THE ACUTE CARDIAC EMERGENCY

Diagnosis and Management

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Formal papers and selected edited discussion drawn from the program sponsored by the AMERICAN COLLEGE OF CARDIOLOGY at Ponte Vedra, Florida.

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A *Symposia Specialists* Book

Published by

FUTURA PUBLISHING COMPANY, INC.

Mount Kisco, New York 10549 U.S.A.

Acknowledgments

In the preparation of this book, the editor would like to thank the excellent contributing authors. I am particularly grateful to the associate editors, Drs. C. G. Chaplin, James W. Holsinger, and Vincent F. Miscia. The organization of the original seminar and assistance of the American College of Cardiology, primarily Miss Mary Anne McInerny, Dr. John Bish, and Mr. William Nelligan, were signal among the major administrative contributors to the success of the program and subsequently the book. The preparation of manuscripts, the organization, invitations, and arrangements for contributing editors, review of manuscripts, etc., was singlehandedly and outstandingly maintained and directed by Mrs. Mary Ann Cobb, whose energy, balance, and accuracy was invaluable. We are deeply indebted to Mr. George Cave and Mr. William Dean for their added efforts and expertise in medical art and photography. I would also like to recognize the assistance of Mr. Cooper Schamblen.

R. S. E.

Foreword

At this point in the history of the affluent countries of the world and the United States in particular, the major health risk is heart attack in the form of sudden death or myocardial infarction. The fundamental understanding of heart attack is improving and the goal of such understanding is prevention. Clinical medicine, however, is not yet at the practical stage of prevention. Instead, we must continue to put out fires after they have started. Indeed, ironically, the community Fire Departments are helping us to do this by competently delivering "pre-hospitally" managed patients to well-prepared emergency receiving centers. We are, thus, attempting to extend the lives of individuals who would be otherwise cut down in their most productive years. Prevention awaits the funding, the research and the understanding which comes from it. Until then we will have to address ourselves to methods of salvaging the lives of individuals who have hearts that are "too good to die."

This book compiles practical information which the physician requires in the detection and management of victims of the acute cardiopulmonary emergency. Realistic problems are defined, priorities in the diagnosis and management of these conditions identified, and useful techniques, management, and therapy outlined. In addition, prevention and community management is reviewed. To highlight specific aspects of management and therapy, a series of panel and open discussions respond to pertinent questions. To implement the practical aspects of the management of cardiac emergencies, an outstanding group of established experts in various fields have contributed their respective contemporary practical approaches to this book. It is hoped that it will serve as a readable, comprehensive, and practical guide to the diagnosis and contemporary management of patients who present with "the acute cardiac emergency."

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I BACKGROUND

The Pathophysiology of Sudden Death and Myocardial Infarction

Robert S. Eliot, M.D. and James W. Holsinger, Jr., M.D., Ph.D.

Acute coronary thrombosis is an unusual event occurring in less than 20% of cases.[1] The question then is whether the plumber's concept of arterial blockage causing myocardial ischemia and necrosis which is so simplistic is tenable. One can easily visualize the sudden blockage of a coronary artery halting the delivery of blood to the myocardium causing necrosis. This is such a simple and seductive concept that we have retained it for many years. Except this is usually not the case. The persistence of this concept represents an intellectual block to our understanding of the mechanisms of infarction and sudden death.

So one may ask: "If there's no thrombus, there must be something that plugs the artery. An alternate possibility is hemorrhage into an atheromatous plaque." This is one of the last bastions of retreat for "the plumbers." But if this were a cause of myocardial infarction or sudden death, several things should be borne in mind. Small adventitial vasa vasora are the sites at which hemorrhage allegedly takes place dislodging a firm, rigid, atheromatous plaque and thus extruding it into the lumen with resultant obstruction. There is a reasonable question whether these small vasa vasora are large enough to really do the job. It is a David and Goliath phenomenon – the tiny vessels rupturing and their contents pushing the large plaque away from the wall. The original stimulus for this event is not clear. And indeed, if this actually happened, we should find cholesterol crystals distributed in the myocardium distal to the point of extrusion as mute testimony to the fact that this did occur. But, such evidence cannot be found to confirm this particular phenomenon.

The majority of myocardial infarctions causing death do occur to patients whose arteries are diseased but patent.[2] So it remains difficult to explain myocardial infarction on an arterial obstruction basis in the presence of diseased, though patent, coronary arteries, and equally the absence of myocardial

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infarction in those individuals who have major obstruction of the coronary blood flow. Of the people who die of causes other than cardiovascular disease, 44% will have one or more major arterial occlusions.[2] On the reverse side of the coin, Drs. Baroldi and Eliot have studied the hearts at autopsy of nine patients with patent coronary arteries yet demonstrating one or more myocardial infarctions.[3] The most severe disease found in any of these hearts was a single instance of 30% occlusion of one coronary artery, most vessels being less than 10% obstructed. This information is pertinent at this point, since there remains some question among observers whether myocardial infarction can occur in this circumstance. Although uncommon, it does occur.

