TWENTY-FIRST HAHNEMANN SYMPOSIUM

Emergency Medical Management

Edited by STANLEY SPITZER, M.D.
and WILBUR W. OAKS, M.D.

Under the general editorship of
JOHN H. MOYER, M.D.

GRUNE & STRATTON

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Preface

The Symposium on Emergency Medical Care sponsored by the Department of Medicine, Hahnemann Medical College and Hospital, was designed to review current methods and innovations. Outstanding authorities were brought together to discuss cardiovascular, pulmonary, endocrine, electrolyte, neurologic, obstetrical, pediatric, surgical and psychiatric emergencies.

With the emergency room coming more and more into focus as a primary care unit, and the development of critical care units such as the coronary and the pulmonary ones, a specific type of physician is developing who is concerned with these areas. We felt that this symposium would provide an overview, and would be an auspicious forerunner of subsequent symposia relating to emergency medical care.

The symposium also stressed the team approach. The cardiologist and the thoracic surgeon, the neurologist and the neurosurgeon, the general internist and the general surgeon are all brought together in the topics discussed. To further delineate the team approach, a concomitant symposium was held for nurses involved or interested in the varied fields of emergency medical care. Certainly the handling of any emergency situation is incomplete without the nurse. Ofttimes it is her response in the management of a patient that will make the difference between a therapeutic success and a therapeutic failure.

In this era the necessity and desirability of providing excellent medical care to the greatest possible number of people are self-evident. The life-or-death threat inherent in an emergency is overriding. The Department of Medicine believes, with other physicians, that much more emphasis should be placed on the diagnosis and management of emergencies. This symposium was not confined to practitioners engaged in emergency care in the emergency room and the categorical units within the hospital; to the contrary, the material presented will be useful to the nurse and to any physician who in his busy clinical practice will encounter diverse emergencies from time to time.

Future symposia will deal with medical care specifically related to the emergency room, and with critical care medicine in the institution and its categorical units.

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WILBUR W. OAKS, M.D.

Philadelphia, Pa.

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I. CARDIOVASCULAR EMERGENCIES

Hemodynamics and Pathophysiology of Rapidly Developing Cardiac Failure

By DOUGLAS M. GRIGGS, JR., M.D.
AND JOHN S. BAUMBER, M.D., PH.D.

RAPIDLY DEVELOPING CARDIAC FAILURE may occur in a number of pathological states, so that no single description of its hemodynamics or pathophysiology is possible. It is possible, however, to classify the underlying pathological conditions which are known to lead to rapid heart failure according to several major types of functional alteration they produce in the heart. Such a classification is shown in Table 1. Three categories, *volume overload, pressure overload,* and *direct myocardial depression,* are listed as the major functional alterations, and a fourth category, designated as *extramyocardial failure,* is listed to identify conditions which cause an obstruction to the flow of blood through the cardiac chambers without producing a concomitant effect on the ventricular myocardium.

Volume overload denotes the condition in which diastolic filling of the ventricle is increased beyond normal limits. This may result from such abnormal states as (1) rupture of the semilunar or atrioventricular valves or supporting structures which produces an acute regurgitation of blood ejected from the ventricle, (2) hypervolemia due to excessive intravenous fluid administration, or (3) arteriovenous fistulas which cause rapid venous return.

Pressure overload denotes the condition in which the generation of pressure by the ventricle during systole is greater than normal because of an increased resistance to blood flow in the aorta or the outflow tract of the ventricle. Conditions which may cause this include hypertensive crisis, extrinsic pressure on the great vessels due to expanding lesions, and a major embolism. Also listed in this category is myocardial infarction, since maintenance of a normal ventricular pressure exerts an increased pressure load on the remaining viable myocardium.

Direct myocardial depression designates a reduction in the functional capacity of the ventricle due to intrinsic abnormalities. This encompasses conditions causing an ineffective ventricular contraction because of reduced contractility of the individual myofibrils or asynchronous excitation of multiple myofibrils.

In the category of extramyocardial failure are included atrioventricular valve obstruction, such as pure mitral stenosis, and rapid accumulation of blood or fluid in the pericardial space of sufficient quantity to cause cardiac tamponade. No

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TABLE 1.—*Functional Classification of Underlying Causes of Rapidly Developing Cardiac Failure*

I.	Volume Overload
	A. Acute Regurgitation
	B. Hypervolemia
	C. Arteriovenous Shunts
II.	Pressure Overload
	A. Hypertensive Crisis
	B. Myocardial Infarction
	C. Outflow Tract Obstruction
	D. Pulmonary Embolism
III.	Direct Myocardial Depression
	A. Myocarditis
	B. Depressant Agents
	C. Hypoxia
	D. Metabolic Defects
	E. Conduction System Disorders
	F. Chronic Volume or Pressure Overload
IV.	Extramyocardial Failure
	A. Atrioventricular Valve Obstruction
	B. Cardiac Tamponade

direct effect on the ventricular myocardium is produced by these lesions, and thus the circulatory impairment is not related to a limitation in myocardial function.

