Advances in Critical Care Cardiology

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editor

CARDIOVASCULAR CLINICS, Albert N. Brest, Editor-in-Chief

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Advances in Critical Care Cardiology

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Preface

Critical Care Cardiology, published in 1981, recognized the advances in diagnostic technologies and treatment for acute disturbances in the heart and vascular system. At that time, pharmacologic and mechanical interventions, cardiac surgery, and monitoring acutely ill patients were considered avant-garde in cardiovascular medicine. The rapid integration and implementation of basic research into the reduction of morbidity and mortality of acute cardiac disorders were noteworthy. In 1981, such achievements were startling, and they no doubt left physicians somewhat incredulous that further advances could be achieved in the field of critical care of cardiac patients.

In the brief period of 5 years, thrombolytic therapy in acute myocardial infarction and coronary angioplasty have become accepted forms of medical treatment. New pharmacologic agents include the calcium-blocking drugs and an increasing array of effective anti-arrhythmic drugs. The technical advances in pacemaker design and insertion, and the improvements in surgical techniques for prosthetic valves, aortic dissection, and acute myocardial infarction have been impressive. Parallel to the technological advances in the intensive care unit, as well as medical and surgical treatments, has been the expansion of the important role of nursing care for critically ill patients.

Our new book, *Advances in Critical Care Cardiology,* draws from clinical experience, research, and the cooperative efforts of nurses, cardiologists, and cardiac surgeons working together within the same institution. Such efforts are being duplicated throughout the country and world and provide testimony to the enormous accomplishments and benefits to critically ill patients with cardiovascular disease.

Charles E. Rackley, M.D.
Guest Editor

Editor's Commentary

An earlier issue of CARDIOVASCULAR CLINICS, entitled *Critical Care Cardiology,* was published 5 years ago. The present coverage includes many important topics that were either unknown or barely in sight at the time of the earlier publication, for example, thrombolytic therapy, calcium-blocking agents, and catheter treatment of acute myocardial infarction. Other topics not previously covered, such as pacemaker emergencies, cardiac tamponade, and recognition and management of postcardiac surgical complications, are also detailed in the present volume. Two new chapters are devoted to the nursing management of acute myocardial infarction and the postoperative cardiac surgical patient, thus providing insight into and recognition of the invaluable contributions of our nursing colleagues in the handling of cardiac emergencies. Additionally, emergency aspects of cardiogenic shock, heart failure, ventricular and supraventricular arrhythmias, complicated infective endocarditis, prosthetic valvular dysfunction, and other topics are all completely updated. Given the notable advances that have taken place since the last publication, it seems quite appropriate to devote another volume to this important topic. I am again indebted to the Guest Editor, Charles E. Rackley, for his guidance in the development of this book, and both of us are extremely grateful to the individual authors for their exemplary contributions.

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Contents

PART 1

Myocardial Infarction

CHAPTER 1

Use of Hemodynamic Measurements for Management of Acute Myocardial Infarction

Charles E. Rackley, M.D., Lowell F. Satler, M.D., David L. Pearle, M.D., Albert A. Del Negro, M.D., Randolph S. Pallas, M.D., and Kenneth M. Kent, M.D., Ph.D.

Clinicians and pathologists have recognized for decades that the extent of myocardial infarction directly contributes to the mortality and morbidity of this condition.[1] Disturbances in left ventricular function reflect the amount of myocardial damage more than any other clinical or physiologic expression.[2] The development of the Swan-Ganz catheter, computer systems, and the modern-day coronary care unit have provided the clinical environment for the acquisition of sensitive measurements of cardiac performance in patients with acute myocardial infarction.[3,4] Thus, by providing measurements of the left ventricular filling pressure and cardiac output, the Swan-Ganz catheter has permitted the clinical application of Starling's Law of the Heart to the left ventricle in acute myocardial infarction. Hemodynamic measurements can further identify the basic determinants of myocardial performance and oxygen consumption of the isolated cardiac muscle in terms of preload, afterload, contractile state, and heart rate.[5]

New methods for obtaining hemodynamic measurements in patients with acute myocardial infarction, measurements of mechanical determinants of oxygen consumption and ventricular performance, initial and serial observations in acute infarction, and the specific pharmacologic agents that affect cardiac function in acute infarction will be reviewed.

