Rupture of the Heart
Complicating Myocardial Infarction

Rupture of the free wall of the heart has been reported to be the cause of death in 4–13% of fatal cases of acute myocardial infarction. The incidence of this fatal complication is second only to cardiogenic shock and arrhythmias as a cause of death. Recent success with acute revascularization in the surgical management of cardiogenic shock, similar success with infarctectomy and direct repair for rupture of the interventricular septum, and success with mitral valve replacement for papillary muscle rupture has prompted consideration of a more aggressive surgical approach to rupture of the heart.

Rupture of the free wall of the heart usually occurs within 2 weeks from the time of onset of the infarct. London and London found in a study of 1000 cases of fatal myocardial infarction that 50% of ruptures occurred within 3 days and 89% within 14 days. The anterior wall of the left ventricle is involved more commonly than the posterior wall. Rupture of the free wall of the heart results in hemopericardium, abrupt hemodynamic deterioration due to cardiac tamponade, and usually death within a very short time following rupture. An occasional patient may survive periods of half-hour to several hours. Any consideration of surgical intervention obviously would require prompt recognition of the complication and immediate surgery.

Clinical features which raise the suspicion of rupture of the heart include an abrupt decline of the arterial blood pressure, observation of paradoxical arterial and venous pressure, and rapidly increasing venous distention. These changes associated with abrupt sinus or nodal bradycardia strongly suggest rupture of the heart and cardiac tamponade. Van Torsel and Edwards, in reviewing 40 cases of cardiac rupture complicating acute myocardial infarction, presented evidence suggesting that the symptomatology in several patients was consistent with a gradual evolution of cardiac rupture. Lautsch and Lanks demonstrated organizing thrombus at the site of rupture in 65% of the 43 cases studied and similarly concluded that this suggested a progressive rather than abrupt rupture. London and London reported repeated and prolonged chest pain occurring in 55% of patients with cardiac rupture and only in 10% of fatal cases of infarction without rupture. This was interpreted as indicating slow leakage of blood into the pericardial space prior to complete rupture and death from cardiac tamponade. Presumably, if imminent or evolving rupture of the heart could be recognized clinically prior to the onset of terminal cardiac tamponade, surgical correction could be undertaken.

Only four attempts at surgical repair of cardiac rupture complicating acute myocardial infarction have been reported, with no long-term survivors. All four patients were moribund at the time of surgical therapy. Two of the patients survived acutely, one dying of irreversible brain damage 1 month later and the other of rerupture 37 days postoperatively. In all cases the ruptured myocardial tissue was described as of poor character, and difficulty was encountered in making sutures hold. Use of patch material of Dacron, pericardium, or suture reinforcement with strips of polyvinyl sponge allowed successful repair in two cases. Rerupture occurred in three of the four cases in adjacent areas of the left ventricular wall, varying in time from several minutes to 37 days following initial repair.

When cardiac rupture is suspected and associated with cardiac tamponade, pericardiocentesis should be performed immediately.
using a relatively large-bore intravenous catheter. The catheter can be left in the pericardial space during transport of the patient to the operating room to prevent recurrent tamponade. This technic has been effective in the surgical management of traumatic wounds of the heart associated with hemopericardium and has allowed successful repair of severe stab or missile injuries of the heart.12

Although the urgency necessitated by abrupt cardiac rupture and cardiac tamponade alters the logistics of any attempt at surgical repair, the concept of surgical repair is not unlike that of emergency infarctectomy in the treatment of cardiogenic shock or rupture of the interventricular septum, complicating acute myocardial infarction.3 Infarctectomy alone has not been effective in the surgical management of patients with massive infarction and cardiogenic shock unless it has been combined with repair of a mechanical defect such as rupture of the ventricular septum. To gain any reasonable salvage of patients in refractory cardiogenic shock without associated mechanical defects, acute revascularization, combined with infarctectomy in some cases, has resulted in significantly improved survival.13 To be successful in a significant number of cases of cardiac rupture treated surgically, it may be necessary to combine revascularization with repair of the rupture. To achieve this effectively would necessitate coronary angiographic equipment located in the operating room in order to obtain "on the table" coronary angiographic study. It is obvious that the patient with cardiac rupture and developing tamponade cannot be safely transported to a catheterization laboratory for study prior to surgery and that this study, if done, would have to be done in the operating room after surgical control of the rupture had been accomplished.

Even if successful surgical repair of a cardiac rupture is achieved, and in some instances combined with revascularization, recovery of left ventricular function may not occur to a sufficient degree to support circulatory demands. In this situation, the institution of mechanical circulatory support might be beneficial in facilitating recovery. Previous experience with acute revascularization and infarctectomy for the treatment of the cardiogenic shock patient has demonstrated the effectiveness of intraaortic balloon pump assistance in early postoperative management. Diastolic augmentation with the intraaortic balloon pump has been shown to increase the mean arterial pressure with a reduction in left ventricular work and in peak left ventricular pressure.14 In addition to the benefit of reducing the left ventricular work load, the latter effect would help lessen the tendency for recurrent rupture of the infarcted myocardium by reducing peak left ventricular pressure.

Although the problems associated with surgical repair of cardiac rupture complicating myocardial infarction appear nearly insurmountable, a well-organized surgical effort, based upon experience already gained from the surgical treatment of other complications of acute myocardial infarction, could probably succeed in a significant number of cases. Successful surgical therapy will require: (1) early diagnosis before the patient sustains a cardiac arrest, (2) catheter pericardiocentesis to prevent fatal cardiac tamponade during transport of the patient to the operating room, (3) a surgical staff and cardiopulmonary support system immediately available at all times, (4) the capacity for intraoperative coronary angiographic study, and (5) an effective mechanical circulatory assistance system available for early postoperative circulatory support.

Eldred D. Munding

References