Basically, there are two types of myocardial infarction, transmural and subendocardial. As we better define terms, let us return to the acceptable generic term "heart attack." Heart attack should encompass myocardial infarction – subendocardial and transmural – and it should also include sudden death of apparent cardiac origin. Approximately 90% of individuals who have transmural myocardial infarction will have a major arterial obstruction, while subendocardial infarction usually occurs in the presence of diseased though patent, coronary arteries.[1] Such obstructions may be new or old. The subendocardium is almost always involved in all types of myocardial infarction.

We look on patients who have myocardial ischemia, necrosis, or sudden death with three factors in mind. First, we are concerned with the state of the arteries. We also look at the condition of the blood oxygen delivery vehicle, often overlooked and yet significant in that it carries oxygen to the heart and the efficiency in which it unloads it is important to myocardial oxygenation. The third factor is the level of myocardial oxygen demand.

One of the reasons the plumber's concept of myocardial ischemia cannot be accepted as an exclusive explanation is that, fundamentally, the heart is not an endarterial system (Fig. 1). The veins cannot be individually obstructed with resulting necrosis because there are too many avenues. As Dr. Baroldi has pointed out, the easiest way to develop collateral circulation is to develop coronary stenosis. The collaterals are already there; they simply enlarge. Coronary stenosis is not sudden, but gradual in development. What we see is the gradual arteriosclerotic narrowing of the coronary arteries and along with this the development of the collateral circulation.

In sickle cell disease, there is no histologic evidence of myocardial infarction.[4] Instead, there is the development of a tremendous number of collateral vessels which apparently help the myocardium to make up for the absence of blood. Likewise, Dr. Baroldi's[5] studies of 39 thrombotic thrombocytopenic purpura patients with major obstructions in small arteries scattered throughout the entire system showed nothing that resembled myocardial infarction. At least in the experiments of nature, it is difficult to support the concept of diffuse small artery disease as a cause of myocardial ischemia and necrosis.

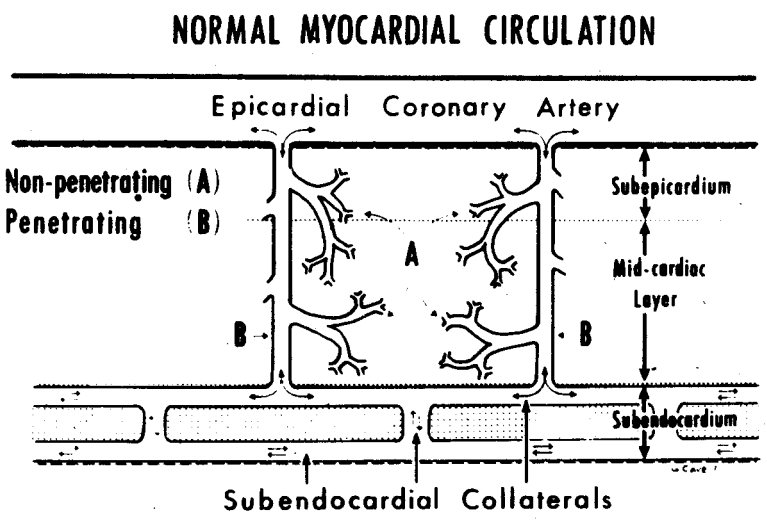

Fig. 1. Normal myocardial circulation. (A) Non-penetrating coronary arteries. (B) Penetrating coronary arteries. (Arrows) Demonstrate real and potential collateral flow. (Reprinted by permission, R. S. Eliot and J. W. Holsinger, Jr.: The Pathophysiologic Panorama of Myocardial Ischemia and Infarction. In: *Myocardial Infarction: A New Look at an Old Subject.* ed. by J. H. K. Vogel, S. Karger, Basel, In press.)

If the heart and the kidneys are compared, weighing 300 gm each, they are entirely different organs; 5% of cardiac output perfuses the heart, 23% the kidneys; yet the heart's oxygen demand is 11.4 volumes per cent, and the kidneys', only 1.4 volumes per cent. So the heart has the highest oxygen demands of any organ in the body and probably has the slowest, poorest flow. It must extract a great deal of oxygen per unit flow in order to survive in aerobic metabolism.

We may question why such a vital organ was designed with such a poor fail-safe system in terms of flow and oxygen demand. There are so many variables in myocardial oxygenation that can change – coronary flow, blood oxygen release, and myocardial oxygen demand can produce at least 27 possible combinations that can be beneficial or detrimental. Among them are the size of the coronary arteries at birth, the ratio and number of capillaries, the flow regulators in the arteries, and the flexibility of these arteries. Another important factor is the state of the blood oxygen delivery vehicles – whether there is anemia, carbon monoxide, hemoglobinopathy, pH change, or other conditions that influence myocardial oxygenation. There is also the level of myocardial oxygen demand: fiber length, strength of contraction, rate of contraction, degree of oxygen wastage. Then there are local metabolic factors – whether the oxygen molecule can be utilized – myoglobin, cytochromes, tissue pH, local

temperature, hypertrophy, and hypermetabolism. There are many, many factors that are purely myocardial.

