It has become clear that to manage most patients supportively, in hopes of deferring operation, is to deprive the great majority of those with postinfarction ventricular septal rupture of the benefits of definitive surgery before irreversible damage caused by peripheral organ ischemia has occurred. Thus, the diagnosis of postinfarction ventricular septal rupture can be regarded as its own indication for operation. Preoperative management is directed towards stabilization of the hemodynamic condition so that peripheral organ perfusion can be best maintained while the patient is being made ready for surgery. The goals of preoperative management are to (1) reduce the systemic vascular resistance, and thus, the left-to-right shunt; (2) maintain cardiac output and arterial pressure to ensure peripheral organ perfusion; and (3) maintain or improve coronary artery blood flow. This is best accomplished by the intra-aortic balloon pump (IABP). Although counterpulsation produces an overall improvement in the patient’s condition, a complete correction of the hemodynamic picture cannot be obtained. Peak improvement occurs within 24 hours and little further benefit has been observed with prolonged balloon pumping. Pharmacological therapy with inotropic agents and diuretics should be instituted promptly. However, it must be stressed that pharmacological therapy is intended primarily to support the patient in preparation for operation and should not, in anyway, delay urgent operation in the critically ill patient. We now admit patients with postinfarction septal rupture directly to the surgical intensive care unit rather than to the coronary care or medical intensive care unit.

Operative Principles
The first repair by Denton A. Cooley et al of an acquired ventricular septal defect was accomplished using an approach through the right ventricle with incision of the right ventricular outflow tract. This approach, which was adapted from surgical techniques for closure of congenital ventricular septal defects, proved to be disadvantageous for many reasons. Exposure of the defect was frequently less than optimal, particularly for defects located in the apical septum. It involved unnecessary injury to normal right ventricular muscle and interruption of collaterals from the right coronary artery. Finally, it failed to eliminate the paradoxical bulging segment of infarcted left ventricular wall. Subsequently, Heimbecker et al introduced, and others adopted, a left-sided approach (left ventriculotomy) with incision through the area of infarction. Such an approach frequently incorporates infarctectomy and aneurysmectomy, together with repair of septal rupture. Experience with a variety of techniques for closure of postinfarction ventricular septal rupture has led us to the evolution of eight basic principles (Table 1). Adherence to these principles in the closure of septal defects in different locations has led to the evolution of individualized approaches to apical, anterior, and inferoposterior septal defects.

<table>
<thead>
<tr>
<th>TABLE 1. Principles of Repair of Postinfarction Ventricular Septal Defects</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Expeditious establishment of total cardiopulmonary bypass with moderate hypothermia and meticulous attention to myocardial protection</td>
</tr>
<tr>
<td>(2) Transinfarct approach to ventricular septal defect with the site of ventriculotomy determined by the location of the transmural infarction</td>
</tr>
<tr>
<td>(3) Thorough trimming of the left ventricular margins of the infarct back to viable muscle to prevent delayed rupture of the closure</td>
</tr>
<tr>
<td>(4) Conservative trimming of the right ventricular muscle as required for complete visualization of the margins of the defect</td>
</tr>
<tr>
<td>(5) Inspection of the left ventricular papillary muscles and concomitant replacement of the mitral valve only if there is frank papillary muscular rupture</td>
</tr>
<tr>
<td>(6) Closure of the septal defect without tension, which, in most instances, will require the use of prosthetic material</td>
</tr>
<tr>
<td>(7) Closure of the infarctectomy without tension with generous use of prosthetic material as indicated, and epicardial placement of the patch to the free wall of the left ventricle to avoid strain on the friable endocardial tissue in patients with posterior defects</td>
</tr>
<tr>
<td>(8) Buttressing of the suture lines with pledges or strips of Teflon felt or similar material to prevent sutures from cutting through friable muscle</td>
</tr>
</tbody>
</table>
Patients are anesthetized using a Fentanyl-based anesthetic regimen. Pancuronium is selected as the muscle relaxant to prevent bradycardia. Pulmonary bed vasodilators such as Dobutamine are avoided to minimize left-to-right shunt fraction. Cardiopulmonary bypass is accomplished with bicaval venous drainage. Systemic cooling to 25°C is employed. Cardiac standstill is achieved with cold, oxygenated, dilute blood cardioplegia\textsuperscript{10,11} using antegrade induction followed by retrograde perfusion through the coronary sinus. Although a number of myocardial protection strategies are currently available, we,\textsuperscript{12} and others,\textsuperscript{13} continue to use cold oxygenated, dilute blood cardioplegia to protect the heart during surgical correction of a ventricular septal defect. A total of 1,200 to 2,000 mL of cardioplegia solution is delivered depending on the size of the heart and the degree of hypertrophy.\textsuperscript{12} Although we have not employed warm cardioplegic induction,\textsuperscript{14} we do administer warm reperfusion cardioplegia just before removing the aortic cross clamp.\textsuperscript{15} Patients with multivessel coronary disease and critical coronary stenoses are revascularized before opening the heart in order to optimize myocardial protection. In most of these patients, the left internal mammary artery is not used.
The technique of apical amputation for apical defects associated with anteropapical infarction was described by Daggett et al in 1970. (A and B) An incision is made through the infarcted apex of the left ventricle. Excision of the necrotic myocardium back to healthy muscle results in amputation of the apical portion of the left ventricle, right ventricle, and septum. The remaining apical portions of the left and right ventricle free walls are then approximated to the apical septum. (C and D) This is accomplished by means of a row of interrupted mattress sutures of Tevdek that are passed sequentially through a buttressing strip of Teflon felt, the left ventricular wall, a second strip of felt, the interventricular septum, a third strip of felt, the right ventricular wall, and a fourth strip of felt. All sutures are placed before any are tied. After all sutures have been tied, the closure is reinforced with an additional over-and-over suture, as in ventricular aneurysm repair, to ensure hemostasis of the ventriculotomy closure (not shown). (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)
The approach to anterior ventricular septal defects is by a left ventricular transinfarct incision, parallel to the anterior descending branch of the left coronary artery, with infarctectomy (Fig 3). (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)

