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Myocardial Infarction 1972
(Part 6)

Surgery for Complications of Acute Myocardial Infarction

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With the greater availability of well-equipped and well-staffed coronary care units, death from arrhythmias complicating myocardial infarction has become less common. This has been a result of improved methods of patient monitoring, earlier diagnosis of arrhythmias, and prompt treatment. Death from myocardial infarction at present is more commonly a result of cardiogenic shock or refractory cardiac failure. The mechanical complications of myocardial infarction, including rupture of the interventricular septum, papillary muscle rupture, and acute left ventricular aneurysm, although uncommon, are associated with a high mortality rate.

Concomitant with the development of an accurate and safe method of selective cinecoronary arteriography for the diagnosis of coronary artery disease, effective surgical procedures for direct coronary arterial revascularization have evolved. Despite an impressive volume of patients undergoing elective revascularization, urgent myocardial revascularization, infarctectomy, repair of ventricular septal defect, or mitral valve replacement in patients with complications of acute myocardial infarction has been infrequent. Until recently, surgery for the complications of acute myocardial infarction less than 4 weeks from the time of the infarct has been associated with a near-prohibitive mortality. After an interval of 4 weeks or more, revascularization or correction of mechanical defects resulting from infarction have been associated with reasonably good clinical results. The latter group obviously includes only those patients remaining hemodynamically stable and surviving 4 weeks or more after myocardial infarction. However, the majority of patients dying of complications of acute myocardial infarction do so within 2 weeks of the time of infarct. The present discussion will be limited primarily to patients in this category.

Cardiogenic Shock

Cardiogenic shock, or left ventricular power failure, is at present the complication of acute
myocardial infarction which most commonly results in death.14,17,19 Most recent series have reported an overall mortality from acute myocardial infarction in the range of 30 to 40%, with approximately 20% of the deaths occurring outside of the hospital.17 In a cooperative study from nine coronary care units the mortality of acute myocardial infarction in 2842 patients ranged from 14 to 30% with an average of 25%.1 The major cause of death was pump failure, or cardiogenic shock, which was associated with a mortality of 80–90% once shock became clinically manifest.

The initial physiologic insult of a major myocardial infarction results in a decrease in left ventricular function manifested by a fall in cardiac output and arterial perfusion pressure. The latter results in diminished coronary perfusion, which further contributes to the functional deficit of the ischemic myocardium. Thus, a vicious cycle of events may ensue, which often results in extension of the original infarct and progressive left ventricular power failure. Left ventricular function is not only adversely influenced by the actually infarcted myocardium, but also by the presence of a functionally compromised ischemic perinfarction zone myocardium. Schelbert et al.20 and Hood et al.21 have shown that myocardial contractility is depressed in ischemic areas of the left ventricle but not eliminated unless cell death has occurred. Myocardial contractility in ischemic areas of the left ventricle can be temporarily improved with administration of inotropic agents such as isoproterenol, but eventually at the expense of increasing the area of myocardial injury.22–24 The cause of this increased myocardial injury is the increased oxygen requirement of the ischemic myocardium induced by the inotropic agent, in a situation where oxygen delivery cannot be enhanced. Hypotension and reduction of coronary perfusion pressure also result in further depression of myocardial function in ischemic zones and lead to extension of the area of injury.25,26 Particularly at low coronary perfusion pressures, left ventricular function is coronary perfusion-pressure dependent.27,28 These observations are of obvious clinical importance. Thus, it is essential to maintain an adequate coronary perfusion pressure in the cardiogenic shock patient, but it is desirable to achieve this without increasing myocardial oxygen requirements, as occurs with the administration of most catecholamines. In experimental studies of cardiogenic shock, Rona, Kahn, and Chappell29 demonstrated the development of focal areas of myocardial necrosis in animals treated with catecholamines. Focal areas of myocardial necrosis resulting from the hemorrhagic shock state were shown by Gomez et al.30 to occur despite maintenance of normal coronary blood flow. This may be blocked by administration of pronethalol as shown by Entman et al.30 Clinical studies of patients in cardiogenic shock have shown that a pressor response is observed with administration of either alpha- or beta-adrenergic catecholamines, but the mortality rate is not significantly influenced.4,5,24,31 Potential adverse effects of catecholamine administration are difficult to ascertain since the overall mortality rate regardless of therapy has been in the range of 80 to 90%.1,3,4

