

SELF ASSESSMENT IN CLINICAL CARDIOLOGY II

Edited by

Michael S. Gordon, M.D., Ph.D., F.A.C.P., F.A.C.C.
Clinical Associate Professor of Medicine;
Director, Annual Teaching Conference in Clinical Cardiology
University of Miami School of Medicine
Miami, Florida



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HEART ASSOCIATION OF GREATER MIAMI FLORIDA HEART ASSOCIATION



TO LEE AND DORÉE JOAN DAVID, KEVIN, AND CATHY

PREFACE

The acceptance of our first effort has stimulated the writing of Self Assessment in Clinical Cardiology II. Again, the work has evolved from the Teaching Conference in Clinical Cardiology held each spring for the last 8 years at the University of Miami. We hope it will continue to be valuable to medical students, house officers and practicing physicians.

It is nearly impossible to please "all of the students all of the time." The book, however, does cover a broad range of topics from basic principles in bedside diagnosis, a core curriculum in electrocardiography and practical drug therapy, to advances in electrophysiology, the hemodyanmics of cardiogenic shock and cardiac scintigraphy. The section on echocardiography is extensive by design: it is an extremely important non-invasive technique deserving wide application. Throughout, we have tried to be teachers - to clarify the old and simplify the new - and relate our efforts to clinical patient care.

As stated in the preface to the first volume, "The deadline we faced for our conference means that the book may not be 'polished.' I believe it does, however, accurately state where we are in most of the important areas of clinical cardiology and it does allow the reader to assess his abilities and knowledge in the practical aspects of caring for cardiac patients an important feature in these days of recertification as a means of improving patient care. It does so at a low cost in dollars, but possibly at a high cost in terms of alienation of colleagues.

If you want to get a visiting professor to hate you, just ask him, two months in advance of his talk, for a comprehensive but succinct "abstract" of his presentation which really says something - not just an outline - but real information. Add to that a request for selected references, graphs and figures, in addition to well thought out questions and answers. Then keep reminding him of the deadline. Finally, take his work and edit it, most often without an opportunity for his further approval. The result is before you. If there are errors, they are my own, born of an urgency that wants to teach what is new new - the kind of urgency that will get this book to the printer within the hour."

Thanks to our guest faculty for their superb teaching and becoming my friends. Thanks to Proc Harvey for teaching me and stimulating me to teach. Thanks to Emanuel Papper for always nurturing innovation in education. Thanks to Vi, Pam, Susan and Pat for their support. And special thanks to Judith Greve whose intelligence and patience have made this book a reality.

M.S.G.

GUEST CONTRIBUTORS

- Lawrence S. Cohen, M.D., Professor of Medicine, Chief of Cardiology, Yale University School of Medicine
- Gordon Ewy, M.D., Professor of Medicine, Director, CCU and Diagnostic Cardiology, Arizona Medical Center
- Robert A. O'Rourke, M.D., Associate Professor of Medicine, Director, Clinical Cardiology, University of California, San Diego
- William C. Roberts, M.D., Chief, Section of Pathology, National Heart and Lung Institute, Professor of Pathology and Medicine, Georgetown University
- James Ronan, M.D., Assistant Professor of Medicine, Georgetown University, Co-Director, Division of Cardiology, Washington Adventist Hospital
- H.J.C. Swan, M.D., Ph.D., Professor of Medicine, UCLA School of Medicine, Director of Cardiology, Cedars-Sinai Medical Center
- Andrew G. Wallace, M.D., Professor of Medicine, Chief, Division of Cardiology, Duke University
- Hein J.J. Wellens, M.D., Professor of Cardiology, University of Amsterdam, Head, Department of Cardiology, Wilhelmina Gasthuis, Amsterdam

