BOOK REVIEWS


As stated in the foreword of this book, the morphology of ventricular complexes has been well studied through the years, but information concerning the atrial signal has been rather limited. The authors have served a useful purpose by summarizing the literature and presenting their own experience in this short book.

The nine chapters of the book deal with atrial activation and repolarization, the normal P wave and P-wave changes in dextrocardia, pulmonary disease, atrial infarction, atrial overloading, and other conditions as well as atrial arrhythmias. The reproduction of the illustrations is excellent and the bibliography is adequate.

It is rather disappointing, however, that the discussion of atrial arrhythmias is quite superficial. The subject of the intra-atrial electrogram has not been mentioned. Some of the illustrations (for example, figs. 37, 45, 58B, 56C, and 90) fail to demonstrate clearly the specific points the authors intend to emphasize.

This volume should be of value to all physicians interested in electrocardiography, but they may find that in some areas it is lacking in depth for such a specialized topic.

Te-Chuan Chou, M.D.


This book is the latest addition to the publisher’s series, Bibliotheca Cardiologica. It is a record of the Proceedings of the Ballistocardiograph Research Society held in the spring of 1967. The editor for this volume is W. R. Scarborough.

Sixteen short presentations are recorded. These deal directly or indirectly with most of the foremost problems challenging investigators in ballistocardiography (BCG). Among these are: What is the meaning of the age dependence of BCG waveform and amplitude? Is it possible to ascertain with acceptable accuracy the velocity of aortic ejection from the BCG? Can the difficulty of using the BCG to record the cardiovascular response to the stress of physical exercise created by waveform abnormality, be overcome? How accurate is prediction of the development of subsequent cardiovascular disease, based upon BCG waveform and amplitude? And finally, can the calculation of stroke volume from the BCG be made more accurate?

There is in addition a comprehensive review by the editor and DeWitt H. Smith of the accomplishments to date of those working in BCG and a prediction of the results of future investigation.

The titles and authors of all of the papers presented to The Society since its first annual meeting in 1956 are listed. Recorded as well are the names and addresses of current members of The Society and of the European counterpart society.

The book is highly recommended to those who employ BCG as well as to those who do not but are merely curious to know what ballistocardiographers think of their work.

Benjamin M. Baker, M.D.


An International Conference on Blood Flow Through Tissues in Glasgow in March, 1967, brought together from Britain, Europe, and North America the leading research workers in this field. Their contributions, as Dr. Arthur Mackey said in the “Foreword,” “span techniques, methodology, physiological studies of the control of blood flow, and investigations of disease.” This book, which is the Proceedings of that conference, can be best reviewed by listing the papers and their authors:

Session I. Blood Flow Through Vessels. W. A. Mackey, Chairman


Session II. Heart and Lungs. W. H. Bain, Chairman.

Radioangiography: Validity and Reproducibility. A. Uhrenholdt.


Session IV. Brain. W. Bryan Jennett, Chairman.


A Neurogenic Factor in the Control of the Cerebral Circulation. T. W. Langfitt, N. F. Kassell.

Clinical Applications of the Xenon-133 Inhalation Technique. N. Veall, B. L. Mallett, M. D. O'Brien.


Session V. Muscle and Skin. Neils A. Lassen, Chairman.

"Catecholamines in Cardiovascular Physiology and Disease" edited by Ralph Reader, Medical Director, National Heart Foundation of Australia, consists of the proceedings of a conference held in January 1967 in Canberra, Australia. The contributors, mostly from Australia, but also from Sweden, England, the U.S.A., and New Zealand, have presented sophisticated and sometimes highly technical reports which will appeal particularly to those involved in catecholamine and cardiovascular research. The following lists the authors and briefly mentions salient features in each paper:

U. S. von Euler expressed the belief that knowledge of noradrenaline-storing particles in adrenergic nerves is indispensable to understanding processes which underlie adrenergic transmission. He has demonstrated a high release rate in nerve granules, supplemented by a high synthetic rate, which offers a means of efficient neurotransmission and rapid action, in contrast to the adrenal medulla where release is slow, thereby protecting the organism from what might otherwise be an explosive release of catecholamines like that which may occur in pheochromocytoma. He suggested that no true analysis of the action of drugs on adrenergic function can be made without considering their action on noradrenaline storage granules. Because of complexities of drug actions, effects on uptake and release of catecholamines in isolated granules permit some insight into the mechanism of action of drugs at the granule level. He further noted enhancement of noradrenaline uptake in isolated granules caused by addition of ATP and magnesium and increased release of noradrenaline with increasing temperature or development of acidosis. These studies shed important light on the dynamics involved in catecholamine storage and release.