The clinical criteria for definition of cardiogenic shock are obviously important if accurate assessment of the effectiveness of various forms of therapy is to be achieved. The incidence of shock complicating acute myocardial infarction and the results of therapy differ in various series primarily because of differences in definition of the shock state.17 It is not uncommon for shock to appear early in the clinical course of a patient with an acute infarct but to subside within 1–3 hours with supportive measures only. Shock persisting longer than 1–3 hours is the primary concern of this discussion. The criteria for cardiogenic shock, as established by Myocardial Infarction Research Unit (MIRU) centers across the country, include: (1) arterial blood pressure less than 90 mm Hg, (2) oliguria, with urine output less than 20 ml/hour, and (3) systemic manifestations of shock including peripheral vasoconstriction, mental confusion, or obtundation. Further
subclassification of the degree of severity of shock based upon hemodynamic criteria has been useful in analyzing the results of therapy. The product of the cardiac index and mean arterial pressure (CI \times AP) yields a rough index of ventricular function. When this product is less than 120, the degree of shock is categorized as severe, class 4. When the CI \times AP product is greater than 120, but the PCWP is elevated above 20 mm Hg, the degree of shock is categorized as class 3. Class 2 shock exists when the clinical criteria for shock are present but the CI \times AP product is greater than 120 and the PCWP is less than 20 mm Hg.

On the basis of a clinical study of 73 patients in cardiogenic shock, Scheidt, Aschheim, and Killip found that there were no common predisposing factors such as a previous infarct, previous angina, hypertension, or cardiac failure. In addition, these authors found no consistent correlation with the development of cardiogenic shock and the location of the infarct or duration of time from the onset of symptoms. Page et al. however, found in their review of 20 patients dying of cardiogenic shock that 65% had a previous infarct, and the lower anterior wall and apex of the left ventricle were involved in all patients. A common finding in all studies was that the area of left ventricular loss was 40% or greater. In the study by Scheidt et al. the SGOT was found to be markedly elevated (> 400 mg/100 ml) more commonly in shock patients. Although a hemodynamic response to norepinephrine, aramine, or isoproterenol was observed in nearly all patients, there was no significant difference in mortality. In this study, where the average cardiac index was 1.1 liters/min/m², the mortality was 50% at 10.2 hours after the onset of shock and 86% overall.

Treatment of the shock syndrome must be individualized. In order to assess the moment-to-moment clinical status of a patient in shock and to determine the effectiveness of therapy, careful physiologic monitoring is essential. The pharmacologic treatment of cardiogenic shock has not been overly effective. Although catecholamine administration usually results in an elevation of arterial pressure and cardiac index, it may cause further injury to the ischemic myocardium as previously discussed. When the shock state is associated with a CI > 2 liters/min/m² and a low total peripheral vascular resistance (mean AP/CI) it may be desirable to increase peripheral vascular tone with an alpha-adrenergic drug such as norepinephrine or metaraminol. Obviously, this increases left ventricular afterload and left ventricular work. Arrhythmias which result in a reduction in cardiac output or in the efficiency of atrioventricular transport must be treated promptly. Atrial or sequential atrioventricular pacing, not infrequently, is the only therapeutic measure required to bring the patient out of shock. Support of an adequate blood volume as determined by left and right heart filling pressures is also essential to maintain an optimal cardiac output. If the patient remains in cardiogenic shock despite these measures, circulatory assistance should be considered (Table 1).

