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Surgical Treatment of Acute Myocardial Infarction

Arthur R. Dresdale, MD,* and Gaetano Paone, MD*

In recent years, advances in surgical techniques and perioperative management of cardiac surgical patients have facilitated more aggressive operative treatment of many serious and even catastrophic complications of acute myocardial infarction (MI). In addition, improved understanding of the natural history of these complications has helped to optimize the indications for and timing of surgical intervention. The role of emergency revascularization for postinfarction angina, evolving acute MI, cardiogenic shock after acute MI, and failed percutaneous transluminal angioplasty has expanded with overall satisfactory to excellent results. Surgical treatment of mechanical complications of acute MI such as free-wall rupture, acute ischemic mitral insufficiency, and ventricular septal defect also is undertaken earlier and more aggressively. Although the mortality rates for these conditions remain higher than desired, salvage of many previously doomed patients now is a reality. (Henry Ford Hosp Med J 1991;39:245-50)

Dramatic advances in surgical technique and perioperative management during the 1980s have increased the prospects for surgical treatment of many catastrophic cardiac maladies. Previously established indications for emergency surgery after acute myocardial infarction (MI) include acute ischemic mitral regurgitation, ventricular septal defect (VSD), and left ventricular (LV) rupture. Formerly less conventional indications such as cardiogenic shock, postinfarction angina, and acute coronary occlusion during angioplasty are also now considered amenable to surgical intervention.

Evolving Acute MI and Postinfarction Angina

Nearly ten years have passed since the appearance of the first papers purporting salutary results for the surgical treatment of acute MI (1,2). These reports were published in an era when conventional wisdom held that emergency revascularization carried unacceptably high risks, and no doubt helps to explain why these optimistic results were greeted with enthusiasm by the community of cardiovascular physicians and surgeons involved in the care of such patients. Ironically, consideration of appropriate indications for surgical revascularization during bona fide acute MI has become a more daunting task because of the poor design and confusing array of patients which characterizes this early body of literature (3). The perception exists that emergency coronary artery bypass grafting can be performed with an operative mortality of 5%. While this number is reasonable if only patients with preserved LV function undergoing first-time operation are considered, it is probably not an accurate assessment of risk when the patient population is expanded (4).

Critical review reveals that the seminal articles were neither randomized nor prospective studies (1,2). The criteria for diagnosis of acute MI were not well defined (5). Surgery frequently was not performed for up to 36 hours after the onset of "so-called" ischemia or failed percutaneous transluminal coronary angioplasty (PTCA), and surgical revascularization more often than not was implemented in low-risk candidates (5). True surgical emergencies either were not included or comprised a minority of the patients. They did not include patients with multivessel disease undergoing complex, aggressive PTCA as is often the case today. Furthermore, the indications for surgical intervention in many cases were not signs and symptoms of profound ischemia, such as intractable chest pain, cardiogenic shock, or full-fledged cardiac arrest. "Emergency" was often defined as a patient with angiographic evidence of ominous anatomic findings. These influential studies contained a dearth of patients with severe three-vessel coronary disease or those who had actually sustained an electrocardiographically or enzymatically documented MI. Indeed, in one study "emergency" was defined as an operation that was not scheduled at the beginning of the surgical day (6). Many studies omitted consideration of patients who required reoperative revascularizations (1-3,5,7,8). When such patients were included, the revascularizations were not done emergently but as long as 24 hours after a failed PTCA (9-11).

Recently, several studies have attempted to clarify the prognosis for the true emergency coronary artery bypass grafting patient who requires revascularization during the course of acute MI. Although these studies are flawed because they are not randomized prospective trials, the conclusions drawn from this cohort of patients seem to predict more accurately the results of revascularization.
vascularization for true surgical emergencies (4,12). Such reviews have included patients undergoing emergency surgery within 4 hours and often less than 1 hour after the onset of ischemic symptoms. These patients had persistent ischemia refractory to aggressive medical treatment with intravenous nitroglycerin or after thrombolytic therapy or PTCA. Operative mortality ranges from 11% to 15%. Important risk factors include left main coronary artery disease, reoperative coronary bypass surgery, cardiogenic shock, depressed LV function, and the need for cardiopulmonary resuscitation.