L. T. Potter reviewed characteristics and functions of small granular vesicles in postganglionic adrenergic nerves and chromaffin granules. He concluded that most noradrenaline in sympathetic nerves is stored in small granular vesicles in axon terminals; noradrenaline is synthesized from dopamine at the level of the vesicle membrane whereas noradrenaline synthesis catalyzed by tyrosine hydroxylase and decarboxylation of dopa appears to occur outside vesicles; a large amount of noradrenaline which is released is transmitted back into adrenergic nerves and can be bound in vesicles for re-use. The capacity of vesicles for noradrenaline appeared utilized to a high extent.
T. Malmfors presented elegant fluorescent histochemical studies on uptake, storage, and release of catecholamines. He concluded that adrenergic nerves have at least two different uptake-concentration mechanisms—one probably located at the cell membrane over the whole neuron and the other situated in storage granules which takes up, accumulates, and stores noradrenaline from inside adrenergic nerves.

G. Burnstock and P. M. Robinson reported electron microscopic studies supporting the concept that noradrenaline is bound in the small granules in autonomic nerves. They also demonstrated that many adrenergic axons are associated with low or moderate levels of acetylcholines- terase. However, existence of a cholinergic-link mechanism in some adrenergic function has not been established.

L. B. Geffen examined evidence that sympathetic nerves take up and re-use the transmitter they liberate. Re-uptake of noradrenaline can be viewed as an economy of the transmission process. He suggested that “under experimental conditions, overflow of noradrenaline into the circulation constitutes the most important avenue of loss of transmitter from the recapture cycle.”

U. S. von Euler and F. Lishajko revealed that tyramine in low concentration enhanced noradrenaline release and inhibited uptake from bovine nerve granules whereas tyramine in high concentrations inhibited noradrenaline release. They proposed that the “releasing” effect of tyramine on nerve granules is partly due to true release, presumably by substitution, and partly due to inhibition of re-uptake. Reserpine was found to inhibit ATP-dependent uptake of noradrenaline and to retard its release from granules.

M. E. Holman reported some electrophysiological aspects of transmission from noradrenergic nerves to smooth muscle and stated that excitatory actions of noradrenaline on some smooth muscle may be related to an increase in permeability (conductance) for sodium ions.

R. W. Ryall reviewed recent electrophysiological and pharmacological evidence bearing on the properties of preganglionic neurons in the spinal cord. Some nerve fibers that descend from the brain and end near preganglionic neurons contain noradrenaline or 5-hydroxytryptamine. Also, some preganglionic neurons are inhibited by noradrenaline but excited by 5-hydroxytryptamine. Whether these monoamines function as transmitter substances, released from descending pathways to act upon preganglionic neurons, is uncertain.

M. J. Rand and J. Wilson presented a paper suggesting that adrenergic neuron-blocking drugs may interfere with the noradrenaline-releasing action of acetylcholine.

G. A. Bentley and G. Smith reported effects of α-adrenergic receptor-blocking drugs on response of smooth muscle to sympathetic drugs and stimulation. They concluded that phentola-mine and phenoxybenzamine probably penetrate to the vicinity of sympathetic nerve endings; however, the reason why these drugs block response of some smooth muscle to noradrenaline whereas they only slightly block or actually increase response to electrical stimulation, is unclear.

L. Austin, B. G. Livett, and I. W. Chubb presented evidence that the complete synthetic mechanism for biosynthesis of noradrenaline is present in nerve terminals.