There are several systems clinically available for mechanical circulatory assistance, each with its own advocates. Presently, the most extensive and successful clinical experience with mechanical circulatory assistance has been with the intraaortic balloon pump. The physiologic benefits of circulatory assistance include an increase in cardiac output, a reduction in left and right ventricular afterload, a decrease in systemic vascular resistance, and an improvement in global oxygen delivery. In addition, hemodynamic support facilitates optimal drug therapy and prevents hemodynamic failure while allowing time for correction of other factors contributing to shock.

Table 1

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<th>Therapeutic Measures for Treatment of Cardiogenic Shock</th>
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<td>1. Supportive</td>
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<td>Oxygen, morphine, blood volume adjustment, correction</td>
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<td>2. Electrical pacing technics</td>
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<td>Atrial or sequential atrioventricular pacing</td>
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<td>3. Pharmacologic</td>
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<td>Antiarrhythmic drugs, catecholamines, digitalis</td>
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<td>4. Circulatory assistance</td>
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assistance to the ischemic injured heart are discussed elsewhere in this symposium. Clinical hemodynamic evaluation of circulatory assistance with intraaortic balloon pumping (IABP) for the treatment of patients in cardiogenic shock has demonstrated a significant reduction in left ventricular peak systolic pressure, a reduction in left ventricular end-diastolic pressure, and significant improvement in cardiac output. Although there have been a number of striking results, the acute mortality of patients treated by IABP assistance has remained in the range of 80%. Most of the patients treated by this means, however, were in severe class 4 cardiogenic shock. Although a concomitant control group of similar patients treated conventionally was not observed, it is probable that the expected mortality of this group of patients treated conventionally would approach 100%.

Braunwald et al. have suggested that the fate of a patient with myocardial infarction and cardiogenic shock depends upon a balance between the myocardial oxygen requirements needed to maintain an adequate circulation and the capacity for oxygen delivery to the ischemic myocardium. Another obvious factor is the extent of the irreversibly injured myocardium. In some instances, the total left ventricular area irreversibly involved may be too extensive for recovery with any mode of therapy short of cardiac replacement. In many instances, as suggested by clinicopathologic studies previously discussed, left ventricular wall loss appears to be progressive during the early course of infarction. In view of this concept and with the development of effective methods for direct coronary arterial revascularization, the possibility of acute myocardial revascularization reversing cardiogenic shock became a therapeutic potential requiring evaluation.

Segmental myocardial ischemia has been shown to result in a reduction in contractility in the ischemic zone which can be reversed by restoring adequate coronary perfusion. As previously noted, persistence of an acutely ischemic perinfarction zone leads to eventual cell death and extension of the infarction. Prompt institution of mechanical circulatory assistance in the clinical setting may prevent this course by reducing the left ventricular work requirement and associated oxygen demand of the ischemic zone of myocardium. Mechanical circulatory assistance, if promptly instituted, may prevent extension of the infarcted zone, but since the relative inadequacy of myocardial oxygen supply persists, hemodynamic recovery may not be adequate when circulatory assistance is eventually terminated. Although Jacobey et al. suggested that brief periods of counterpulsation opened intercoronary collaterals in the acutely ischemic heart enabling hemodynamic recovery after only a brief period of circulatory assistance, subsequent clinical experience has not corroborated this. The development of intercoronary collaterals on the basis of experimental studies normally requires weeks to months in response to the stimulus of myocardial ischemia. The combination of prompt institution of mechanical circulatory assistance and direct coronary artery revascularization thus emerged as a possible new mode of therapy for the patient with refractory cardiogenic shock. Direct coronary arterial revascularization, by providing improved perfusion of the ischemic perinfarction zone of myocardium, may allow sufficient recovery of left ventricular function for survival.

Despite the critically ill status of patients with an acute myocardial infarction, coronary angiographic study can be performed with relative safety. The patient in cardiogenic shock presents a considerable risk for any diagnostic study. However, Leinbach, et al. demonstrated that complete evaluation of the shock patient can be safely achieved, including coronary arteriography and left ventriculography, with the protection of intraaortic balloon pump assistance.