Contemporary management of acute MI includes thrombolytic therapy followed, if necessary, by angiographic intervention to delineate coronary anatomy and guide future therapy. PTCA and coronary bypass surgery both provide alternatives for treatment of patients with persistent ischemia. To optimize salvage of jeopardized myocardium and reduce postoperative infarct rates, the interval between symptoms and coronary blood flow restoration must be minimized. When considering surgical intervention for acute MI, it must be remembered that the resources to administer surgical care to acutely ill patients require enormous logistic support in order to accomplish myocardial reperfusion within a reasonable time. Furthermore, reperfusion of hemorrhagic infarcts is fraught with the potential for disastrous complications including malignant postoperative ventricular arrhythmias which may be refractory to medical management (13). As more attention is turned to refinement of chemical cardioplegia and development of resuscitative solutions to ameliorate reperfusion injury, the incidence of this potentially fatal complication hopefully will decline. At present it seems reasonable to consider the early use of thrombolytic agents and heparin to stabilize the patient and further define the clinical situation.

Continued or recurrent angina pectoris following a documented MI represents the presence of viable but persistently ischemic myocardium either within the area of infarction in the so-called “border zone” or at some area remote from the infarction supplied by a patent but critically stenosed artery (14-16). Despite intensive medical therapy, these patients have a high risk of reinfarction and overall a poorer short- and long-term prognosis (17,18). If ischemic symptoms persist in an otherwise good candidate, angioplasty or coronary bypass surgery should be performed if feasible within hours to days after the initial onset. Overall, coronary bypass grafting can be accomplished with relative safety within 30 days of acute MI in hemodynamically stable patients with preserved LV function (12,19-22). Emergency revascularization of a transmural infarction in a patient with impaired LV function may carry a higher mortality than does conservative medical management. Several series have documented higher operative mortalities for patients operated on early after transmural MI and have advised a period of medical stabilization whenever possible (11,19,20). Surgical intervention after non-Q wave MI appears to be associated with less risk. However, at least one group has found a higher operative mortality in the non-Q wave subset (23). When possible, it is prudent to postpone surgical revascularization for at least one week, unless symptoms of intractable ischemia persist. Given the adverse natural history associated with recurrent cardiac events in these patients, however, surgical revascularization should be undertaken without delay when ischemia cannot be easily controlled with appropriate medical management.

**Cardiogenic Shock**

Cardiogenic shock occurs in approximately 10% to 25% of patients after acute MI (24). In patients without associated mechanical defects, its presence signifies involvement of greater than 40% of ventricular mass in the infarction process (25). Medical therapy alone is associated with a mortality rate of 80% to 90% (24). The addition of intraaortic balloon pump to medical therapy may result in temporary improvement in clinical status but does not significantly improve overall early and late mortality (26). In view of this grim prognosis, cardiogenic shock after acute MI has come to be viewed as a medical and surgical emergency whereby inotropic and mechanical support is promptly instituted, cardiac catheterization performed, and myocardial blood flow restored as efficaciously as possible. Depending upon the circumstances, either PTCA or bypass surgery may be appropriate. For the surgical approach to have a reasonable chance of success, the patient must have identifiable distal vessels suitable for bypass grafting and the procedure must be undertaken and completed within a time frame compatible with significant myocardial salvage. Operative mortality is approximately 34% (12% to 66%) (27). Routine use of early operation, a strategy of complete revascularization, and development of solutions capable of resuscitating ischemic myocardium and ameliorating reperfusion injury hopefully will improve the surgical results of this catastrophic complication of acute MI.