A. Spoerdsma reported that inhibition of catecholamine synthesis by α-methyl-para-tyrosine (α-MPT) was effective in controlling hypertension in patients with pheochromocytoma; however, since in patients with essential hypertension, lowering of blood pressure with α-MPT was not impressive, even though the synthesis of catecholamine was inhibited by more than 50%, he concluded that maintenance of hypertension in this condition could not be due to a subtle abnormality of catecholamine metabolism. Evidence was also presented that a component of the action of monoamine oxidase inhibitors and α-methyl dopa is a reduced rate of catecholamine biosynthesis.

R. Laverty and A. Robertson revealed that α-methyl tyrosine has both a catecholamine-depleting action and a considerable hypotensive action in hypertensive rats. However, there was no strict correlation between noradrenaline content in the brain or peripheral tissues and blood pressure decrease.

W. C. deGroat reported that ganglionic response to exogenous adrenaline and noradrenaline is both excitatory and inhibitory and is related to change in membrane potential of the ganglion cells. Two adrenergic sites appear to be involved: an α and β type, mediating catechola-mine-evoked depression and facilitation respectively. Whether the catecholamines play any physiological role in ganglionic transmission is unknown.

C. Roper studied receptors of catecholamines in skeletal muscle and demonstrated that sympathomimetic amines produced a prejunctional facilitatory effect, due to increased transmitter release, and also a post-junctional hyperpolarizing action which resulted in depression of transmission. In skeletal muscle no correlation between anti-adrenaline and anti fibrillary activity of β-receptor-blocking drugs was evident. On cardiac muscle specific β-receptor-blocking activity of these drugs plays a vital role in abolishing experimentally induced arrhythmias involving a
sympathetic component; however, their activity against arrhythmias that do not involve a sympathetic component may depend on a "quinidine-like" effect.

P. I. Korner, J. P. Chalmers, and S. W. White reported effects on the circulation caused by reflex stimulation of the sympatho-adrenal system by arterial hypoxia (via chemoreceptors) and primary tissue hypoxia (via baroreceptors). They indicated that difference in cardiovascular responses noted was due to a preponderance of sympathetic nerve activity over adrenal medullary hormone secretion during arterial hypoxia whereas in primary tissue hypoxia total sympatho-adrenal activity was smaller and activity of both sympatho-adrenal components was balanced more evenly.

R. F. Whelan presented evidence that actions of catecholamines on peripheral blood vessels in man could be accounted for by the concept of α- and β-receptor stimulation. Tyramine acted by releasing a constrictor substance more closely resembling noradrenaline than adrenaline or dopamine.

I. S. de la Lande, D. Frewin, J. Waterson, and V. Cannell applied drugs separately to intima or adventitia of the isolated perfused artery. The intraluminal application caused a higher degree of vasoconstriction than extraluminal application. This difference in activity tended to disappear in the presence of cocaine and after chronic sympathectomy. It was hypothesized that uptake by sympathetic neurons, located in the medial adventitia, reduced the concentration of amine reaching the smooth muscle to cause vasoconstriction. Cocaine administration or sympathectomy eliminated the uptake process.

M. L. Mashford and R. Zacest reported that adrenaline increased blood kininase activity and decreased blood kinin levels. They suggested that their results "made more plausible the hypothesis, bradykinin or similar peptides may play an important role in local control of blood flow integrated with the sympathetic nervous system."

C. J. Schwartiz and N. G. Ardlie demonstrated in vitro platelet-clumping activity by adrenaline and noradrenaline but not dopamine. All three catecholamines enhanced platelet aggregation due to ADP or ATP. It was suggested that catecholamines may prove important in the mechanism of thrombosis.

D. E. L. Wylcken, D. Brender, G. J. Macdonald, C. D. Shorey, and H. Hinterberger showed that reserpine caused a depletion of catecholamines which paralleled structural changes in myocardial mitochondria. These changes were reversible and may be important in the mechanism of action of reserpine.

W. G. Nayler presented investigations suggesting that myocardial contraction depends on continuing availability of ionized calcium which is partly dependent on the action of catecholamines.

