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YEAR BOOK OF CARDIOLOGY® 1988

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The Year Book of CARDIOLOGY®

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Publisher's Preface

We welcome Norman Kaplan, M.D., as an Editor of the YEAR BOOK OF CARDIOLOGY. Dr. Kaplan, a Professor of Internal Medicine and Chief of the Hypertension Division at the University of Texas Southwestern Medical Center in Dallas, selected and commented on material related to hypertension.

Introduction

This 1988 YEAR BOOK OF CARDIOLOGY is the 28th in the series. The volume continues the objectives and formats of its predecessors. We are pleased to welcome Norman M. Kaplan to the Editorial Board, where he will have responsibility for hypertension. As before, all section editors have complete freedom to select the articles for inclusion in their areas. Articles are selected for abstracting throughout the year, and occasionally another fine article on the same or a similar subject is published later in the year. In this situation, the editors attempt to refer to the later article in their comments whenever possible and appropriate.

All of the section editors again thank the staff at Year Book Medical Publishers for their assistance, patience, and understanding. In particular, we are indebted to Ms. Nancy Gorham and Ms. Bonnie Meyers.

Robert C. Schlant, M.D.

Journals Represented

Year Book Medical Publishers subscribes to and surveys more than 700 U.S. and foreign medical and allied health journals. From these journals, the Editors select the articles to be abstracted. Journals represented in this YEAR BOOK are listed below.

Acta Medica Scandinavica
American Heart Journal
American Journal of Cardiology
American Journal of Diseases of Children
American Journal of Epidemiology
American Journal of Medicine
American Journal of Noninvasive Cardiology
American Journal of Pathology
American Journal of Physiology
Anesthesiology
Annals of Internal Medicine
Annals of Surgery
Annals of Thoracic Surgery
Archives of Disease in Childhood
Archives of Internal Medicine
British Heart Journal
British Medical Journal
Canadian Family Physician
Cardiovascular Research
Chest
Chinese Medical Journal
Circulation
Circulation Research
Clinical Pharmacology and Therapeutics
Critical Care Medicine
Current Problems in Cardiology
Hypertension
International Journal of Cardiology
Journal of the American College of Cardiology
Journal of the American Medical Association
Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology
Journal of Cardiovascular Surgery
Journal of Chronic Diseases
Journal of Clinical Investigation
Journal of Clinical Pharmacology
Journal of Hypertension
Journal of Nuclear Medicine
Journal of Pediatrics
Journal of Surgical Research
Journal of Thoracic and Cardiovascular Surgery
Klinische Wochenschrift
Lancet
Mayo Clinic Proceedings
Medical Journal of Australia
Nephron
New England Journal of Medicine
Pediatric Cardiology

Pediatrics
Postgraduate Medical Journal
Presse Medicale
Psychosomatic Medicine
Quarterly Journal of Medicine
Surgery

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The material covered in this volume represents literature reviewed up to November 1987.

1 Normal and Altered Cardiovascular Function

Introduction

Clinically relevant, recently published articles concerning cardiovascular physiology, myocardial metabolism, commonly used noninvasive methods, and newer diagnostic and therapeutic techniques are reviewed in this section of the YEAR BOOK OF CARDIOLOGY. Both experimental animal and clinical studies are abstracted and discussed; a list of additional references is provided at the end of each subsection.

In the first subsection on ventricular hypertrophy, the effects of chronic pressure overload or hormonally induced left ventricular hypertrophy on direct ventricular interaction, echocardiographically determined left ventricular mass, rest and exercise myocardial blood flow, myocyte microtubule reorganization, myosin heavy chain isozyme transitions, and left ventricular diastolic function are detailed. The unanswered question concerning the desirability of reversing left ventricular hypertrophy with antihypertensive therapy is again addressed.

The subsection on ventricular diastolic function includes reports on the various determinants of left ventricular isovolumic relaxation, the effects of aging on left ventricular diastolic function, and the direct and indirect effects of calcium entry blocking drugs on left ventricular relaxation. Areas of controversy are highlighted in the editors' comments and in the additional references listed.

The ventricular systolic function subsection provides further detailed information on the left ventricular end-systolic pressure-volume relationship (ESPVR) and the limitations of ESPVR as a sensitive, load-independent indicator of the inotropic state or changes in the inotropic state, particularly when applied in clinical patient studies. Other reports in this subsection concern the effects of verapamil, endurance training, cyclic AMP phosphodiesterase, and aortic valve disease on left ventricular systolic performance; the sympathetic nerve activity during dynamic exercise; and abnormal calcium handling in myocardium from patients with end-stage heart failure.