**Coronary Artery Bypass Grafting After PTCA Failure**

Since its introduction by Gruentzig et al (28) in 1978, PTCA has gained widespread acceptance and emerged as the technique of choice for revascularization of patients with single-vessel coronary artery disease. More recently, proponents of PTCA have expanded its use to selected subsets of patients with two- and three-vessel disease. Despite improvements in catheter design and increased operator experience, between 3% and 8% of patients undergoing PTCA will require emergency coronary bypass surgery for complications resulting in acute MI (29). Acute arterial occlusion results from either thrombus formation, plaque rupture, intimal tear, or coronary dissection and is generally associated with acute electrocardiographic changes and chest pain. Varying degrees of hemodynamic instability may be present. Cardiogenic shock due to severe LV dysfunction occurs in 10% to 27% of these patients (27). Emergency coronary bypass surgery with complete revascularization is the preferred therapy for such patients with acute ischemia following an unsuccessful PTCA and should be undertaken expeditiously. In our opinion, repeated efforts to reopen the culprit vessel are ill-advised and may unnecessarily prolong the period of ischemia. Similarly, utilization of an intraaortic balloon pump is frequently advocated, especially in patients with hemodynamic stability or
when there will be a delay in getting to the operating room. While it may be helpful, balloon insertion should be done in a manner so as not to delay the time to surgical revascularization. More recently, reperfusion catheters placed across the lesion allowing distal perfusion and temporary amelioration of ischemia have proved beneficial in extending the use of internal mammary artery grafting in this setting (30). Patients in whom angioplasty has been unsuccessful but who do not have ongoing ischemia do not need emergency revascularization. They can be managed semi-electively.

A widespread perception exists among both patients and medical personnel that PTCA offers a "no lose" situation; failure simply results in emergency rather than elective surgical revascularization with no other untoward affects (31). Although the reported surgical mortality following failed PTCA is acceptable, it fails to support this perception. In a recent review of 701 patients without cardiogenic shock who were reported in the literature between 1982 and 1988, Barner et al (32) found an average operative mortality of 3.6% (0% to 7.7%). Parsonnet et al (33) reviewed 15 series, totaling 15,802 PTCA and 902 emergency operations, and found an average mortality of 5.9% (0% to 12%). A comparable cohort of patients operated on electively would have an operative mortality of less than 2%. Factors associated with increased risk include poor LV function, presence of cardiogenic shock or cardiac arrest, prolonged time to revascularization, and multivessel disease.

Despite the well-documented onset of ischemia and ability to perform revascularization within the generally accepted time frame of 4 to 6 hours before the onset of irreversible myocardial ischemia, perioperative MI occurs on average in 37.6% of patients, with some series reporting rates as high as 60% to 70% after failed PTCA (33).

### Surgical Treatment of Mechanical Complications of Acute MI

The sudden development of profound hemodynamic compromise manifested by acute pulmonary edema and/or cardiogenic shock in a patient who has suffered an acute MI from one day to two weeks previously should raise concern that a catastrophic mechanical complication may have occurred. While the final common pathway of presentation appears deceptively similar for some of these catastrophic mechanical problems, history, physical examination, and hemodynamic assessment readily yield the diagnosis in most cases. With expeditious diagnosis and medical stabilization, which we believe includes intraaortic balloon pump insertion, surgical survival of patients with acute mechanical complications after MI has reached about 50% whereas survival without surgical treatment is only 10% (34).