C. L. Gibbs has studied the production of tension-independent heat in the myocardium which is probably involved in setting muscle in a state in which it can shorten and do work. It was found that epinephrine significantly increased this heat production and that this amine effect depended upon external calcium concentration.

A. D. Jose and F. Stitt reported that heart rate after β-adrenergic and cholinergic blockade paralleled depression or augmentation of myocardial contractile performance. He suggested that in absence of nervous stimuli rate and myocardial contractility share some common determinant.

D. A. Bloomfield and E. Sowton demonstrated a rate-independent reduction in myocardial contractility following β-adrenergic blockade in patients with complete A-V block treated with fixed-rate artificial pacemakers. Their studies pointed to a peripheral vasodilating action of propranolol in addition to its action on the heart. A sympathetic role in cardiac output maintenance was noted in the resting heart.

This monograph is a valuable contribution which presents concisely and clearly a wealth of information in a variety of areas involving catecholamine research. It embodies many new concepts and hypotheses which will stimulate further investigations in this rapidly expanding field of catecholamines.

WILLIAM M. MANGER, M.D., PH.D.


The biography of one physician by another is always something special. It is likely to be read by their fellow physicians with a combination of warm interest and critical expert knowledge. This book is no exception. Its theme is essentially biographical but has, as a counterpoint, the evolution of the Rockefeller Foundation in its role as an initiator in medical education and research throughout North America and the world. Set as pearls upon this richly interwoven fabric are the aphorisms of Alan Gregg, brief philosophical reflections on life, privately recorded in a notebook begun in 1918 while Gregg was immersed in the medical backwash of the 1914-18 war. A lucky find, these aphorisms, for any biographer despite the one (p. 97) which queries, "Is it to be expected that any experience memorable, satisfying or reliable can be had by spending
much time over other men’s resumés of other experience than your own?” The whole book provides the clear-cut answer, “Yes.”

The opening chapters follow the traditional pattern, sketching the family background and early education of Alan Gregg, and the journey east from his Colorado Springs birthplace to Harvard College. Soon he made some remarkable acquaintances, T. S. Eliot at Harvard, and Freud and Jung at the Putnam camp in the Adirondacks. Did these foreshadow his later interest in psychiatry and neurology? But, in general, Gregg’s years as a medical student and intern were as uneventful as those of any other ambitious, hard-working student. The change came after his return from the war with a decision to enter the public health program of the Rockefeller Foundation rather than settle into traditional medical practice. He went off to the remote regions of Brazil, to a medical and social environment equivalent to the Middle Ages in Europe. There he threw himself into an itinerant campaign to eliminate hook worm infestation—very different from medical practice in the Boston teaching hospitals and from his later years as an administrator with headquarters in Manhattan.

Gregg was no medical missionary in the traditional sense, but a humanist with a critical sense of agnostic detachment. This sense of independence, this questioning of the organization of society and agnostic from the Foundation he served so long, comes through very clearly in the book. They were undoubtedly valuable assets in his years spent surveying the medical schools of Europe and in his forays to Russia, China, and India. In small matters, too, he had a talent for the crisp incisive comment: of a Russian dermatologist turned bureaucrat, “. . . I’ve never met one yet who, having examined hairs for a living, wasn’t willing to shout ‘Wrong!’ when a hair was split incorrectly”; of a fellow administrator, “. . . the blunt if regretful candor of a pathologist.”

The chapter dealing with the founding and endowment of Montreal Neurological Institute by the Rockefeller Foundation, with Gregg acting as their agent, has a special impact; for it was this endeavor that brought the subject and his biographer together. Here too are raised questions of philanthropic strategy, of endowment as distinct from term grants and fellowships, of what to back and when, that permeate the latter half of the book and provide its title, “The Difficult Art of Giving.” One regrets the absence of some of the close-grained reports that must have been written during Gregg’s innumerable visits to medical schools, but perhaps these lie within the confidentiality of the Foundation. There is much here for argument about private philanthropy and the role of the State, but for many the charm of the book will lie in the close interweaving of the story of developing medical education and a rich and original personality.