METHODS

The Swan-Ganz catheter is optimally introduced under fluoroscopic visualization. Several peripheral venous sites are accessible, including the antecubital vein, subclavian vein, and the jugular vein. Although the Swan-Ganz catheter can be introduced in the femoral vein during standard cardiac catheterization procedures, the incidence of venous thrombosis is higher when the catheter remains for prolonged periods in the femoral vein than when it is in the brachial or jugular vein. Standard sterile precautions should be taken with the introduction of the Swan-Ganz catheter and should include not only sterile preparation of the field but also the use of surgical attire, gowns, caps, masks, and gloves. After the Swan-Ganz catheter has been advanced to a peripheral pulmonary artery, the balloon can be inflated for the recording of the pulmonary capillary wedge pressure. The wedge pressure reflects the mean left atrial pressure, which correlates closely with the left ventricular end-diastolic pressure. Left ventricular end-diastolic pressure is the most sensitive parameter of acute alterations in left ventricular function.[6]

Once the wedge pressure has been recorded, the balloon can be deflated and the catheter withdrawn into one of the main branches of the pulmonary artery. Recording of the systolic and diastolic pressure should reveal a close correlation between the pulmonary artery end-diastolic pressure (PAEDP) and the pulmonary capillary wedge pressure.[7] Should the PAEDP be 3-5 mm Hg higher than the wedge pressure, pulmonary arteriolar constriction should be suspected and the possibility of pulmonary embolism entertained. Once the catheter tip is positioned, the PAEDP can be monitored continuously as an expression of left ventricular filling pressure and left ventricular end-diastolic pressure. Cardiac output can be measured by the injection of a cold solution in the right atrium and the recording of the thermodilution changes at the tip of the Swan-Ganz catheter.

In addition to the measurements of the left ventricular filling pressure and cardiac output, the blood volume can be expanded by the rapid infusion of low molecular weight dextran into the pulmonary artery, and subsequent changes in the PAEDP and cardiac output can be related to baseline measurements.[8] Incremental changes in PAEDP and cardiac index can be related by constructing a ventricular function curve (Fig. 1). The ventricular function curve is a classic physiologic technique for relating a diastolic parameter, such as the left ventricular filling pressure, to a systolic expression of ventricular performance, such as the cardiac output or cardiac index. The relationship between changes in filling pressure and cardiac index are graphically displayed by