#### Ventricular free-wall rupture

Rupture of the ventricular free wall occurs in approximately 8% to 10% of patients who die in-hospital following an acute MI (35). Unfortunately, it is most commonly identified at autopsy. Most frequently seen in hypertensive, elderly females, this condition usually affects the anterior or lateral wall of the left ventricle in the domain of the terminal blood supply of the anterior descending coronary artery. Ventricular free-wall rupture is uncommon in patients suffering less than a 20% transmural infarction and usually is seen three to five days after acute MI. It is uncommon in an area that is well collateralized. With the dawn of the era of thrombolytic therapy, the incidence of free-wall rupture may increase. Just as streptokinase will dissolve intracoronary thrombus, it might well lyse the clot forming on damaged endocardium at the site of a transmural infarct. Since the pathophysiology of this condition is thought to involve a dissecting hematoma that perforates a necrotic area of transmurally infarcted myocardium, thrombolytic therapy might lyse the endothelial clot, thereby facilitating dissection of blood through necrotic myocardium and eventually rupturing through the epicardium. At least one report documents surgical salvage of a patient who had received streptokinase followed by heparinization to treat a transmural infarction and who suffered a massive free-wall rupture about 10 hours later manifested by total circulatory arrest (36). In view of the increasing prevalence of thrombolytic therapy, increased scrutiny of hemodynamic instability early after acute MI is warranted to detect such catastrophic events.

If massive, sudden ventricular rupture into the pericardial cavity occurs, hemopericardium with cardiac tamponade, electromechanical dissociation, and death rapidly ensue. This condition arguably may be the most obscure and hence challenging to identify in a severely compromised individual suffering from a massive MI. The hemodynamic derangements of profound cardiogenic shock can obfuscate the clinical picture of acute pericardial tamponade. A spectrum of clinical scenarios may be seen, from a catastrophic complete tear with immediate death to an incomplete, more insidious defect which can be associated with late rupture or false aneurysm formation. While surgical treatment is the only chance for survival in the most catastrophic cases, it is usually unsuccessful.

When organizing hematoma and thrombus adhere to pericardium and epicardium to seal or confine the LV rupture thereby preventing hemopericardium, a false aneurysm may form. These pseudoaneurysms can become enormous, reaching a dimension equal to the LV cavity with which they communicate by a narrow neck opening observable on left ventriculography. When identified, they warrant expeditious surgical resection because of the high risk of rupture.

In a patient suffering a recent acute MI, elevated equalization of diastolic pressures in all chambers suggest the diagnosis (37). The sudden onset of profound heart failure associated with shock and in some cases electromechanical dissociation should stimulate consideration of acute ventricular rupture. Expeditious pericardiocentesis confirms the diagnosis and transiently improves the patient's condition. If the patient is hemodynamically stable, echocardiography may be useful to confirm this diagnosis. Only if the patient's condition allows, coronary angiography should be done quickly to delineate coronary artery anatomy and the need for bypass grafting. Otherwise immediate surgical intervention is mandatory to repair the rupture. If coronary angiography is not feasible, the surgeon may consider performing coronary bypasses blindly if proximal coronary artery lesions are palpated at the time of surgery (38).
Left ventricular aneurysm

A ventricular aneurysm is a circumscribed noncontractile out-pouching of the left ventricle. It develops in 5% to 15% of survivors of a MI. A true aneurysm most often is associated with total occlusion of a poorly collateralized left anterior descending coronary artery. True aneurysms rarely are seen with multi-vessel disease when there are either extensive collaterals or the left anterior descending artery is not completely occluded (34). They occur approximately four times more often at the apex and in the anterior wall than in the inferior posterior wall. Usually the overlying pericardium is densely adherent to the wall of the aneurysm which may become partially calcified after several years. Patients with a LV aneurysm are six times more likely to die than those without aneurysms with comparable LV function. Death is often sudden and presumably related to the high incidence of ventricular tachyarrhythmias that occur with aneurysms. The diagnosis is easily made by echocardiography, radionuclide ventriculography, or left ventriculography. Mitral valve regurgitation of moderate-to-severe degree may be identified because of dysfunctioning LV wall adjacent to the papillary muscle. Survival of patients with cardiogenic shock who undergo resection of infarcted, aneurysmal myocardium is reported to be about 60% (39). If LV dysfunction is not severely diminished, surgical resection will be necessary. If patients are stable enough to survive for such a long period without intervention. Therefore, the consensus is that when hemodynamic instability with cardiogenic shock and/or pulmonary edema is present, emergency operation should be performed (42-45). Contraindications to emergency surgery include objective evidence of neurologic impairment, severe renal dysfunction, and ischemic bowel or limb injury. Operative mortality is between 10% and 40% (42-44). The prognosis is most grim for patients with posterior defects, those in cardiogenic shock, and those undergoing early surgery after infarction.