The subsection on experimental myocardial ischemia/infarction contains several reports on the pathogenesis and pathophysiology of the "stunned" myocardium; the effects of myocardial ischemia on regional myocardial function and stiffness; and the role of granulocytes and oxygen radical formation in myocardial ischemia/infarction and reperfusion injury. The importance of defining the left ventricular region at risk by

noninvasive or invasive means when assessing an agent's beneficial effects on "infarct size" is stressed and the use of isopotential surface mapping for detecting experimental right ventricular infarction is detailed. Additional reports concern the detection of myocardial necrosis with Indium-III antimyosin Fab; the detrimental effect of frequent ventricular ectopy on the extent of infarction; and the ability to produce successful coronary thrombolysis in experimental animals by the early intramuscular administration of human tPA with selected absorption enhancers.

The subsection on coronary artery spasm and stenosis details studies concerning a miniature swine model of coronary spasm produced by balloon denudation of regional coronary artery endothelium and the effects of leukotrienes C_4 and D_4 and of a thromboxane A_2 analogue on coronary artery vasomotor tone. Additional information concerning the mechanism by which calcium blockers induce preferential coronary artery dilation (α_1 adrenergic blockade) is described, and the usefulness of high-frequency epicardial echocardiography and quantitative angiographic methods for assessing the extent of coronary atherosclerosis is reviewed.

The noninvasive testing subsection includes reports on ECG exercise tests in the detection of asymptomatic myocardial ischemia; the usefulness of Doppler echocardiography for measuring cardiac output, stenotic valve areas, and left ventricular diastolic function; and the role of nuclear cardiology methods in defining patients needing catheterization and for assessing the results of coronary thrombolysis in patients with acute myocardial infarction.

The final subsection on newer diagnostic and therapeutic techniques describes the potential usefulness of ultrafast computed tomography for quantitating regional myocardial perfusion; the accuracy of nuclear magnetic resonance imaging (NMRI) for quantitating aortic and mitral regurgitation in patients; the possible use of NMRI for early detection of adriamycin toxicity; and the potential application of NMRS for detecting cardiac allograft rejection. Additional animal studies of clinical importance concern the safe and effective catheter ablation of the atrioventricular junction using radio frequency energy and the lack of myocardial injury produced by multiple defibrillating shocks in dogs undergoing serial testing of the efficacy of an automatic implantable cardioverter/defibrillator.

Robert A. O'Rourke, M.D.

Ventricular Hypertrophy

Chronic Pressure Overload Hypertrophy Decreases Direct Ventricular Interaction

Bryan K. Slinker, Antonio Carlos, P. Chagas, and Stanton A. Glantz (Univ. of California, San Francisco)

Am. J. Physiol. 253:H347–H357, August 1987 1–1

Right ventricular volume can influence left ventricular volume through series interaction, as the right heart output becomes the left heart input,

and through direct interaction of the shared interventricular septum. Disease states that alter direct ventricular interaction will complicate assessment of left ventricular function. In hypertrophied hearts, direct interaction has not been well characterized. Therefore, the relative importance of direct interaction at end diastole and end systole, with and without the pericardium, was studied in dogs with concentric hypertrophy induced by chronic renovascular hypertension.

At end diastole, direct interaction was only approximately 10% as important as series interaction in determining left ventricular size. At end systole, direct interaction was approximately 20% as important as the pressure-volume relationship in determining left ventricular size. After removal of the pericardium, direct interaction became even less important in determining left ventricular size.

Because of the changing role of direct ventricular interaction, the slope of the end-systolic pressure-volume relationship is not comparable between normal and hypertrophic hearts. This further complicates clinical application of the end-systolic pressure-volume relationship.

▶ Direct ventricular interaction normally causes both left ventricular end-diastolic and end-systolic pressure-volume relationships to deviate from what they would be if no direct interaction occurred. The effects of ventricular interaction are reduced when the pericardium is removed. The study by Slinker and associates indicates that the influence of direct interaction between the ventricles on assessment of left ventricular function is decreased significantly when the left ventricular hypertrophies. Moreover, since the magnitude of direct ventricular interaction is different in normal as compared with hypertrophied hearts, pressure-volume relationships from normal and hypertrophied hearts cannot be compared simply.—R.A. O'Rourke, M.D.