This classification does not preclude the possibility that more than one functional alteration may exist in the same heart. For example, a consequence of acute depression of the left ventricular myocardium is papillary muscle dysfunction and mitral valve dilatation, both of which can cause mitral regurgitation. Chronic volume or pressure overloading of the ventricle not infrequently leads to acute myocardial depression, particularly if the metabolic demands of the peripheral tissues are increased by disease or other forms of stress or if metabolic acidosis ensues from a borderline cardiac output. From this it is apparent that individual variations in the underlying causes of rapidly developing cardiac failure are almost infinite. No attempt has been made to present an exhaustive list of single or interrelated causes.

In the course of studies on myocardial metabolism in our laboratory, we have induced rapidly developing cardiac failure in the open-chest dog by partially constricting the main left coronary artery. The hemodynamic changes we have observed are similar to those occurring in patients with direct myocardial depression of acute onset. The results obtained in one such animal experiment are shown in Figure 1. This is an illustration of serial tracings showing aortic blood flow, aortic pressure, and left ventricular end-diastolic (LVED) pressure at intervals following a 35 per cent reduction in main left coronary blood flow. The important features to be noted include (1) a progressive increase in ventricular end-diastolic pressure, which reached 27 mm Hg; (2) a decline in the rate of rise of left ventricular pressure during early systole, here shown as a decrease in the slope of a line drawn through the ascending portion of the ventricular pressure curve; and (3) a decrease in aortic flow, secondary to a decrease in cardiac output. These

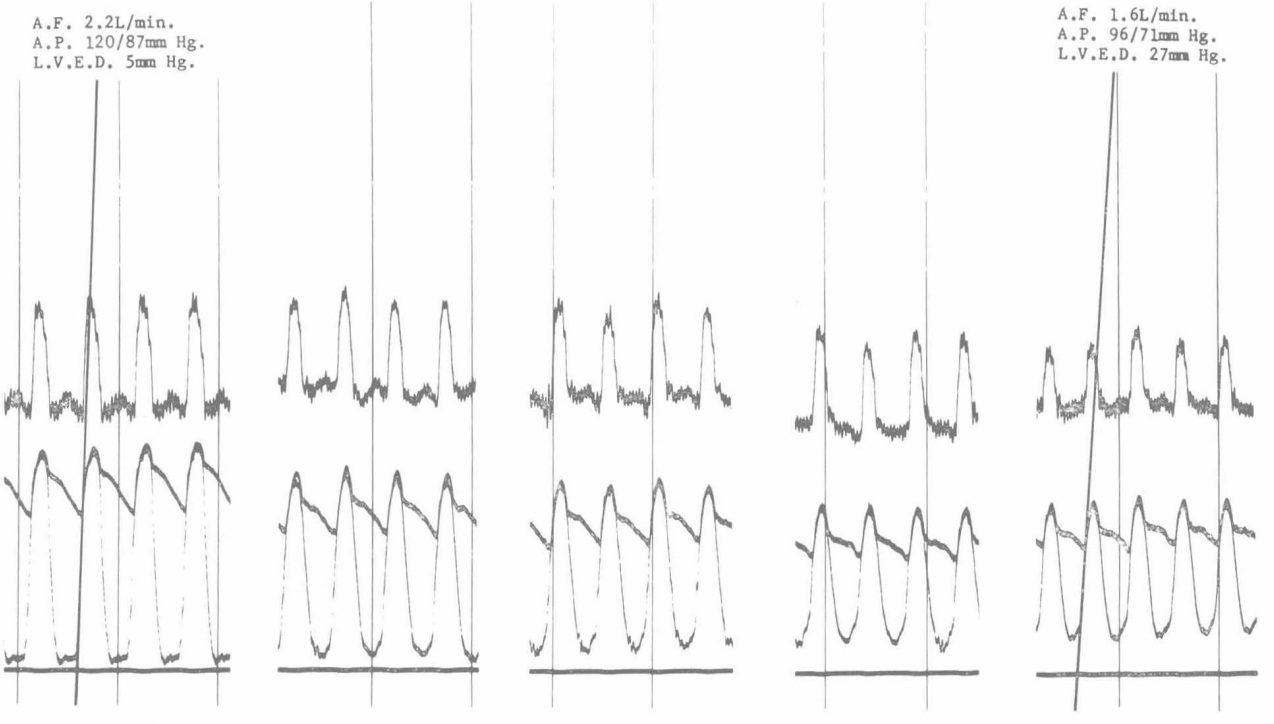

FIG. 1.—Records from animal experiment showing changes in aortic blood flow (*top signal*), aortic pressure and left ventricular pressure over a 10-minute period following acute reduction of left coronary blood flow. Also shown is a decrease in the slope of the left ventricular pressure rise. Time lines equal 1 second.

three hemodynamic findings form a classical triad of myocardial insufficiency of the low-output type.