The design is such that a tremendous pressure gradient exists from the inner surface to the outer surface of the left ventricle.[6] The inner surface of the left ventricle is under greater tension all of the time, whether it is in diastole or systole. Mathematically, the inner lining of the heart is under the most stress,[7] and the longest sarcomere is in the subendocardium.[8] All this results in more oxygen consumption per contraction. The heart is also designed so that it can shut off its own blood supply with the result that in systole the coronary arteries are not perfused at all. Coronary sinus flow goes up as the sponge-like systolic squeezing of the heart produces a flow into the coronary sinus. Left ventricular pressure is high – so high that subendocardial resistance is elevated to the point where perfusion doesn't take place.[9] The faster the heart rate, the poorer it perfuses itself – within limits, of course. There is a redistribution of blood flow every time the heart contracts so that the subendocardium either is underperfused or redistributes its blood to the subepicardium. The subendocardium also has a greater capillary-to-fiber ratio than the subepicardium, yet these capillaries are at 94% of maximal utilization at rest – leaving little reserve. Little surprise then that the tissue partial pressure of oxygen is lowest in the subendocardium of all cardiac layers or for that matter in the body.

Since the subendocardium is almost always involved in the process of myocardial infarction, the earliest evidence of necrosis can be found in this layer. Consequently, it may be that the initial site of myocardial infarction resides in this layer of the heart. In fact, it may be possible to relate the morphologic forms of this condition to initial subendocardial necrosis. It is quite difficult to investigate the early effects of ischemia on the subendocardium due to its inaccessibility in both human and experimental animals. The basic assumption of the hypothesis to be outlined below is that the mechanism for initial subendocardial necrosis parallels observations following protracted periods of ischemia on skeletal muscle of animals. There are a number of observations which demonstrate that the skeletal muscles are incapable of sustaining reflow after a protracted period of ischemia following release of a limb tourniquet.[10-19] This observed inability to reinstitute flow may be due to a variety of phenomena (Fig. 2) which include among others swelling of perivascular tissue, swelling of endothelial cells, and the formation of intravascular blebs arising from the endothelium, all of which impinge upon the microvasculature of the subendocardium.

The basic assumption that must be made here is that obstruction similar to that for skeletal muscle may indeed occur in the ischemic subendocardium with the resulting inability to reinstitute blood flow at the microcirculatory level. Such failure to reinstitute flow into ischemic areas of the subendocardium results from the formation of an endarterial system which is not normally

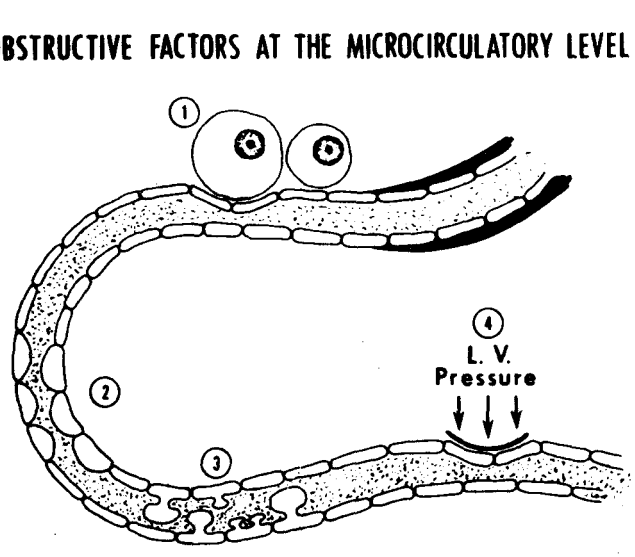

Fig. 2. Obstructive factors at the microcirculatory level: (1) Swelling of perivascular tissue; (2) Swelling of endothelial cells; (3) Endothelial blebs; (4) Compressive left ventricular pressure. (Reprinted by permission, S. Karger, Basel.)

present. The formation of such an endarterial system could cause the subendocardium, or a portion of it, to be much more vulnerable to infarction if the process of ischemia continues. Consequently, blood flow is compromised into and out of the ischemic myocardial region undergoing early necrotic change (Fig. 3). Such a decrease in the cross sectional area of the collaterals would be affected by an increase in coronary arterial resistance which would potentially impede coronary flow.

The expansion of such a subendocardial infarction would then depend, in part, upon the status of the major arterial supply to the subendocardium and/or the continuation of inordinate myocardial work during the period of necrosis. The first is determined by the degree of arterial obstruction which pre-exists (Fig. 4) at the time the subendocardial necrosis takes place or which develops later. The continuation of inordinate myocardial work occurs due to such pre-existing conditions as hypertension or such individualized conditions as the denial of pain and the continuation of effort in the presence of early initial subendocardial necrosis. Thus, there would be a predisposition to transmural myocardial infarction in such an individual in the area subserved by the obstructed major epicardial coronary artery. Also, in such a condition hemorrhage into an atheromatous plaque or thrombosis may develop secondarily, again setting the stage for transmural myocardial infarction. Should the patency of the collateral circulation between adjacent subendocardial