GORDON MATHEISON, M.B., Ch.B.

Factors Influencing Myocardial Contractility.


The invitation to review this multi-authored book to which I contributed provided an unexpected stimulus to examine the end-product of the efforts of my coauthors and myself. Based on a Gordon Conference held in August 1966 under the auspices of the Cardiac Muscle Society, this book represents an extensive review of the physiology, biochemistry, biophysics, ultrastructure, pharmacology and pathophysiology of cardiac muscle contractility. The book itself includes material not presented and omits some of the topics that were presented at that conference.

This book might be best characterized as a series of progress reports in many of the disciplines relevant to the regulation of myocardial contractility. The scope is ambitious, both in terms of the number of topics considered and viewpoints presented (78 authors have contributed 56 chapters). As a result, the individual chapters are brief. In the 665 pages of text, the average chapter length is slightly less than 12 pages and many articles comprise only 6 pages, whereas few exceed 20 pages. Thus the reader will find most individual contributions lack the breadth of a “Physiological Review.”

The book is divided into nine sections. The first, “Contractile ‘Defect’ in Heart Failure,” is probably the least successful. The role of altered myocardial performance in contributing to clinical heart failure is carefully examined and certain biochemical and anatomic features of the failing heart are discussed. However, the relevant chapters are brief. That no definite conclusions can be reached about the causes of myocardial failure clearly reflects the present state of confusion and contradiction found in the literature on this subject.

The second section, “Contractile Behavior of Cardiac Muscle,” is well organized and effectively serves to acquaint the reader with the unique physiological properties of cardiac muscle. The problems encountered in applying the principles elucidated in frog skeletal muscle by A. V. Hill and others to the contractile performance of cardiac muscle are clearly set forth. The difficulties in defining the performance of the myocardium in terms of parameters such as $V_{max}$ and $P_{o}$. 

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which are difficult to define even in simple cardiac muscle preparations, are stressed, and the additional problems in deriving these parameters for the intact heart are clearly set forth in discussions of the structural and functional heterogeneity of ventricular contraction. The key chapters in this section are clear and substantial, and taken together constitute one of the more valuable portions of this book. Section III, "Contractile Behavior of the Heart," is, in effect, an extension of Section II. A series of well-written chapters describes further the changes in cardiac dimensions and wall thickness during the cardiac cycle in experimental animals.

These contributions provide an excellent background with which to understand the problems encountered in analyzing the function of the intact heart in terms of concepts introduced to describe the contractile behavior of the frog sartorius muscle.

The following Section, "Behavior of Heart Cells in Tissue Culture," surveys a variety of experiments which can be carried out with this preparation. This section contains a number of excellent discussions, such as the control of the pathways of energy production, the relationship of high energy phosphate levels to myocardial performance, and the electrophysiology of cardiac muscle cells in tissue culture.

Section V, "Excitation-Contraction Coupling" includes chapters on the role of Ca++ in the initiation of cardiac contraction, the control of myocardial calcium exchange, the localization of sarcotubular ATPase by electron microscopy, and the behavior of isolated subcellular fractions of cardiac muscle. These four chapters represent useful reviews of previous work, and some material is presented here for the first time.

The section on "Energetics" is introduced by a lucid and timely discussion of our present understanding of the force-velocity relationship of muscle. The experimental findings which caused A. V. Hill to modify his 1938 formulation of muscle energetics are described, and the early determinations of heat production by active cardiac muscle are discussed. A subsequent chapter briefly describes chemical determinations of the energy costs of various components of myocardial energy utilization. The possible role that modulation of the activity of the cardiac contractile proteins may play in causing the variable shortening velocity and contractile force of the myocardium is also considered in this section.