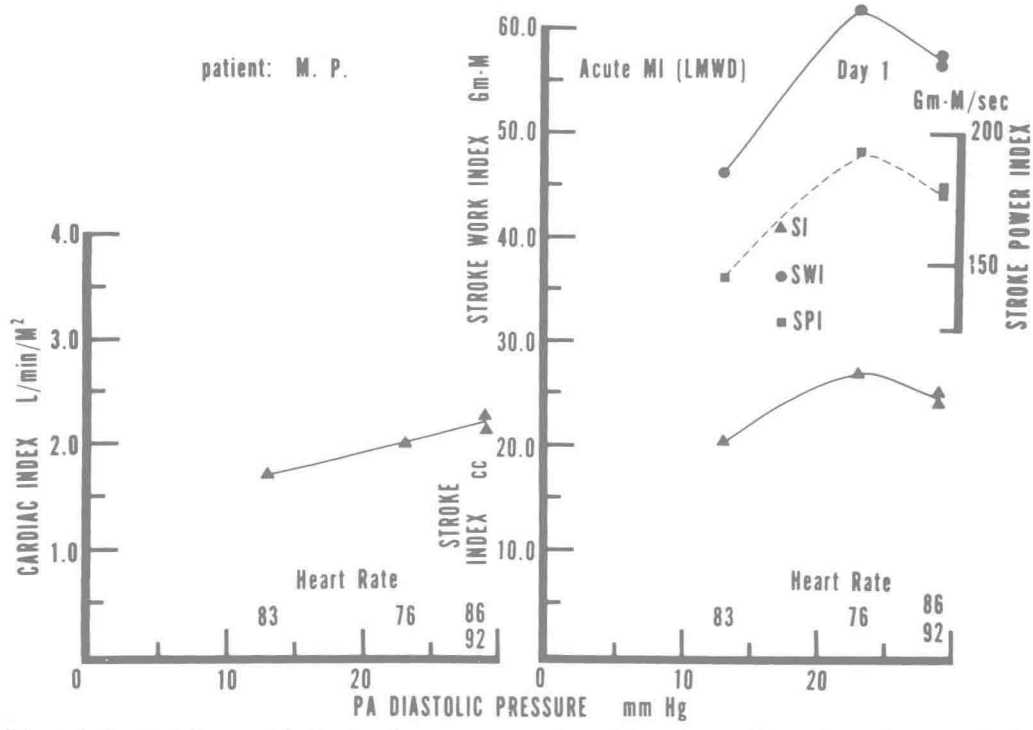

Figure 1. On the left, a ventricular function curve is constructed by relating changes in cardiac index to the pulmonary artery (PA) diastolic pressure after serial increments of dextran infusion on day 1 after acute myocardial infarction. On the right, function curves are constructed by relating the stroke index (SI), the stroke work index (SWI), and the stroke power index (SPI) to the pulmonary artery diastolic pressure. There is an initial ascent to the function curve followed by the descending limb the first day after acute myocardial infarction. (From Rackley and Russell,[8] with the permission of the American Heart Association, Inc.)

the slope and contour of the left ventricular function curve. In addition to hemodynamic measurements of the left ventricular filling pressure and cardiac output, systemic blood pressure can be recorded noninvasively or with intra-arterial pressure monitoring. Finally, the heart rate can be monitored from standard leads of the electrocardiogram.

From studies on the isolated papillary muscle, expressions of preload, afterload, contractile states, and frequency of stimulation were found to characterize both mechanical performance of the isolated muscle as well as the oxygen consumption.[9] In the intact left ventricle, preload is equated to the left ventricular filling pressure inasmuch as the diastolic volume and wall thickness do not significantly change in acute situations. Afterload, or the force resisting shortening of the myocardium, is equated to the systemic arterial pressure. The contractile state, which determines the force and velocity of myocardial fiber shortening, can be assessed by the slope of the ventricular function curve. The heart rate can be monitored from the electrocardiogram. Thus, the Swan-Ganz catheter provides measurements of the preload and contractile state and, along with the systemic arterial pressure and heart rate, the major determinants of mechanical performance and oxygen consumption of the intact left ventricle can be measured and monitored in patients with acute infarction.

INITIAL HEMODYNAMIC MEASUREMENTS IN ACUTE MYOCARDIAL INFARCTION

The clinical classification by Killip and Kimball[1] documented a low mortality in patients without clinical evidence of heart failure. Such patients presenting without hypotension and without clinical signs of left ventricular failure were designated as uncomplicated and experienced a lower mortality than patients who developed signs of either heart failure or shock during the acute phase of myocardial infarction. In Figure 2, initial measurements of the PAEDP and cardiac index are illustrated in patients with uncomplicated acute myocardial infarction.[10] These patients usually present with their first myocardial infarction, normal blood pressure, clear lungs, no audible ventricular gallop or pulmonary congestion by chest x-ray examination. As illustrated in Figure 2, approximately 80 percent of the patients in the uncomplicated category exhibited abnormal elevations of the PAEDP, and 20 percent of the patients revealed significant reduction of the cardiac index. Thus, the majority of patients without clinical signs of heart failure demonstrated abnormal disturbances in the sensitive hemodynamic measurements of the PAEDP and the cardiac index.

Patients with clinical evidence of heart failure experience a higher hospital mortality than those without such clinical findings. These patients comprise Killip classes II and III, exhibiting mild heart failure or pulmonary edema, respectively. In both categories the protodiastolic or ventricular gallop is required for this clinical designation. In Figure 3, measurements of the PAEDP or left ventricular pressure are shown in patients with acute infarction with and without an audible ventricular gallop.[11] In the patients without an audible ventricular gallop, left ventricular filling pressure was normal or mildly, moderately, or severely elevated. Thus, in the absence of an audible S3 gallop, patients may have a normal or abnormal left ventricular filling pressure. In those patients with an audible ventricular gallop, 25 of 27 individuals had filling pressures above the normal range of 12 mm Hg. The extent of the elevation was mild, moderate, or severe, ranging from 13 to almost 60 mm Hg. Thus, an audible ventricular gallop in the patient with acute infarction is not only a feature of left ventricular failure but also indicates abnormal elevation of the left ventricular end-diastolic pressure, which can range from mild to moderate or severe.