Also apparent in these tracings is a progressive decline in aortic pressure. This is not an invariable finding in cardiac failure, but occurs when the fall in cardiac output is exceptionally rapid or profound. Maintenance of a relatively normal blood pressure in the face of a declining cardiac output is possible under many circumstances because of a compensatory rise in peripheral vascular resistance. The mechanism for this is diagramed in Figure 2. A decline in cardiac output initiates a decline in aortic pressure, since one is a direct function of the other according to the simple equation: Cardiac output = aortic pressure/peripheral resistance. A drop in aortic pressure is sensed by the baroreceptors in the systemic circulation. These include the carotid sinus and aortic arch receptors. The afferent nerve traffic to the vasomotor center is reduced as a consequence of the reduced baroreceptor stimulation. Parasympathetic inhibition of the sympathetic nervous system is thereby released in the body, and peripheral vascular resistance increases. This is a negative feedback system, so that for a declining cardiac output there is a tendency for continuous self-correction of aortic pressure. When the drop in cardiac output is exceptionally rapid or profound, the response rate or capacity of the blood pressure regulating mechanism is exceeded.

An important consequence of the changes in peripheral vascular resistance is a redistribution of the cardiac output. The increase in resistance is more profound in some vascular beds than in others, resulting in changes in regional blood

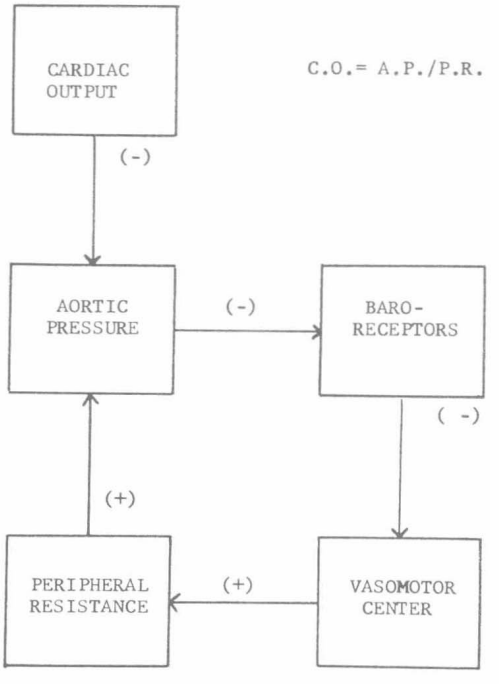

FIG. 2.—Diagram of negative feedback system for maintenance of normal blood pressure in the face of declining cardiac output.

flow to the various organs. Blood flow is shunted away from the kidneys, splanchnic organs, and skin in favor of the brain, heart, and other vital areas.

Having described the major functional alterations of the heart produced by pathological lesions and the hemodynamic changes which occur in rapidly developing cardiac failure, we shall now relate them more directly to the mechanical behavior of the heart as a pump. To do this, it is necessary to review certain basic principles of myocardial mechanics.

There are two fundamental laws governing the behavior of cardiac muscle. One is the length-tension relationship (Fig. 3), which states that increasing the resting length of a cardiac muscle fiber increases the amount of tension it will develop during an isometric contraction. There is, however, a certain optimal length beyond which the amount of tension developed is reduced for further increments in resting length. This law, applied to the intact ventricle, states that the stroke volume will increase in relation to an increase in end-diastolic volume of the ventricle. The original observations of this law were reported by Starling, and it bears his name. The other fundamental law governing the behavior of cardiac muscle is the force-velocity relationship, which states that the force developed by a contracting muscle fiber is inversely related to the speed at which it shortens. Stated another way, the more force the muscle has to develop against a resistance, the slower it shortens. This law also governs the behavior of skeletal muscle. One may recall how much slower the arm muscles lift a heavy object than a light one. In recent years studies on the heart have shown that the two laws can be combined into a single law called the force-velocity-length relationship.[1, 2] The manner in which the heart attempts to compensate for a sudden volume or pressure overload or for direct myocardial depression can be explained within the context of this relationship.