Sections VII to IX describe the actions of three classes of agents which modify myocardial contractility. Section VII provides an excellent review of the actions of catecholamines on myocardial carbohydrate metabolism, and our present understanding of the relationship of these effects of catecholamines on intermediary metabolism to their positive inotropic action is clearly set forth. This section also provides a review of the innervation of the heart and the consequences of surgical, immunological, and pharmacological reduction in the heart's sympathetic supply. The chapters on cardiac glycosides (Section VIII) review the present state of uncertainty regarding the mechanism responsible for the positive inotropic action of these drugs. Digitalis actions on the ion transport by the cell membrane, on the uptake of calcium by sarcoplasmic reticulum, on the cardiac contractile proteins and on the release of catecholamines by the myocardium are examined, and the possible roles of each of these potential mechanisms in causing enhanced myocardial contractility are discussed. That this important problem remains unsolved is apparent. A final section deals with the cardiac actions of thyroid hormone, corticosteroids, the sex steroids, angiotensin, vasopressin, and oxytocin. These chapters provide useful discussions in an important area of myocardial physiology.

This text is exceptionally readable, as it has large clear type and an attractive format. The overall organization is poor, however. For example, chapters on diastolic compliance are found in Sections I and II, on thyroid hormone in Sections VI and IX, and on catecholamines in Sections V, VII and IX. Tables of subheadings at the beginnings of the longer chapters are lacking, but for a book of this nature the index is surprisingly good.

"Factors Influencing Myocardial Contractility" cannot be considered a reference book and thus may be inappropriate for the libraries of physicians who have only a peripheral interest in myocardial function. On the other hand, as a collection of viewpoints on most of the important unresolved questions in the physiology of myocardial contractile behavior, this book can be recommended to the cardiologist who wishes a deeper understanding of the disputes and problems which face investigators in the field of myocardial physiology.

ARNO LD M. KATZ, M.D.

A Low Cholesterol Diet Manual. Department of Internal Medicine, College of Medicine, University of Iowa. Iowa City, Iowa, 1968, 61 pp. Price $1.00.

It is now firmly established that the incidence of coronary heart disease and probably of other complications of atherosclerosis rises steeply with the serum cholesterol level. It is also clear that suitable dietary adjustments will significantly lower the cholesterol level in the great majority

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of persons, especially those with high values, say 260 mg/deciliter or more. Accordingly, physicians, and their patients as well, increasingly ask for guidance in dietary prescription.

The Low Cholesterol Diet Manual of the Department of Internal Medicine of the University of Iowa College of Medicine is the latest addition to the list of publications that attempt to offer such guidance. For the patient, it has the virtues of simplicity, absence of technical discussions, and modest price. The physician who recommends it can be sure that the serum cholesterol will fall if the prescription is followed. But serious questions can be asked about practicality; the restrictions are drastic indeed.

The basic plan is to provide only 20% of calories in the form of fat and to reduce exogenous cholesterol to 100 mg in 2,400 calories. No account is taken of the fact that substitution of unsaturated vegetable oils for saturated animal fats in the diet will lower the cholesterol even more than merely reducing the saturated fats. And insistence on such severe dietary restriction of cholesterol-containing foods gives too much credit to the effect of cholesterol in the diet. Changing from an ordinary U. S. diet to the Iowa diet should produce an average fall of 12 to 15 mg/deciliter because of the change in dietary cholesterol alone. That modest change in the blood should be welcome, of course, but the dietary restriction is severe.

Within the rigid limitations set by this self-imposed pattern, the dietitians have done well. The recipes, so far as they go, are good and the directions clear. Some 114 recipes would scarcely add up to a lifetime of good eating, but after experience with those recipes the ingenious cook could devise many variants. The trouble, according to this reviewer, is that a much less rigid dietary plan could accomplish the same result in terms of serum cholesterol.

Ancel Keys, Ph.D.


The clearly organized presentations which we have come to expect of Dr. Braunwald's group characterizes this short book on cardiac physiology. Based to a large extent on studies of the authors and their collaborators, the text examines the ultrastructure, the biophysics, and the biochemistry of cardiac muscle, emphasizing whenever possible the relevance of these basic aspects of myocardial function to the physiological and clinical performance of the intact heart. Major topics at the basic level include: (1) the relationship of ultrastructure to the length-tension diagram of cardiac muscle (the Frank-Starling law of the heart); (2) the mechanics of heart muscle, both in simple papillary muscle preparations and in the much more complex situation of the intact ventricle; (3) myocardial energetics; and (4) the biochemistry of the cardiac contractile proteins.

