumulation of hemodynamic and other physiologic data. We particularly thank Mrs. Judy Adcock, Mrs. Karen Korn, the Misses Monica Coyne, Antoinette Criscitiello, Jane Griesbach, Carolyn Jurasits, Betty Lambright, Zennete Marin, Jeniva Nuzzo, Mary Lou Oldfield, Felicity Reimold, Sally Solan, Carmen Rivera, Mary E. Stuart, Mr. Donald Armstrong, Mr. Dennis Cagney and Mr. Richard Quiroz.

A dedicated group of secretaries have been responsible for the tabulation of patient material and the preparation of this manuscript. We acknowledge the assistance of Mr. Robert Evans, Miss Romayne Bianchi, Miss Suzanne Howatt and Miss Ann Simpson. Massy Nakamura and Rudolph Henning contributed their expertise to the preparation of many illustrations.

S. Ayres
J. Gregory
M. Buehler

Introduction

The growth of Cardiology as a specialty may be examined by a study of the attention given to acute myocardial infarction in several of the great textbooks of cardiology. Acute myocardial infarction is now known to occur at least one million times each year in the United States, but the frequent occurrence of this most common of heart diseases was not always appreciated. Although William Heberden described angina pectoris in 1768,[1] the clinical syndrome of acute myocardial infarction and the recognition of the relationship between coronary artery disease and cardiac pain were not accepted for another 150 years. Stokes in his *Diseases of the Heart and Aorta*,[2] published in 1854, summarized the then prevalent view when he wrote that coronary obstruction "is probably not infrequent; but as a cause of angina, its action is remote and its existence unnecessary." These sentiments were echoed some fifty years later by Sir James MacKenzie who wrote in his *Diseases of the Heart*[3] that angina pectoris could not be "directly due to diseases of the coronary artery" but was "an evidence of exhaustion of the function of contractility."

Pathologists on the continent, particularly Cohnheim, believed that the coronary arteries were end-arteries and that occlusion must always lead to sudden death. This view was accepted by most clinicians and Broadbent, for example, in his *Heart Disease*,[4] wrote that there "are no characteristic physical signs or symptoms by which thrombosis of the coronary vessels can be diagnosed." The great American physician, Sir William Osler, perpetuated this belief by writing in his *Modern Medicine*,[5] published in 1908, that "the symptoms of obstruction of the coronary vessels are not very characteristic, and it is only rarely that the lesion can be diagnosed during life." Osler did, however, correctly understand the relationship between coronary artery disease and angina pectoris. Reviewing the con-

troversy over the causes of angina pectoris he concluded that the condition was "associated with an impairment of the blood irrigation of the myocardium."

The syndrome of acute myocardial infarction was first clearly described in 1912 by James Herrick who emphasized that "there are reasons for believing that even large branches of the coronary arteries may be occluded—at times even acutely occluded—without resulting death" in an important contribution entitled *Clinical Features of Sudden Occlusion of the Coronary Arteries.*[6] His concepts were rapidly accepted, and Henry Christian in his *The Diagnosis and Treatment of Diseases of the Heart*[7] (1930) and Sir Thomas Lewis[8] in his text of similar title (1937) included extensive sections on the recognition and treatment of coronary thrombosis. One of the final pages in this tortous pathway to an understanding of the nature of coronary artery disease was the important paper of Blumgart, Schlesinger and Zoll, *Angina Pectoris, Coronary Failure and Acute Myocardial Infarction.*[9] These authors performed postmortem coronary artery injections, made clinical-pathologic correlations, and emphasized the central role of coronary artery obstruction.

While it took 200 years to correctly understand coronary artery disease, almost all of our present therapeutic methods have developed in the last 10 years. Kouwenhoven, Jude and Knickerbocker[10] described closed-chest resuscitation in 1960, Lown, Amarasingham and Neuman[11] developed direct current counter-shock for terminating arrhythmias in 1962, and Brown,[12] Day,[13] Meltzer,[14] and Grace[15] began developing coronary care units for continuous electrocardiographic monitoring in 1963. The past five years have seen an emphasis on "pump failure" with the treatment of such failure by physiologic techniques. Most recently, the importance of the first twenty four hours following acute myocardial infarction has been emphasized. Early identification and treatment of the "pre-shock" phase has been emphasized and effective therapy facilitated by the development of mobile coronary care units. Underlying all of this, however, has been the realization that the treatment of established coronary artery disease is at best a rear guard action and that practical control of this major public health problem is only possible by prevention. At this writing, dietary manipulation, sensible programs of exercise, and shifts in life style and achievement goals appear to be hopeful shafts of light in the generally dark shadow of coronary artery disease.

And what of the busy practitioner faced daily with an unending parade of sick people? Has this complicated collection of new information been digested, integrated, filtered and passed on in the form of better patient care? Can the modern information explosion be harnessed and used to construct a practical and meaningful system of physiologic diagnosis and treatment?

This book attempts to stress the *deductive* approach to diagnosis and the empiric approach to treatment. The fundamental body of cardiologic knowledge is composed of clinical, hemodynamic, electrocardiographic, and radiographic knowledge. Frequently, however, the practitioner of each discipline works in a vacuum created by time-worn institutional tradition. The electrocardiographer tells all—even age and sex—from a graph of electrical activity; the radiologist extrapolates from a world of blacks and whites, the clinician enshrines his eyes, hands, and ears as the true key to all worthwhile knowledge. As long as 100 years

ago, Leyden warned his associates not to become so fascinated by the newly developed techniques of auscultation and percussion that they would forget all previous methods of observation. Coronary artery disease was generally unappreciated in part because the great clinicians of that day believed that disease could not exist in the absence of physical signs.