UNIVERSITY OF MIAMI CONTRIBUTORS

- Benjamin Befeler, M.D., Associate Professor of Medicine, Chief, Cardiovascular Laboratory, Veteran's Hospital
- Robert J. Boucek, M.D., Professor of Medicine
- Agustin Castellanos, M.D., Professor of Medicine, Chief, Clinical Electrophysiology, Jackson Memorial Hospital
- Nabil El-Sherif, M.D., Assistant Professor of Medicine, Chief, Coronary Care Unit, Veteran's Hospital
- Michael S. Gordon, M.D., Ph.D., Clinical Associate Professor of Medicine
- Stuart Gottlieb, M.D., Assistant Professor of Radiology and Medicine, Head, Ultrasound and Cardiovascular Units, Division of Nuclear Medicine, JMH
- Gerard Kaiser, M.D., Professor of Surgery, Chief, Division of Thoracic and Cardiovascular Surgery
- Nilza Kallos, M.D., Instructor of Radiology(Nuclear Medicine and Ultrasound, Jackson Memorial Hospital)
- Ralph Lazzara, M.D., Associate Professor of Medicine, Chief of Cardiology, Veteran's Hospital
- Louis Lemberg, M.D., Professor of Clinical Cardiology
- James R. LePage, M.D., Associate Professor of Radiology, Chief, Cardiac and Vascular Radiology Section, Jackson Memorial Hospital
- Stephen M. Mallon, M.D., Assistant Professor of Medicine, Chief, Cardiovascular Laboratory, Jackson Memorial Hospital
- Joan Mayer, M.D., Clinical Associate Professor of Medicine
- Alvaro Mayorga-Cortes, M.D., Assistant Professor of Medicine, Director, Coronary Care Unit, Jackson Memorial Hospital
- Robert J. Myerburg, M.D., Professor of Medicine, Director, Division of Cardiology
- Eliseo Perez-Stable, M.D, Professor of Medicine, Chief, Medical Service, Veteran's Hospital
- David S. Sheps, M.D., Assistant Professor of Medicine, Director, Exercise and Non-Invasive Laboratory, Jackson Memorial Hospital
- Manuel Viamonte, Jr., M.D., Professor and Chairman, Department of Radiology, Director of Radiology, Jackson Memorial Hospital and Mt. Sinai Hospital
- Willis H. Williams, M.D., Assistant Professor of Surgery

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BEDSIDE DIAGNOSIS AND AUSCULTATION

REVIEW OF CARDIAC ANATOMY AND PHYSIOLOGY

Robert A. O'Rourke, M.D.

A brief discussion of the functional anatomy of the cardiovascular system is a necessary prelude to further instruction concerning the diagnosis and treatment of cardiac disease. Differences in anatomy between the right atrium and right ventricle which return unoxygenated blood to the low resistance pulmonary circulation should be compared to the left atrium and left ventricle which function respectively as a receiving chamber for oxygenated blood and a thick walled high pressure chamber for pumping blood against the systemic vascular resistance.

A diagramatic representation of pressure tracings recorded within the right and left-sided cardiac chambers and great vessels together with simultaneous electrocardiogram and phonocardiogram are shown in Figure 1. The diagonal cross-hatched areas labeled ISOV represent the isovolumic phases of left ventricular contraction and relaxation. Isovolumic right ventricular contraction and relaxation is represented by double cross-hatching. M₁ and T₁ represent sounds produced by closure of the mitral and tricuspid valves respectively. A₂ and P₂ are sounds produced by closure of the aortic and pulmonic valves while OT and OM represent sounds produced by opening of the tricuspid and mitral valve, respectively. PEP represents the pre-ejection period from the onset of the Q wave on ECG to the opening of the aortic valve. LVET is the left ventricular ejection time.

As the physician cares for his patients, he should be thinking of these hemodynamic relationships, as they form the foundation for understanding and practicing cardiology at any level. As his sophistication increases, more practical data is added, e.g., the volume curve of the left ventricle, the jugular venous pulse, the apex cardiogram, and today, even echocardiographic movement of the valves.

An understanding of the anatomy of the mitral valve and its functional components (annulus, leaflets, chordae tendineae, papillary muscles, left atrium and left ventricle) is important in understanding the causes and consequences of acute or chronic mitral regurgitation (Figure 2). With this understanding there follows an appreciation of why "mitral regurgitation begets mitral regurgitation," i.e., when the mitral valve leaks, the left atrium is volume or preloaded, delivers this extra volume to the ventricle during the next diastole, chronically enlarging the ventricle, which pulls on the papillary muscles and results in further unseating of the valve in systole and more regurgitation. Similarly, alteration in the continuity of geometry of any one or combination of these structures can produce severe mitral regurgitation and markedly elevated pulmonary venous pressure: This sequence depends particularly on the compliance characteristics of the left atrium, i.e., in acute severe

mitral regurgitation, the left atrium is small and relatively non-compliant and ventricular pressures are hence reflected back into the pulmonary circulation. Finally, thickening of the mitral leaflets, fusion of the commissures and shortening of the chordae produce mitral stenosis with or without coincident mitral regurgitation in many patients with rheumatic heart disease.