A schematic representation of myocardial function regulation based on the force-velocity-length relationship is shown in Figure 4. The two most important physiologic variables which regulate the force of ventricular contraction are the

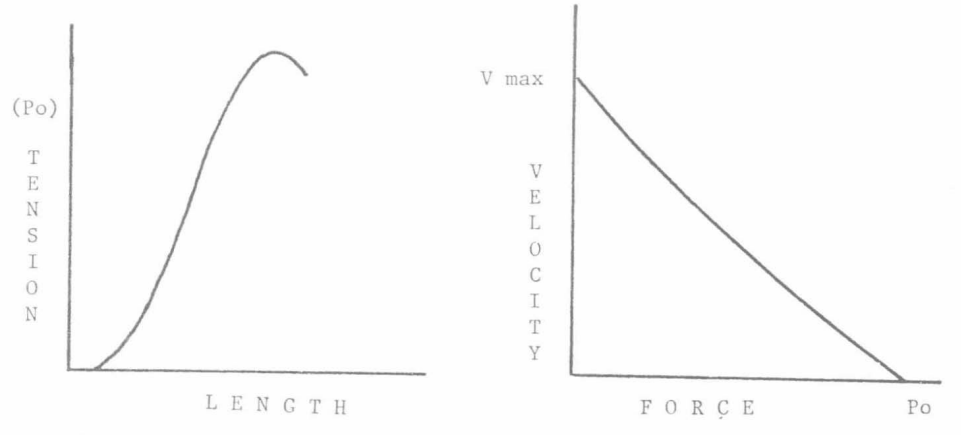

FIG. 3.—Diagrams of length-active tension relation (*left*) and force-velocity relation (*right*) of cardiac muscle. Po=force developed during isometric contraction; V_{max}= theoretical maximum rate of shortening of contractile element.

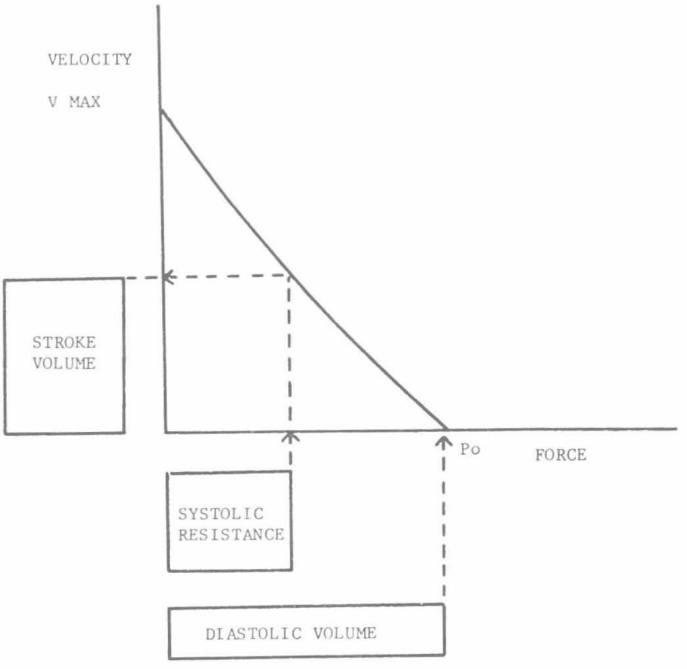

FIG. 4.—Schematic representation of relationships among diastolic volume, systolic resistance, and stroke volume of the ventricle according to the dictates of the force-velocity-length relationship of cardiac muscle.

diastolic volume, which presets the fiber length before contraction, and the resistance to ejection met by the ventricle after systole begins. These two variables are often referred to as the preload and the afterload, respectively. The systolic resistance is primarily related to aortic blood pressure except under conditions where there is significant outflow obstruction, in which case it is related to the size of the opening into the aorta. In a general way, the volume of blood ejected for any contraction is dependent upon the systolic resistance according to the dictates of the force-velocity relationship, since the decrease in ventricular chamber size is dependent upon the rate of myocardial fiber shortening.

Figure 5 illustrates how an increase in diastolic volume shifts the force-velocity relationship to a new position because of an increase in myocardial fiber length, so that for the same systolic resistance the ejected volume is greater. Another important mechanism (Fig. 6) by which the ejected volume is increased or decreased is a change in the intrinsic contractility or inotropic state of the myocardium, such as occurs from adrenergic stimulation or the administration of digitalis or beta-acting catecholamines. The primary effect on the behavior of the myocardium is an increase in the maximal attainable velocity of contraction,[3] designated by the term V_{max}, which results in a shift of the force-velocity curve to a higher point on the velocity axis.

Consideration can now be given to the influence on myocardial mechanics of the previously described functional alterations associated with rapidly developing cardiac failure. Figure 7 represents a hypothetical case of acute valvular insuffi-