Conclusions and approaches to therapy must be based on the dispassionate collection of all observations rather than on the application of theoretical systems of knowledge. An atrial gallop, prominent apex cardiographic A waves, systolic murmur, and small vigorously contracting heart lead to the probability of hypertrophic subaortic stenosis—not because a noted authority has listed these as evidence of that disease—but because the series of observations point to the existence of a noncompliant muscle-bound ventricle with outflow obstruction.

It is no coincidence that the greatest detective of them all—Sherlock Holmes of Baker Street—was created by a physician, Arthur Conan Doyle—and was modeled after Dr. Joseph Bell, an Edinburgh physician who constantly amazed his colleagues with his powers of observation and deduction. Listen to the great detective extolling the virtues of the science of deduction. "From a drop of water, a logician could infer the possibility of an Atlantic or a Niagara without having seen or heard of one or the other." And so must the physician deduce the possibility of internal malfunction from the logical interpretation of external manifestation. We hope this book will assist the physician in the transformation of hemodynamic, radiographic, electrical and clinical observations into accurate diagnosis and effective treatment.

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Contents

1 Manifestations of Cardiac Disease —Symptoms

Most skillful clinicians acknowledge that a careful history, properly interpreted, will generally reveal the correct diagnosis, frequently even before a physical examination is made. This is particularly true in diseases of the cardiovascular system.

Dyspnea is probably the most common symptom in cardiac disease. The term implies difficult or uncomfortable breathing. Normal individuals will experience dyspnea at high levels of exertion. Dyspnea only becomes significant when it occurs at levels below those expected to be tolerated. Since the absolute level may be difficult to interpret, a record of serial changes over a period of time is extremely valuable. The historian should review the number of flights of stairs climbed one year ago without dyspnea compared to the number climbed at the present time. It is useful to relate questions to routine activity, such as shopping, carrying packages, making beds, mowing the lawn, walking to work, and so forth. The differential diagnosis of pulmonary and cardiac dyspnea is presented in Table 1.

A number of physiologic studies have demonstrated that dyspnea is best correlated with either the amount of intrathoracic pressure (or force) necessary to produce a given tidal volume or the total work required to produce that ventilation (work equals pressure times volume integrated over the tidal volume). Any pathologic process that increases pulmonary venous pressure will produce pulmonary vascular congestion, make the lung less distensible (less compliant), and increase the work of breathing. The two major causes of increased pulmonary venous pressure are mitral valvular obstruction and left ventricular failure from any cause. Note that dyspnea in mitral stenosis is caused by valvular obstruction, not by heart failure.

Orthopnea, a common symptom of pulmonary congestion, implies dyspnea when recumbent but not when upright. Characteristically, the patient states he

requires two to three pillows under his head in order to sleep comfortably. Of great importance is the report that he awakens with dyspnea if his head falls off the pillows. Physiologic studies have demonstrated a decreased pulmonary compliance in patients with mitral stenosis lying supine compared with sitting upright. This probably results from the fact that almost all of the pulmonary vessels are below the level of the heart in the supine position, increasing the hydrostatic pressure in the pulmonary veins.

Paroxysmal nocturnal dyspnea may be an early and only symptom of heart disease. The patient may suddenly awaken with a choking sensation and feel as if he is about to strangle. He is terrified, sweaty, and pale. Standing up or sitting in a chair almost immediately relieves the sudden shortness of breath. Many patients rush to the window and experience relief as soon as the window is opened. The symptom is probably due to a redistribution of blood volume from the periphery to the lungs during sleep and recumbency.

TABLE 1

Differential Diagnosis of Cardiac and Pulmonary Dyspnea

If several positive findings are present from both columns, the patient probably has both heart and lung disease.

	Cardiac	*Pulmonary*
History	Exertional dyspnea, orthopnea, true paroxysmal nocturnal dyspnea, ankle swelling, angina pectoris	Usually exertional dyspnea only, chronic cough, long history of being "bronchial," frequent URIs or pneumonias
Electrocardiogram	Definite abnormalities or completely normal, notched P waves may be seen, evidence of old myocardial infarction	Right axis deviation, peaked P waves or completely normal
Physical Examination	Heart sounds prominent, atrial or ventricular gallops present, systolic murmurs present	Heart sounds frequently obscured, sounds best heard below xiphoid
	Good diaphragmatic excursion and normal transmission of breath sounds, basilar coarse or crepitant rales	Poor diaphragmatic excursion and poorly transmitted breath sounds, wheezing on forced expiration or at rest
Chest X-ray	Cardiac enlargement, pulmonary congestion	Normal or small heart, overinflated lungs
Pulmonary Function Test	Vital capacity and maximum breathing capacity moderately reduced in parallel fashion, normal expiratory flow rates	Maximum breathing capacity and expiratory flow rates markedly reduced, vital capacity may be normal or slightly reduced
Response to Diuretic	Decrease in dyspnea and amelioration of rales, loss of weight > 3 pounds	No effect
Response to Nebulized Bronchodilator	No definite effect	Improvement