ure is seen after acute myocardial infarction. Early use of IACP may prevent the development of severe coronary shock or may stabilize cardiac energetics in severe shock, facilitating subsequent surgical intervention. (*Mueller, H., and others: The Effects of Intra-aortic Counterpulsation on Cardiac Performance and Metabolism in Shock Associated with Acute Myocardial Infarction, J. Clin. Invest 50:1885, 1971.*) ABSTRACTER'S COMMENT: This is an elegant and daring approach involving the study of many variables under apparently well controlled conditions in patients who probably would have died in cardiogenic shock after infarction. Although it is a *tour de force*, two fundamental questions remain to be answered if we are to accept it for widespread use. First, does this complicated therapeutic approach represent an advance in the treatment of myocardial shock, and second, will it alter the natural history of the disease?

ANGINA AND CIRCULATORY OVERLOAD The hemodynamic effects of dextran infusion on the angina threshold elicited by atrial pacing were determined in 17 patients. Every patient was either thought to have coronary heart disease or known to have angina pectoris or a history of myocardial infarction. All gave informed consent to the study. At cardiac catheterization myocardial stress was elicited by atrial pacing, which was reapplied after infusion of low-molecular-weight dextran in 5 per cent glucose in water. Three groups were studied: patients with angina, those with coronary heart disease without angina, and normal subjects. The angina group (9 patients) responded to an average infusion volume of 300 ml (range 215 to 360 ml), given at an average rate of 29 ml/min, with an appreciably faster onset of anginal pain (average 1.8 min *vs.* 5 min for patients who were paced but did not receive the infusion). In addition, pain was reported as more severe, and in four cases angina was invoked by atrial pacing after the infusion had been given only. Three patients developed angina during the dextran infusion; one of these had been pain-free during the control pacing period. During postinfusion pacing, left ventricular end-diastolic pressure rose an average of 125 per cent, in contrast to a 10 per cent increase in the modified tension–time index. (*Khaja, F., and others: Effect of Volume Expansion on the Angina Threshold, Circulation 43:824, 1971.*) ABSTRACTER'S COMMENT: This paper complements other studies by these workers in which they described the relief of pacing-induced angina by phlebotomy as well as the role of nitroglycerin in diminishing left ventricular volume. The role of endogenous catecholamine release in pacing-induced angina is far from clear. The anesthetist managing the patient with severe coronary heart disease may have to readjust his sights in the evaluation of myocardial response to the stresses of the anesthetic drug and surgery. It has been assumed tacitly that an autonomic stimulus elicited during anesthesia with otherwise "myocardial depressant" drugs was beneficial. Perhaps the days of such "cavalier physiology" are over and we may have to consider the possibility that the "sick heart" seldom tolerates an increase in myocardial oxygen requirements without serious consequences. After all, how many anesthetists are concerned with the possibility that angina may occur while the patient is asleep? One should keep in mind that with severe coronary disease even a handshake may cause severe pain.