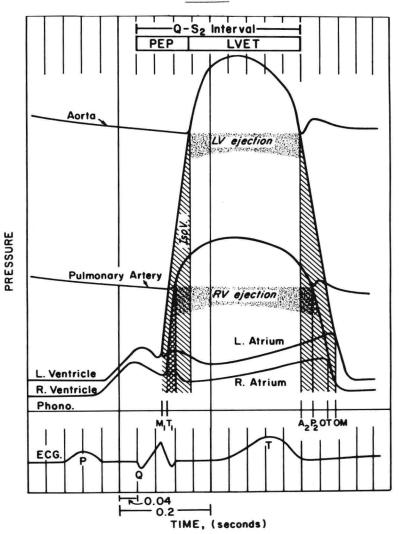
Both semilunar valves (pulmonic and aortic) usually have three leaflets, but some patients have a bicuspid aortic valve. With time, significant aortic stenosis and/or aortic regurgitation develop in many of these patients, and a congenital bicuspid aortic valve, frequently calcified, is the most common cause of isolated aortic valve disease.

Knowledge of the anatomy of the coronary circulation and the distribution of the left anterior descending, left circumflex and right coronary arteries to the left and right ventricular myocardium, the papillary muscles of the mitral valve, the ventricular septum, the SA and AV nodes, and the branches of the trifascicular conduction system is a prerequisite for understanding the various complications of severe coronary artery disease and acute myocardial infarction (Figures 3 and 4).

The relationship between the left ventricular stroke volume and the left ventricular filling pressure or end-diastolic volume is shown in Figure 5. In the normal ventricle, the volume of blood ejected from the left ventricle is related to the volume of blood distending the left ventricle at end diastole (ventricular function curve). In patients with a reduced left ventricular performance, the curve relating cardiac output to left ventricular end-diastolic fiber length is shifted downward and to the right so that a higher left ventricular filling pressure is necessary to maintain a normal cardiac output and the cardiac output resulting from any given left ventricular filling pressure is reduced. This relationship should be kept in mind when using a variety of cardiac drugs, e.g., digitalis and diuretic therapy in patients with congestive heart failure.

Selected References

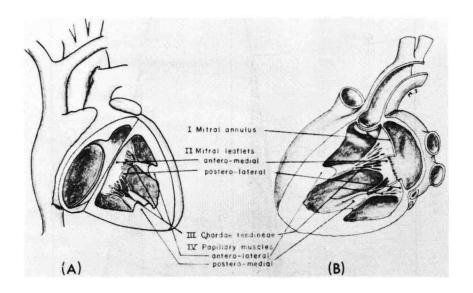
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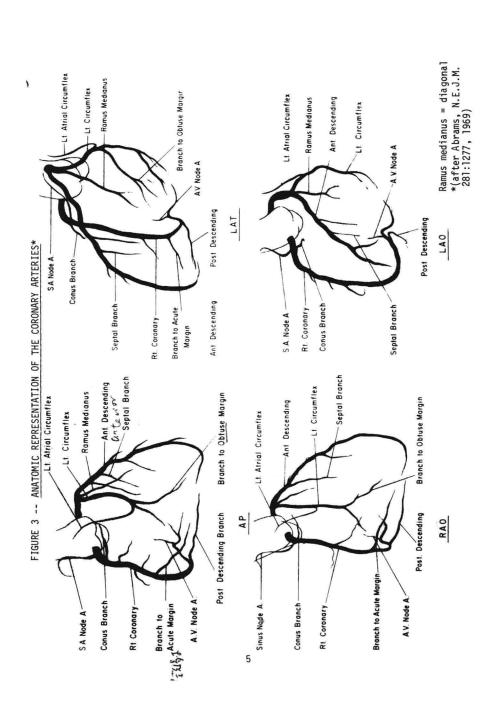
The first third of systole and diastole are the periods of rapid ejection and filling during which two-thirds of the stroke volume leaves or enters the ventricle. Hence murmurs related to flow and turbulence alone are heard early in their respective phases of the cardiac cycle.

The S3 and S4 diastolic filling sounds are heard as blood rapidly decelerates after initial rapid filling, and again as it decelerates in presystole after rapid filling from atrial contraction.

FIGURE 2



The mitral valve is the most complex of the cardiac valves. Its leaflets are unequal in size: one is continuous with the left atrial endocardium and the other with the aorta. The valve contains about 120 chordae tendineae connecting the leaflets to myocardial pillars (papillary muscles), which in turn are continuous with the left ventricular free wall. It has only a partial annulus fibrosis (only at the base of the posterior mitral leaflet). Since the mitral valve connects with the left atrial wall via the mural endocardium, conditions affecting this chamber also may affect mitral function.



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