Clinical Consequences of Acute Pericarditis

Physicians must anticipate an amazing variety of acute, subacute, and chronic complications and sequelae in any patient with acute pericarditis. Elsewhere in Chest, three very instructive reports make solid contributions to our understanding of several of these complications and sequelae. In their article entitled “Pericarditis of Acute Myocardial Infarction” (see page 647), Toole and Silverman provide many much-needed numbers to characterize both the “epistenocardiac” pericarditis of acute infarction and the postmyocardial infarction syndrome (PMIS). In a comparative study they emphasize the similarity of the course and characteristics of transmural infarction with and without pericarditis, except for the greater frequency of anterior infarct with clinical pericarditis. While others have pointed out the paucity of auscultatory as compared with roentgenographic findings indicating pulmonary congestion during infarction, this work discloses a tendency to overdiagnose congestion when the patient has the constellation of pericardial and pleuro-pulmonary findings which eventuate in the PMIS. Although Toole and Silverman have somehow omitted electrocardiographic data on infarct plus pericarditis and have inexplicably chosen to exclude patients with monophasic pericardial rubs, their data indicate the importance of documenting the occurrence of the acute-phase pericarditis to help differentiate subsequent recurrent infarct, pulmonary embolism, and other chest syndromes.

In their article entitled “Acute Effusive-Constrictive Staphylococcal Pericarditis” (see page 721), Ehrich, Widmann, and Abelmann describe a unique hemodynamic study of a case fulfilling the criteria for acute constrictive pericarditis. This instructive report illustrates the occasional rapidity of onset of epicardial constriction and includes clinical and physiologic nuggets, such as the presence of a pericardial rub during an effusive-constrictive process, and the 26 mm Hg pulsus paradoxus (a degree which is characteristic of lobe tamponade) with Kussmaul’s sign (which is more characteristic of constriction). Odd findings in this patient include the presence of a dominant y-descent in the right atrial pressure curve despite compression by fluid, with the appearance of both x and y descents after paracentesis and the absence of an abnormal early diastolic sound after paracentesis unmasked the constriction.

Although uncommon, and only occasionally symptomatic, congenital and inflammatory pericardial cysts and diverticula can coexist with and confound clinical graphic signs in the diagnosis of coexistent cardiovascular abnormalities. The report entitled “Pericardial Cyst Ten Years after Acute Pericarditis” by Peterson, Zatz, and Popp (see page 719) adds to the already stupendous achievements of echocardiography (in which field Dr. Popp is a pioneer) the capacity to diagnose pericardial cysts without thoracotomy.

These articles point up the protean manifestations and consequences of pericardial afflictions, a disease spectrum replete with pitfalls and paradoxes requiring a wide acquaintance with clinical, physiologic, and graphic approaches. They set an example of expert diagnosis and management.

David H. Spodick, M.D., F.C.C.P. *
Boston

*Professor of Medicine, Tufts University School of Medicine, and Director, Cardiology Division, Lemuel Shattuck Hospital, Boston.

REFERENCES

4 Spodick DH: Chronic ond Constrictive Pericarditis. New York, Grune and Stratton, 1964, 322-327

Antiangina or Antimyocardial Ischemia Drugs

Proof of the effectiveness of an antianginal drug requires a convincing demonstration that the drug causes anginal distress to be relieved to the point of disappearance under circumstances in which the pain would persist if the drug were not given. Since angina is a subjective symptom appreciated in its various degrees of intensity only by the patient himself, the drug tester must rely upon the patient’s testimony as to whether anginal discomfort is or is not present and to what degree it is present under the conditions of the test. Evidence that most angina patients have a high degree of discrimination in evaluating the intensity of anginal pain is provided in a study by Smokler et al showing the high degree of reproducibility between duplicate treadmill exercise tests in a group of such patients; in this investigation, the test end point was a specifically defined level of symptomatic intensity of 3+ angina discomfort according to which 1+ was mild, 2+
Angina, however, implies more than a symptom; it is a manifestation of myocardial ischemia, a state which alters human physiology. The ischemic heart has an electrocardiographic hallmark, ST segment deviation. It also has a mechanical counterpart, reduced myocardial contractility with concomitant reduction of cardiac output. In most instances it also seems to give rise to reflex peripheral vasoconstriction, for a sharp rise of blood pressure in cardiac patients frequently heralds by a few moments the onset of anginal discomfort. On the other hand, the build up of anginal distress frequently is associated with a falling blood pressure as myocardial contractility becomes impaired and cardiac output falls.

This pattern of symptomatic, electrocardiographic and hemodynamic abnormality can be elicited with regularity by having the patient walk on a treadmill at work load titrated to allow the angina to build up slowly so that ECG pattern and blood pressure response can be correlated with the symptomatic level of pain intensity. There is thus time for observations of the effects of drug administration without the rapid build up of pain that would require cessation of the exercise.

A study published in this issue (see page 640) utilizes this method to demonstrate the anti-myocardial ischemia effects of nitroglycerin and of the chewable form of isosorbide dinitrate. Using the controlled challenge of carefully titrated treadmill exercise, the symptomatic, electrocardiographic and physiologic manifestations of myocardial ischemia were elicited and then reversed by the therapeutic agents during continuation of the angina-provoking walking work load. Both drugs effectively relieved angina, although the isosorbide dinitrate acted a little more slowly than nitroglycerin. In those cases in which the ECG could be interpreted in terms of an ischemic pattern, this pattern was reversed, in some instances completely and in other cases incompletely. The blood pressure changes were not as consistently predictable, probably because of the many other factors influencing the arterial pressure during exercise.

This study, then, demonstrates the effectiveness of two so-called antianginal drugs in relieving not only the pain and discomfort of angina pectoris, but also the physiologic consequences of myocardial ischemia. It is shown that ECG changes also regress and that the heart has contractility restored, as manifest by its capacity to sustain an increased systolic pressure \( \times \) heart rate product following the relief of angina.

The data suggest that antianginal drugs might better be thought of as anti-myocardial-ischemia agents for their range of usefulness may have far wider application than the mere relief of pain. It has been well documented that patients may demonstrate extensive electrocardiographic evidence of myocardial ischemia without experiencing angina pain. Such persons are also at high risk for the development of coronary events. It might well be that an anti-myocardial-ischemia drug would be of great benefit to such a patient, not to relieve pain but to protect him from myocardial ischemia leading possibly to a coronary event with a 50-50 chance of sudden death.

Albert A. Kattus, M.D.*
Los Angeles

*Professor of Medicine, UCLA School of Medicine, Los Angeles.

Reprint requests: Dr. Kattus, Department of Medicine, UCLA Medical Center, Los Angeles 90024

REFERENCES