Editorial Comment

Cardiogenic Shock Complicating Myocardial Infarction*

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Despite more than 25 years of observation and treatment in the cardiac care unit, cardiogenic shock complicating myocardial infarction is the leading cause of death after hospital admission. Mortality remains high despite the widespread use of pressor agents, newer inotropic drugs and mechanical circulatory support (1).

The incidence of cardiogenic shock complicating myocardial infarction appears to have declined in recent years. Of 2,933 patients meeting MILIS study group criteria for presumed myocardial infarction, only 131 (4.5%) were in functional class IV (cardiogenic shock) on entry into the study (Moller JLS, personal communication). Trends toward earlier hospitalization of the symptomatic patient and, more recently, administration of thrombolytic agents may be contributing to the apparent decline. Additionally, the clinician avidly seeks treatable causes of circulatory collapse, including hypovolemia, acute mitral valve dysfunction, acquired ventricular septal defect and, occasionally, pericardial hemorrhagic tamponade or external rupture with pseudoaneurysm.

Pathologic studies (2,3) have established that cardiogenic shock develops when a major portion of the left ventricle—usually >40%—is dysfunctional because of ischemia or infarction. The loss of left ventricular mass may reflect extensive recent infarction, or some combination of one or more old infarct scars combined with fresh necrosis that exceeds a critical threshold of destruction. It is the total amount of left ventricular damage that counts, not when it occurred (2). The occasional survivor from massive infarction usually has severe functional limitations and a poor prognosis. A recent echocardiographic study (4) suggests that altered function of the noninfarcted myocardium may also contribute to the shock syndrome.

Current treatment remains unsatisfactory. Because the amount of dysfunctional myocardium appears to be the major factor contributing to the development of cardiogenic shock complicating myocardial infarction, treatment aimed at relieving coronary artery occlusion and restoring nutritional flow to the ischemic myocardium appears reasonable. Certainly, the evidence that treatment with vasodilators, pressors or inotropic agents improves survival has not been convincing. Mechanical support of the circulation has also had limited success (5). Emergency coronary bypass surgery has had its proponents, but reports (6) of success have been viewed with skepticism because of possibilities of selection bias. Thrombolytic agents may be effective, but definitive studies are lacking. More recently, reports (7) on the value of emergency angioplasty in cardiogenic shock have appeared. Although suggestive, such studies have lacked controls or have comprised retrospective comparisons of treated and untreated patients.

The present study. Treatment of cardiogenic shock would clearly be enhanced if patients at risk could be identified early in the course of illness. In this issue of the Journal, Hands and coworkers (8) from the MILLS study group report data on the in-hospital development of cardiogenic shock from >2,800 patients with myocardial infarction studied prospectively. Shock developed after hospitalization in 7.1% with a mortality rate of 65%. In more than half of the patients, the complication developed >24 h after admission; average time to development was 3.4 days after arrival. Not surprisingly, evidence of poor left ventricular function had both predictive and prognostic value.

Why is shock delayed, often for several days after the initial insult? The authors (8) suggest that the major culprit is infarct extension, which occurred in almost two-thirds of the patients with shock before or at the onset of shock. Their hypothesis is reasonable because pathologic studies (9) have shown that many patients succumbing to shock have multiple recent infarcts of different apparent histologic age. Infarct expansion with consequent hemodynamic deterioration may also play a role (10). Persistent hypoxia secondary to left ventricular failure and pulmonary congestion may also contribute to collapse of hemodynamic compensation in states of persistent low cardiac output.

Statistical analysis of the data (8) revealed that independent predictors for the in-hospital development of cardiogenic shock included age, initial left ventricular ejection fraction <0.35, high MB creatine kinase level, history of diabetes mellitus and previous myocardial infarction. The presence of three or more of these factors predicted a risk of about 18% for the subsequent development of cardiogenic shock; with five risk factors, the probability was slightly >50%.

Hands et al. (8) suggest that the delay from onset of acute infarction to development of cardiogenic shock as observed in this study may permit early recognition of the patient at

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risk and, hence, the application of aggressive preventative therapy. This is an interesting proposal although even the presence of five risk factors predicted shock only half the time. The hypothesis of Hands et al. is testable. Cardiogenic shock in myocardial infarction implies massive myocardial damage. Myocardial infarction is most often precipitated by coronary thrombosis. Restoration of nutrient flow may limit the damage. Angioplasty may restore coronary artery patency in the acutely ill patient. It all makes sense. If angioplasty is an effective treatment for cardiogenic shock, it will have to be demonstrated in a well designed, randomized trial in suitable cases. Perhaps the study of Hands et al. (8) offers some new, exciting possibilities for reducing mortality in cardiogenic shock by identifying patients at risk after the initial insult and permitting a definitive trial of aggressive therapy.

References