A number of centers have reported series of patients undergoing emergency revascularization for impending or evolving myocardial infarction with generally excellent results. In none of these series did the patients have associated shock except in one patient who died at the time of surgery,
reported by Scanlon et al.\textsuperscript{12} Our experience has indicated that the combination of circulatory assistance with acute revascularization salvaged a significant number of patients in refractory cardiogenic shock.\textsuperscript{16, 33, 47, 48} The role of infarctectomy in the management of these patients is not yet clarified. Heimbecker’s original reports of experimental studies evaluating the efficacy of infarctectomy for the treatment of acute myocardial infarction were encouraging.\textsuperscript{49} Their studies indicated that acute infarctectomy facilitated defibrillation and resuscitation of canine hearts subjected to sudden ligation of the left anterior descending coronary artery. The clinical application of the procedure, however, was combined with closure of a ventricular septal defect in three patients, which undoubtedly contributed to their left ventricular functional impairment preoperatively. Glass et al.\textsuperscript{50} and Jude\textsuperscript{51} have confirmed the experimental studies of Heimbecker, showing an improvement in the ability of the heart to be defibrillated and an increase in cardiac output with resection of infarcts less than 30\% of the left ventricular wall. Resection of larger portions of the ventricular wall resulted in no survival. In a careful experimental study comparing infarctectomy with ¼ hour of circulatory assistance with cardiopulmonary bypass, Danielson, Resnicoff, and DeWeese\textsuperscript{52} found that there was no improvement in left ventricular power, arrhythmia control, or survival with infarcts greater than 13\% of the left ventricular total weight, whether treated by infarctectomy or a brief period of circulatory assistance. In a theoretic analysis based upon the left ventricle considered as a sphere, Klein et al.\textsuperscript{53} concluded that the Starling mechanism would fail with resection of greater than 20–25\% of the left ventricular surface area.

Clinical experience with acute infarctectomy less than 3–4 weeks following myocardial infarction has been limited. Isolated reports of success following infarctectomy have been, for the most part, in association with closure of a significant ventricular septal defect.\textsuperscript{49} The largest single experience with both infarctectomy and revascularization is that from the Massachusetts General Hospital.\textsuperscript{16, 33, 48}

As yet, no clear-cut criteria for selection of patients for surgery has evolved, but certain general guidelines have been established. When cardiogenic shock is profound or persists for more than 1–2 hours after commencing general supportive care, circulatory assistance should be considered. It is quite clear from the experience with intraaortic balloon pump assistance that the maximum hemodynamic benefit is attained within 24–48 hours of circulatory assistance.\textsuperscript{33} If, at that time, hemodynamic studies without circulatory assistance indicate a cardiac index of less than 2 liters/min/m\textsuperscript{2} or a pulmonary capillary wedge pressure of over 20 mm Hg, it is unlikely that the patient will ultimately recover, in spite of continued circulatory assistance. Urgent coronary angiography should usually be advised and surgery considered if a surgically remediable situation exists.\textsuperscript{33, 47, 51} Criteria for establishment of a surgically remediable situation have been difficult to establish. Of those patients operated upon who failed to survive surgery, pathologic examination revealed left ventricular wall loss of over 60\% in most. It is quite apparent that a number of patients with cardiogenic shock will have such extensive myocardial loss that revascularization and/or infarctectomy could not be effective. Careful analysis of the hemodynamic and angiographic findings in the 16 patients undergoing surgery did not reveal any obvious common denominator in terms of patient selection. However, several findings appeared to be relative contraindications to surgery: (1) chronicity of clinical course; (2) poor or no response to inotropic agents; (3) severe hypokinesia or akinesia involving greater than 60\% of the left ventricle; (4) inadequate coronary vessels distally for bypass; and (5) avascularity of hypokinetic areas of left ventricle adjacent to the infarct.

The decision as to the need for infarctectomy in addition to revascularization has been made at the time of surgery. We presently feel that several factors favor infarctectomy: (1)
the presence of a large paradoxically bulging infarct or acute aneurysm which appears to interfere with left ventricular dynamics; (2) thinned-out necrotic infarct where rupture appears imminent; or (3) a localized infarct associated with recurrent ventricular arrhythmias.