Small defects beneath anterior infarcts can be closed by the technique of plication as suggested by Shumaker.\textsuperscript{16} (A) This involves approximation of the free anterior edge of the septum to the right ventricular free wall using mattress sutures of 0 Tevdek over strips of felt. (B-D) The transinfarct incision is then closed with a second row of mattress sutures buttressed with strips of felt. An over-and-over running suture is then used to ensure a secure ventriculotomy closure (not shown). (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)
Most anterior defects require closure with a prosthetic patch (Hemashield or Dacron) in order to avoid tension that could lead to disruption of the repair. (A) After debridement of necrotic septum and left ventricular muscle, a series of pledgeted interrupted mattress sutures are placed around the perimeter of the defect. Along the posterior aspect of the defect, sutures are passed through the septum from right side to left. Along the anterior edge of the defect, sutures are passed from the epicardial surface of the right ventricle to the endocardial surface. All sutures are placed before the patch is inserted. (B) All sutures are then passed through the edge of a synthetic patch, which is seated on the left side of the septum. Each suture is then passed through an additional pledget and then all are tied. We use additional pledgets on the left ventricular side overlying the patch (C) to cushion each suture as it is tied down to prevent cutting through the friable muscle. The edges of the ventriculotomy are then approximated by a two-layer closure consisting of interrupted mattress sutures passed through buttressing strips of Teflon felt. An over-and-over running suture completes the repair (not shown). (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)
Closure of posterior septal defects, which result from transmural infarction in the distribution of the posterior descending artery, has posed the greatest technical challenge. Early attempts at primary closure of these defects by simple plication techniques similar to those used in the repair of anterior defects was frequently unsuccessful because the sutures tore out of soft, friable myocardium that had been closed under tension. This resulted in either reopening of the defect or catastrophic disruption of the infarctectomy closure. Exposure at operation is achieved by dislocating the heart up out of the pericardial well and retracting the heart cephalad as in the performance of a distal vein bypass to the posterior descending coronary artery. (A) The margins of the defect may involve the inferior aspects of both ventricles, or of the left ventricle only. (B) A transinfarct incision is made in the left ventricle, and the left ventricular portion of the infarct is excised, exposing the septal defect. Complete excision of the left ventricular portion of the infarct is important to prevent delayed rupture of the ventriculotomy repair. The left ventricular papillary muscles are inspected. Only if there is frank papillary muscle rupture is mitral valve replacement performed. When it is indicated, we prefer to perform mitral valve replacement through a separate conventional left atrial incision, to avoid trauma to the friable ventricular muscle. After all infarcted left ventricular muscle has been excised, a less aggressive debridement of the right ventricle is accomplished, with the goal of resecting only as much muscle as is necessary to afford complete visualization of the defect. Using this technique, delayed rupture of the right ventricle has not been a problem. (C and D) If the posterior septum has cracked or split from the adjacent ventricular free wall without loss of a great deal of septal tissue, then the septal rim of the posterior defect may be approximated to the edge of the diaphragmatic right ventricular free wall using mattress sutures buttressed with strips of Teflon felt. The surgeon can perform repair of posterior septal rupture to best advantage by standing at the left side of the supine patient. Following closure of the septal defect, the resultant left ventricular free wall defect, created by the infarctectomy, should be closed by a prosthetic patch as described in the next section. (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)
The cross-sectional view of the completed repair illustrates the restoration of relatively normal ventricular geometry, which is accomplished by the use of appropriately sized prosthetic patches. (Reprinted with permission from The McGraw-Hill Companies. Madsen, JC, Daggett, WM: Cardiac Surgery in the Adult, 1996.)
COMMENTS