Following this discussion of basic myocardial physiology, which occupies the first hundred pages of the book, the authors turn to an examination of the function of the human heart. The feasibility of defining myocardial performance in man in terms of principles elucidated for various experimental preparations is examined and applications of these principles are illustrated by a consideration of the myocardial adaptations to exercise. A chapter on heart failure provides a brief but balanced survey of this controversial subject. Possible abnormalities in the reactions leading to ATP regeneration in the failing heart are discussed, as are a potential abnormality of the contractile proteins, the mechanics of the failing myocardium, and abnormalities of catecholamine metabolism in this condition. This discussion of myocardial failure demonstrates the advantage to be gained from consideration of myocardial function in terms of the previously elucidated concepts of muscle mechanics.

The book concludes with an examination of the mechanisms which modify cardiac performance and cardiac output. The factors that act to modify the myocardial contribution to the control of cardiac output (ventricular end-diastolic volume, afterload, and contractility) are well set out, but the other half of this control system, the regulation of venous return, is only briefly mentioned. It is regrettable that no mention is made of Guyton's lucid formulation of the interplay between venous return curves and cardiac response curves in determining the level of cardiac output.

This book, based on a "Medical Progress Report" that appeared in the New England Journal of Medicine, is directed primarily to the practicing physician and clinical cardiologist. It is not surprising, therefore, that experts in the many areas covered will find significant omissions and errors. In the discussion of the heart's contractile proteins, for example, troponin, the protein that sensitizes actomyosin to Ca++, is not described, and its function is incorrectly ascribed to troponyosin, and the abundant evidence that cardiac and skeletal myosin have different amino acid sequences is overlooked. In discussing the imprecisions inherent in estimating the force-velocity curves of cardiac muscle, the authors correctly point out that Pe (maximum force devel-
op ed) cannot be determined for cardiac muscle with the degree of accuracy that it can be measured in a skeletal muscle because the myocardium cannot be tetanized. On the other hand, little attention is given to the difficulties inherent in estimating \( \nu_{\text{max}} \) in cardiac muscle, in which the active state is slow in onset and thus incompletely developed at the time, early in systole, when the muscle develops its highest velocity of shortening.

In a text of this nature, however, such areas of potential disagreement are of relatively minor importance and do not detract from the remarkable achievements represented by this book. This well-written, clearly organized introduction to the contractile physiology of the normal and abnormal heart is highly recommended for the physician who wishes to understand the current conceptual framework by which the function of the myocardium can be analyzed.

Arnold M. Katz, M.D.


Drs. Rubin, Gross, and Arbide have taken on a Herculean task in an attempt to cover the treatment of all forms of adult cardiac disease in one book. The authors discuss separately the treatment of each form of heart disease, outlining the treatment in cookbook fashion. The book is not intended to be a reference manual, but is intended to aid the practicing physician in the care of his cardiac patient.

One of the strong points of this book is the charts. These are concise, clearly labeled and allow the physician the opportunity of seeing at a glance the present-day therapy of many cardiological problems. The authors are not able to discuss in detail many aspects of treatment in all forms of heart diseases included in this text.

A reviewer tends to look for deficiencies and there are some. This particular reviewer would take exceptions to some statements in the text. The statement, "Repair for aortic valve stenosis without replacement is no longer considered satisfactory," should be modified to exclude the young adult with congenital bicuspid aortic stenosis. The discussion of the "ideal subject" for mitral comissurotomy lacks some of the important criteria that should be included in this description. The statement that surgical intervention for congenital heart disease carries a higher risk in the adult than in children cannot be applied universally and should be followed by some qualifying comments. The chapter on cardioversions for cardiac dysrhythmias expresses the opinions of the authors but does not necessarily reflect the opinion of many cardiologists working in this field.

As a whole, the book represents a most commendable attempt to represent the current trend in the treatment of heart disease in the adult patient. The book should be helpful to the intern primarily, and to the practicing physician.

Emilio R. Giuliani, M.D.
BOOK REVIEWS

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