It has been evident from our experience that mechanical circulatory assistance was essential to the successful management of the surviving patients. The prompt institution of intraaortic balloon pump assistance prevented continued hemodynamic deterioration or extension of the infarct, enabled safe complete angiographic study in each case, and facilitated management of the patient both during surgery and after operation. The effect of coronary arterial revascularization on left ventricular function was immediately apparent in surviving patients. Visibly improved left ventricular contractility in these patients was associated with a significant improvement in left ventricular hemodynamics in the early postoperative period. It is apparent from this experience that direct coronary arterial revascularization has the potential of improving left ventricular function and reversing cardiogenic shock associated with myocardial infarction in carefully selected patients.32

Rupture of the Ventricular Septum

Rupture of the ventricular septum is a relatively uncommon complication of myocardial infarction. The incidence has been reported in the range of 0.5 to 1% and accounts for about 2% of all deaths following myocardial infarction.17

Rupture of the ventricular septum usually occurs in the lower septum associated with an anteroseptal infarct.17 Swithinbank, in a review of 113 cases, found 66% involving the low septum and 17% involving the posterior septum.54 This complication occurs most commonly in the first week after infarction and clinically is characterized by a sudden deterioration in the patient’s condition. The left-to-right shunt produced by the defect precipitates severe biventricular failure in an already compromised heart. Cardiogenic shock also may develop in association with increasing failure. The mortality of this complication is extremely high: 24% die within 24 hours, 87% within 2 months, and over 90% at 1 year.55

The diagnosis of this complication of acute myocardial infarction can be suspected when there is a sudden deterioration in the clinical course of a patient associated with the abrupt onset of a harsh systolic murmur at the left sternal border. It is difficult clinically to distinguish rupture of the ventricular septum from dysfunction or rupture of the papillary muscle and acute mitral regurgitation.15, 17 Cardiac catheterization is necessary to establish the diagnosis definitely.

The feasibility of surgical repair has been firmly established.14 Earlier experience indicated that successful surgical repair was much more likely beyond 4 weeks from the time of perforation.15, 16 Attempts at surgical repair less than 2 weeks following the development of perforation were associated with an extremely high mortality. However, statistics have demonstrated that 24% of patients with this complication die within 24 hours and 65% within 2 weeks from the time of septal rupture.55 Interest in attempting closure of a septal defect in the acute postinfarct period was stimulated by the report of Cooley et al.56 in 1959 of a patient undergoing patch closure 1 day following rupture of the septum. The patient survived acutely, but succumbed 8 weeks postoperatively. Autopsy demonstrated a residual defect at the edge of the patch.

Heimbecker49 and associates in 1967 first reported successful closure of a septal defect in the acute postinfarct period combining infarctectomy with closure of the septal perforation. The patient succumbed 1 month later of a complication of tracheostomy. Subsequently Stinson et al.57 Lajos et al.11 and Daggett et al.58 reported successful long-term survivals in three patients operated upon 9 days, 5 days, and 3 days after septal rupture. From the accumulated earlier experience it was apparent that an operative approach through a right ventriculotomy resulted in an
overwhelming burden to the already compromised right ventricle.\textsuperscript{16, 58} Since the majority of acquired septal perforations occur low in the septum associated with a low anteroseptal or posteroseptal infarct, surgical closure of the defect is usually feasible through a left ventriculotomy in the infarcted area.\textsuperscript{16} Resection of the infarct, which, in some cases is an acute left ventricular aneurysm, may offer further hemodynamic benefit to left ventricular function. Buckley et al.\textsuperscript{16} have recently reported four survivors of five patients operated upon less than 10 days following septal rupture, using this approach. The average follow-up of these patients is 14 months, and all four are clinically well. Although this small experience has been encouraging, it is obvious that the risk of surgery in the acute postinfarct period is high. Every attempt should be made to support the patient medically until further time has elapsed, if it is at all possible. Clinical experience has been excellent with elective closure of acquired ventricular septal defects 8 weeks to 6 months after an acute myocardial infarction.\textsuperscript{14, 16} Although there are a few reported long-term survivors of this complication of myocardial infarction who have not undergone surgery, the majority who survive initially remain in a severely compromised functional status until the septal defect is surgically closed.\textsuperscript{17, 18}