Acute mitral insufficiency

Acute mitral insufficiency represents a spectrum of pathophysiologic conditions, some of which are more amenable to surgical correction than others. Between 0.4% to 5% of patients with acute MI succumb from overwhelming mitral regurgitation as a result of acute papillary muscle rupture (46). Total acute papillary muscle rupture is not compatible with life because of the overwhelming mitral insufficiency which ensues. Unlike VSD which occurs in large Mls, papillary muscle rupture ironically may occur with a relatively small infarction in at least 50% of cases. Only 20% of cases of papillary muscle rupture involve the anterolateral muscle. Most commonly the posterior medial papillary muscle is the site of rupture. Usually the infarction is inferior and secondary to occlusion of the posterior descending coronary artery, thereby explaining the prevalence of rupture of the posteromedial papillary muscle which receives its only blood supply from this vessel. The anterolateral papillary muscle receives a dual blood supply from both the diagonal branch of the left anterior descending coronary artery and the obtuse marginal coronary artery, rendering it less vulnerable to ischemic injury (47). Alternatively, acute MI may be associated with papillary muscle infarction without rupture, only partial rupture, or isolated chordal rupture leading to mitral incompetence compatible with life but necessitating surgical intervention.

As in the case of patients with acute VSD, individuals who suffer marked papillary muscle dysfunction present with a loud, holosystolic murmur followed by the development of increasingly severe heart failure and pulmonary edema. In either situation the murmur may disappear as the arterial pressure falls. Echocardiogram is particularly helpful in recognizing partial or complete papillary muscle rupture and should be obtained immediately on any patient in whom the diagnosis is suspected because of hemodynamic deterioration or the new onset of a murmur. While it is easy to detect a problem in a patient with an acute MI who soon develops a loud holosystolic murmur with hemodynamic compromise, the differential diagnosis of ventricular septal rupture from mitral regurgitation can be difficult. As noted, patients with VSD will demonstrate a pathognomonic step-up in oxygen saturation in the right ventricle and pulmonary artery compared with the right atrial blood. Patients with acute mitral regurgitation will lack this step-up in oxygen saturation and instead will demonstrate tall V-waves in both the pulmonary capillary and left atrial pressure tracings. Large V-waves, however, may be seen in patients with VSD. Thus the presence of this finding is not necessarily diagnostic in any indi-
vidual patient (34). Cardiac output will be significantly diminished in patients suffering from either condition. MI is subendocardial as opposed to transmural in about half the cases of significant papillary muscle dysfunction. In most cases the infarction is neither massive nor associated with a marked reduction in ejection fraction. It is possible to develop mitral insufficiency in association with a postinfarction VSD or free-wall rupture (48).

About 25% of patients treated nonsurgically survive more than 24 hours after total rupture of a papillary muscle. After partial papillary muscle rupture, 70% may survive at 24 hours but only 50% will survive at least one month (47). Once again, the need for expeditious intraaortic balloon insertion is emphasized when this diagnosis has been made. The patient is immediately prepared and taken for coronary surgery after coronary angiography before operative intervention, providing other critical organ systems are still intact.

**Conclusion**

The role of emergency revascularization for postinfarction angina, evolving acute MI, cardiogenic shock after acute MI, and failed PTCA has expanded with overall satisfactory to excellent results. Surgical treatment of mechanical complications of acute MI such as free-wall rupture, acute ischemic mitral insufficiency, and VSD also is undertaken earlier and more aggressively. Although the mortality rates for these conditions remain higher than desired, salvage of many patients previously moribund now is a reality.

**Reference**