Echocardiographic Left Ventricular Mass and Function in the Hypertensive Baboon

Michael H. Crawford, Richard A. Walsh, David Cragg, Gregory L. Freeman, and Jacelyn Miller (Univ. of Texas, San Antonio)

Hypertension 10:339–345, September 1987 1–2

Cardiac anatomy of nonhuman primates is particularly close to the human situation, and larger animals are better adapted to sophisticated hemodynamic studies. Left ventricular (LV) mass was investigated in the baboon by M-mode echocardiography, and LV size and function in chronic renal hypertension was characterized (Fig 1–1). Chronic hypertension of gradual onset was produced by either the two-kidney, one-clip Goldblatt procedure or by bilateral cellophane-wrap perinephritis.

Autopsy studies validated echographic estimates of LV mass. The reproducibility of echographic LV mass estimation was quite good. Body weight and heart rate were similar in hypertensive and normotensive animals, but peak systolic LV pressure was greater in the hypertensive animals, as was end-diastolic LV posterior wall thickness. The LV cross-

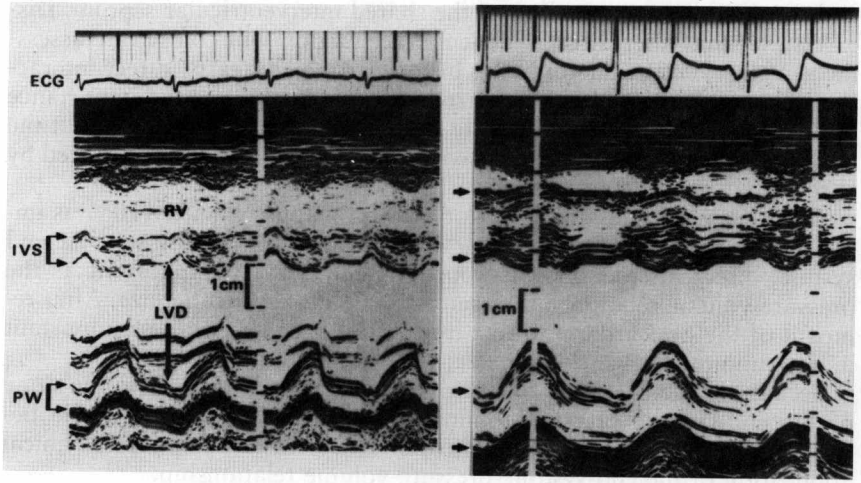

Fig 1–1.—M-mode echocardiograms of the left ventricle in a normotensive control baboon (**left**) and a hypertensive animal (**right**). *ECG*, electrocardiogram; *RV*, right ventricle; *IVS*, interventricular septum; *LVD*, left ventricular dimension: *PW*, posterior wall. *Arrows* indicate wall thickness. (Courtesy of Crawford, M.H., et al.: Hypertension 100:339–345, September 1987.)

sectional area and calculated mass were 44% greater in hypertensive animals, despite similar LV cavity dimensions. Rates of LV dimensional change and wall-thickness change in systole and diastole were about 25% less in hypertensive than in normotensive animals, despite matched heart rates and LV stress values. The overall percent change in cavity dimension and wall thickness in systole was not significantly altered in the hypertensive group.

The biochemical basis for load-independent changes in LV function resulting from hypertensive pressure-overload hypertrophy in higher mammals requires further study. M-mode echocardiography appears to be a useful means of following LV performance in a nonhuman primate model of pressure-overload hypertrophy.

▶ This study shows the feasibility of using M-mode echocardiography for the serial noninvasive assessment of left ventricular size and performance in the nonhuman primate with experimentally produced renal hypertension. Echocardiographically determined left ventricular mass accurately estimated postmortem left ventricular weight, and the echocardiographic measurement of mass was highly reproducible. Factors contributing to the hypertrophy process, such as altered myocardial contractility, relaxation, or material properties, may have accounted for the abnormal left ventricular chamber performance observed in this animal model. The depressed rates of LV chamber emptying and filling observed in these hypertrophied baboons are consistent with those of other studies of experimental renal hypertension.—R.A. O'Rourke, M.D.

Myocardial Blood Flow in Left Ventricular Hypertrophy Developing in Young and Adult Dogs

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