The question as to whether these patients should have coronary arterial revascularization in addition to closure of the septal defect is not yet fully answered. In those patients with acute refractory clinical deterioration less than 2 weeks from the time of septal rupture, the existence of a left-to-right shunt (Qp/Qs) of greater than 3:1 is probably a sufficient hemodynamic reason for their deterioration. In this situation, closure of the defect through a left ventriculotomy combined with infarctectomy is probably the procedure of choice. In the clinical situation where the left-to-right shunt is marginal and left ventricular power failure appears to be the dominant hemodynamic deficit, direct coronary arterial revascularization probably should be combined with the former procedure. This presupposes recognition of this at the time of cardiac catheterization so that selective coronary angiography and left ventriculography can be carried out. These basic guidelines probably also apply to the consideration of surgery in patients who have survived with medical support for longer than 4–8 weeks following infarction and septal rupture. There may be a stronger indication for complete study including coronary arteriography and left ventriculography at this stage since it carries much less risk and allows a more complete evaluation of the patient. As in patients with a chronic left ventricular aneurysm following myocardial infarction, many cardiac surgeons and cardiologists would advise coronary arterial revascularization in addition to correction of the mechanical defect, particularly if there is significant disease in major vessels not anatomically or functionally related to the area of the mechanical defect.\textsuperscript{59}

**Papillary Muscle Rupture**

Rupture of a papillary muscle resulting in severe mitral regurgitation is a relatively rare complication of myocardial infarction, accounting for approximately 1% of deaths from infarction.\textsuperscript{17, 60} The location of the infarct is posterior in the majority of reported cases with involvement of the posterior papillary muscle.\textsuperscript{60} Disruption of the papillary muscle allows prolapse of the corresponding mitral valve cusp during ventricular systole resulting in severe mitral valve incompetence. The development of severe mitral regurgitation associated with an already functionally compromised left ventricle in the acute postinfarct period results in abrupt clinical deterioration characterized by intractable left ventricular failure and frequently, cardiogenic shock.\textsuperscript{17, 18, 60} The mortality of this complication has been reported as 70% within 24 hours of the development of the murmur and near 90% within 2 weeks.\textsuperscript{60}

Papillary muscle rupture is usually characterized by the sudden development of a loud apical systolic murmur associated with abrupt clinical deterioration 2–10 days after infarction. Papillary muscle rupture may be difficult to
differentiate from rupture of the ventricular septum in this setting, and cardiac catheterization is necessary to establish the definitive diagnosis. In the critically ill patient, right heart catheterization, with measurement of right ventricular oxygen content and pulmonary capillary wedge pressure, often is sufficient to establish the diagnosis. Papillary muscle dysfunction due to infarction of the muscle can also produce severe mitral regurgitation in the early postinfarct period. The murmur of mitral regurgitation and clinical deterioration may develop more slowly with this entity but the hemodynamic effect is similar.

Successful surgical repair of this mechanical defect complicating myocardial infarction was first reported by Austen et al. in 1965. In this case, mitral valve replacement was carried out 2 1/2 months following infarction. Subsequently, Austen and associates reported four survivors in five patients undergoing mitral valve replacement for treatment of this complication. The four survivors were operated upon 2 1/2-14 months after infarction. The one patient operated upon less than 2 weeks after myocardial infarction failed to survive surgery. Similar experience was reported by Cohen et al. Although Horlich et al. reported success with suture reattachment of the ruptured papillary muscle and plication of the mitral annulus, many surgeons prefer valve replacement over direct suture repair of the friable infarcted papillary muscle.

