Mysteries of Sudden Death and Myocardial Infarction

Death of cardiac origin results from electrical or mechanical faults. "Mysterious sudden death" can be more clearly understood as deranged electrical stability of the heart. The basis for such instability ranges from congenital deformity of the conduction system to pharmacologic misadventure. James has unraveled and reviewed the interrelationships between form and function of the conduction system. Included are mechanisms by which the heart maintains its stable rhythm and conduction, those structural abnormalities which alter function, the role of the circulation in conduction abnormalities, and humoral influences. This comprehensive synthesis assembles critical pathophysiologic links between anatomy, pathology, physiology, pharmacology, and the electrical events. When viewed from this perspective, mechanisms for mysterious sudden death loom largely electrical.

Mechanical faults were reviewed by Eliot and Holsinger who discussed the pathologic features of hearts from victims of sudden death and myocardial infarction. Victims of sudden death rarely display acute arterial obstruction. Of note also is the presence of infarction frequently independent of coronary arterial obstruction, and equally, coronary arterial obstruction without infarction. The incomplete correlation between arterial obstruction and myocardial infarction represents a stumbling block to pathophysiologic understanding. Well known is the separation between subendocardial and transmural infarction by the nearly complete absence of arterial obstruction in the former, contrasted with the ubiquitous presence of such obstruction in the latter. Also confounding to the observer is delay of histologic evidence of myocardial necrosis for from four to six hours from the onset of symptoms of myocardial infarction.

Uniform among the pathologic features is subendocardial necrosis in either subendocardial or transmural infarction. As reviewed elsewhere, clusters of observations testify to inordinate subendocardial vulnerability. Furthermore, this layer, in our experience, demonstrates the earliest recognized histologic or chemical features of necrosis. Thus, it is proposed by Holsinger and Eliot that myocardial infarction may begin with coagulation necrosis in the subendocardium independent of acute arterial obstruction. Its limitation to this area or its extension to involve the transmural regions would relate primarily to the presence of preexistent arterial obstruction or to the advent of a new occlusion. Important also would be the continuation of myocardial work in the face of decreased compliance and altered contractility of the heart with subendocardial necrosis. The potential for secondary arterial obstruction relates to the diminution or cessation of blood flow into the involved subendocardial area of microcirculatory collapse. Thus, the much discussed thrombus might more logically be understood in this circumstance as a secondary phenomenon.

Although admittedly unproved, such a hypothesis correlates gross and histologic features in sudden death and myocardial infarction. Further, in the living patient with preinfarction angina, vein bypass surgery has suggested that adequate perfusion...
with the new vein implant can occur prior to subendocardial necrosis.19 After subendocardial necrosis develops, however, there is diminished native circulation to the involved area and vein implantation into the patent area serving the necrotic subendocardial region may be attended by poor nutritional flow due to the distal microcirculatory collapse or perhaps to myocardial “steal” syndrome.

Finally, electrical and mechanical events may blend their mutual potentials for disaster. For example, myocardial infarction can alter normal wall stress compressing the vulnerable adjacent conduction system. Electrical instability may then develop by direct mechanical pressure upon or interruption of the conduction system.13,14 The conduction system per se has a lower threshold for Po2 than does the myocardium.15 Hypoxia, then, would appear to be an unlikely solitary mechanism for altered conduction or electrical instability. Hypoxia, however, may increase sensitivity to inotropic agents or electrolyte changes.

Singly or in concert, electrical and mechanical cardiac catastrophes remain a frequent mode of exodus from the 20th century, and one of its major mysteries and challenges.

Robert S. Eliot, M.D., Omaha*

*Professor of Medicine and Director, Division of Cardiology, University of Nebraska Medical Center.

REFERENCES

8 Baroldi G, personal communication
14 Hastor JA, Spear JF, Moore EN: Localization of ventricular irritability by epicardial mapping: origin of digitalis induced ventricular tachycardia from left ventricular pурkinje tissue. Circ 45:952, 1972
15 Truex RC, Synthple MQ: Recent observations on the human cardiac conduction system with special considerations of the atrioventricular node and bundle. Electrophysiology of the Heart. Milan, Pergamon, 1964

Physician Assistant

In an effort to foster better communications between chest physicians and health care support personnel in the field of cardiopulmonary diseases, Chest has adopted a policy of simultaneous publication with Respiratory Care* of articles of special interest to both groups. Such an article on training of physician assistants appears in this issue (see page 732). Emphasis in the training program described is placed on technologic expertise in relation to cardiopulmonary patient care.

Lack of technologic expertise in diagnostic and therapeutic procedures often limits effective care of the cardiopulmonary patient in the smaller community medical facility. There is little hope that sufficient numbers of qualified physicians will be available in the near future. To meet this challenge, there is a desperate need to revise the character of medical student and house-staff training to provide a better level of expertise for more physicians who can function in the role of medical directors for allied health personnel. However, too few qualified teachers are available to provide this training.

I have proposed the development of a group of allied health workers, physician assistants, who could extend the potential of the specialist from the larger medical centers so that expert care might be brought to every patient. In this situation, the physician assistant, acting under the direction of the specialist, will serve as an aide to the primary physician in the outlying community facility. Audiovisual communication and computerized function evaluations between medical facilities will greatly enhance the efficiency and reliability of such a program.

The specialist also could increase his potential in

*Official publication of the American Association for Inhalation Therapy.