The construction of ventricular function curves in acute myocardial infarction by

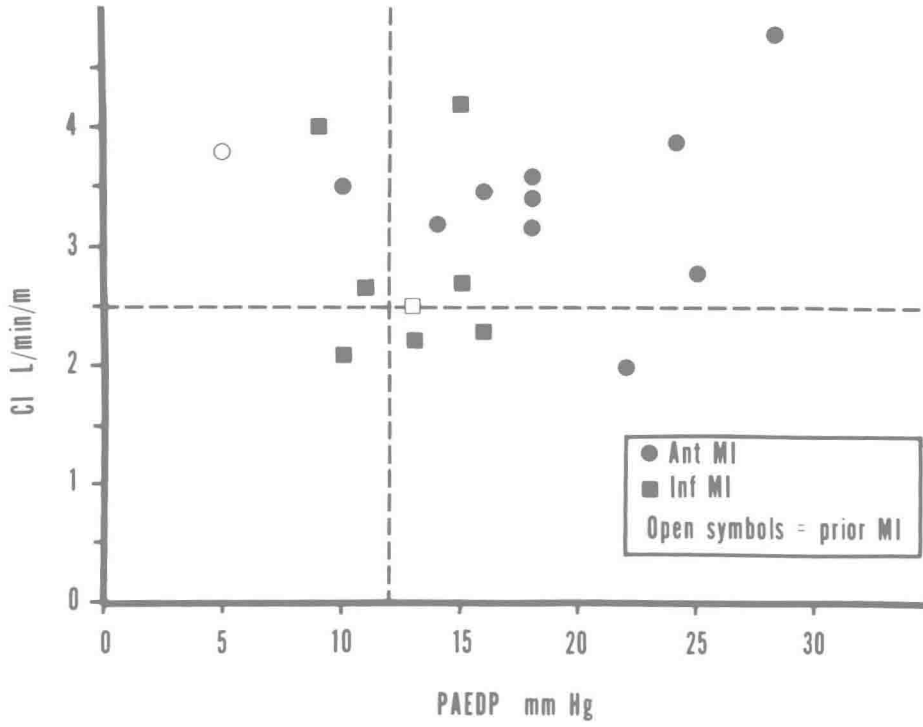

Figure 2. The initial hemodynamic measurements of the pulmonary artery end-diastolic pressure (PAEDP) and the cardiac index (CI) are shown in patients studied within the first 12 hours of acute infarction without pulmonary rales, ventricular gallop, or hypotension. The normal PAEDP is 12 mm Hg or less, and the normal CI greater than 2.5 l/min/m^2. (From Rackley, Russell, Mantle, et al,[10] with permission.)

relating serial changes of the left ventricular filling pressure to alterations in cardiac index or stroke index is shown in Figure 4. The function curves reached a peak or plateau ranging from 20 to 24 mm Hg.[12] Above 24 mm Hg, several of the curves displayed the descending limb of the ventricular function curve. In patients with acute myocardial infarction, the left ventricular filling pressure or preload can be raised above the normal limits of 12 mm Hg to 20 to 24 mm Hg in order to achieve the optimal mechanical performance of the ventricle. Even though the filling pressure can be raised above 24 mm Hg, patients frequently developed symptoms of dyspnea and audible rales in the lungs indicative of pulmonary congestion and failure. Thus, the slope of the ventricular function curve indicates the reserve of the infarcted left ventricle and also provides a basis for regulating the preload within the narrow optimal range of 20 to 24 mm Hg.

PHARMACOLOGIC AGENTS AND HEMODYNAMIC CHANGES IN ACUTE INFARCTION

Preload

Inasmuch as an optimal range of the left ventricular filling pressure or preload in acute infarction has been identified by hemodynamic measurements, the preload can be increased by expansion of the blood volume with dextran or can be reduced with