Since the majority of patients with this complication become moribund with left ventricular failure and die within 24 hours or, at most, within 1-2 weeks, surgical repair frequently must be undertaken in the acute postinfarct period. In a recent report Buckley et al. reported two survivors of four patients operated upon with mitral valve replacement within 2-10 days following the acute infarct. In comparison, 15 of 18 patients undergoing mitral valve replacement 3-7 months following infarction are surviving and clinically well. A ruptured papillary muscle was found in 10 in this group and eight had dysfunction of a fibrotic papillary muscle.

It is apparent from accumulated experience that the chance for survival with surgery is improved when several weeks or months have elapsed from the time of infarction. As a general guideline, vigorous supportive medical therapy is continued in the early postinfarct period as long as the patient improves or remains stable with an adequate hemodynamic status. Although the patient may remain sufficiently stable clinically to delay surgery, it is unusual for functional recovery to be sufficient enough to be able to avoid surgery. Clinically the patient presents with the manifestations of severe mitral regurgitation with additional compromise of left ventricular function due to ischemic myocardial disease. Frequently, a patient who develops rupture of a papillary muscle in the acute postinfarct period fails to respond adequately to vigorous supportive medical care. It is imperative to proceed urgently with cardiac catheterization and to define the hemodynamic diagnosis. In the patient who goes into severe left ventricular failure, pulmonary edema, and shock, it may be necessary to initiate prompt circulatory assistance to attain some degree of cardiovascular stabilization in order to proceed with diagnostic study and facilitate preparation of the patient for surgery. In this clinical setting, complete hemodynamic evaluation is usually advisable, including coronary arteriography and left ventriculography in addition to right heart catheterization. For significant salvage of the patient in cardiogenic shock, coronary arterial revascularization and/or infarctectomy may be required in addition to mitral valve replacement. Present experience is too small to allow definition of criteria for patient selection in terms of operability or choice of procedure. In the clinical setting being discussed, the expected mortality with medical therapy is virtually 100%. If there is a significant possibility of salvage with urgent surgery, it obviously must be considered, particularly in the younger-aged patient.

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Ventricular Aneurysm and Left Ventricular Wall Dyskinesia

Left ventricular aneurysm develops as a complication of myocardial infarction in approximately 10–15% of cases. The incidence depends upon the extent and detail of diagnostic efforts made in each case. With combined electrocardiography, fluroscopy, and left ventriculography the incidence of ventricular aneurysm may be as high as 20–25%.

The aneurysm involves the anterior wall and/or apex in 80% of cases and is usually associated with an anterior infarction. An acute aneurysm may develop in the early postinfarct period and be associated with cardiogenic shock, severe congestive heart failure, recurrent ventricular arrhythmias, or not contribute significantly to the patient's clinical status. Cardiogenic shock is more commonly associated with a massive infarct with loss of greater than 40% of the left ventricular wall.

Clinical features and management of this type of patient have been discussed previously. Frequently, a ventricular aneurysm is not clinically apparent for weeks or even months after infarction. The electrocardiogram may demonstrate persistently elevated S-T segments during recovery from myocardial infarction which should raise the clinical suspicion of ventricular aneurysm. The characteristic S-T changes, inverted T waves, and persistent Q waves are present in 70–80% of patients with a clinically significant aneurysm.

The clinical features of left ventricular aneurysm are usually those related to left ventricular dysfunction, particularly persistent cardiac failure. Persistent or recurrent ventricular arrhythmias, although relatively uncommon, may be the primary clinical manifestation of ventricular aneurysm. Occasionally, peripheral arterial thromboembolic complications may be the first indication of a ventricular aneurysm. Layered thrombus is nearly always present in ventricular aneurysms and probably is responsible for the majority of peripheral embolic complications of myocardial infarction.

The physiologic consequences of an acute ventricular aneurysm in a nonischemic heart were studied experimentally by Austen et al. With a large ventricular aneurysm, there was a marked depression in left ventricular stroke work, cardiac output, and mean aortic arterial pressure. It is apparent that these changes would be even more marked if significant myocardial ischemia were present in addition to the mechanical effect of the aneurysm itself. The left ventricular ejection fraction decreases and the left ventricular end-diastolic volume increases in the presence of an aneurysm or a significant area of left ventricular akinesia.

Recovery in performance of the left ventricle has been shown to be not only related to improvement in the ventricular ejection fraction and end-diastolic volume but also dependent upon the capacity for fiber shortening in adjacent myocardium. When there is involvement of greater than 25% of the left ventricular wall, the physiologic limit of compensatory myofibril shortening is reached, and maintenance of left ventricular stroke volume can be achieved only by increase in ventricular volume (Starling mechanism).

The natural history of large ventricular aneurysms or areas of akinesis is usually that of progressive and eventually intractable left ventricular failure. In a retrospective study, Shlichter, Hellerstein, and Katz found that 73% of patients with left ventricular aneurysms died within 3 years and 88% within 5 years due to heart failure, recurrent myocardial infarction, or peripheral arterial embolization. The study of Douglas, Sferazza, and Marici yielded similar results.

Surgical excision of a left ventricular aneurysm with cardiopulmonary bypass was first achieved by Cooley et al. in 1958. The procedure has had extensive clinical application subsequently with an acceptably low operative mortality (5–15%). The overall clinical results of aneurysm resection have been quite good. Favaloro et al. analyzed a series of 130 patients undergoing ventricular aneurysm resection between 1959 and 1967. Complete follow-up ranging from several months to 7 years was available in 68 patients. Forty-nine were living and 41 of this group...
were free of symptoms at the time of their report. When possible, surgical resection of a ventricular aneurysm is delayed 3 months or more following infarction in order to allow demarcation of aneurysmal scar from that of viable adjacent myocardium.

As is true of all types of surgery for the complications of coronary artery disease, resection of left ventricular aneurysm or large akinetic areas is strictly palliative. The eventual prognosis depends upon the course of the underlying disease. Recently there has been increasing enthusiasm for combining coronary arterial revascularization with aneurysm or akinetic wall resection. This is true particularly for those patients who have significant angina in addition, which suggests the presence of ischemic myocardium in addition to the inert scar of the aneurysm. Whether direct coronary arterial revascularization can alter the natural course of coronary artery disease remains unknown. Only the passage of time and careful analysis of the results of surgery can provide the answer.

The socioeconomic implications of surgery and overall care of the patient with life-threatening complications of acute myocardial infarction present a major potential challenge to medical care delivery. Provision of adequate facilities, equipment, and trained personnel to deal effectively with the problems and therapeutic approaches outlined in the preceding pages will obviously be difficult outside of major institutions that can absorb sizeable numbers of this type of critical care patients, in addition to the many salvageable patients requiring more conventional care. The problem is of such great magnitude simply because of the large numbers of patients involved. An unpleasant, but nevertheless pertinent, question is that related to priorities. Should hospitals, physicians, and possibly the federal government devote large numbers of highly trained personnel, facilities, time, and money to highly complex and perhaps overly heroic attempts to salvage patients in an extremely high-risk group when delivery of general medical care to the public as a whole may not be entirely optimal? The extensive time and personnel commitment required in the care of these patients may subtract subtly but recognizeably from the care of eminently salvageable and rehabilitable patients. An appropriate example is the growing volume of patients with symptomatic coronary artery disease who must wait 3–4 months for elective surgery due to the volume load of patients undergoing cardiac surgery in major centers. The risk of the necessary delay in symptomatic patients awaiting surgery has been estimated at a 3% mortality per month. Despite these considerations, it is exceedingly difficult to deny any critically ill patient suffering the effect of a complication of myocardial infarction the potential benefit of surgery, however small the chances of success are. The dilemma at present is not easily soluble. The passage of time with a continued aggressive approach to therapy balanced with careful detailed analysis of the results should eventually provide the appropriate answers.

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