Weaning From Cardiopulmonary Bypass and Highlights of Postoperative Care

Intraoperative transesophageal echocardiography is essential to assess ventricular function, ventricular dimensions, residual shunt, and mitral regurgitation when weaning from bypass. The two most common problems encountered while separating from bypass following repair of a postinfarction ventricular septal defect are low cardiac output and bleeding. Although the treatment of low cardiac output following cardiac surgery is beyond the scope of this article, a few agents and principles are worth mentioning. First, most of these patients will have had an IABP inserted before surgery. If not, one should be inserted in the operating room, especially if the low output state is secondary to left ventricular dysfunction. Also, IABP may benefit patients with right ventricular failure by improving right coronary artery blood flow caused by diastolic augmentation. We have found intravenous milrinone, a phosphodiesterase inhibitor, to be very effective in reversing low output states secondary to left ventricular dysfunction. Milrinone possesses a balance of inotropic and vasodilatory properties which together produce an increase in cardiac output and reduction in right and left filling pressures and systemic vascular resistance. It is less arrhythmogenic than dobutamine, causes less hypotension than amrinone, and is not associated with thromboembolism. We have successfully treated right ventricular failure with a prostaglandin E1 infusion (0.5 to 2.0 μg/min) into the right heart, counterbalanced with a norepinephrine infusion titrated into the left atrium. Inhaled nitric oxide (20 ppm-80 ppm) which selectively dilates the pulmonary circuit has also proven efficacious in the treatment of right heart failure. More recently, it has been shown that intravenous adenosine (50 μg/kg/min) is as effective as nitric oxide in diminishing pulmonary vascular resistance and increasing cardiac output without diminishing systemic hemodynamics, although we have had no personal experience with this treatment modality. In our experience, inability to separate from bypass has not been noted if the repair has been successful. However, if a patient could not be weaned from bypass using conventional therapy and was under 70 years old with no residual hemodynamically significant lesion, we would consider a ventricular assist device.

To prevent postpump coagulopathy, we begin antiarrhythmic therapy with e-aminocaproic acid (Amicar) before commencing cardiopulmonary bypass. Amicar is administered by loading patients with 10 g before commencing bypass and then adding another 10 g to the pump prime. During the procedure Amicar is continuously infused at 1 g/hr for the duration of surgery. We avoid giving more than 30 g of Amicar. Postpump suture line bleeding may be reduced by application of a fibrin sealant to the ventricular septum around the septal defect before formal repair. Biological glue may be effective in controlling bleeding suture lines following repair but we have had limited experience with these compounds. As a last resort, Baldwin and Cooley have suggested insertion of a left ventricular assist device solely as an adjunct to the repair of friable or damaged myocardium to reduce left ventricular distension and thus, control bleeding. In the ICU, early postoperative diuresis and positive end-expiratory pressure ventilation are used to decrease the A-a gradient induced by the increased extravascular pulmonary water associated with cardiopulmonary bypass. Once the patient has warmed, we commonly use an intravenous infusion of Lasix combined with mannitol (1 g of Lasix in 400 mL of 20% mannitol) at a rate of 1 mL to 20 mL per hour to keep the urine output greater than 100 mL per hour. If renal function has been compromised preoperatively, continuous veno-venous hemofiltration (CVVH) is employed postoperatively and managed by our ICU nurses. Intractable postoperative ventricular arrhythmias secondary to reperfusion injury are sometimes difficult to control using standard therapy. We have been impressed with the efficacy of intravenous amiodarone in these situations (10 to 20 mg/kg over 24 hours).

Recurrent or residual septal defects have been diagnosed by Doppler color flow mapping early or late postoperatively in 10% to 25% of patients. Recurrent defects may be caused by reopening of a closed defect, to the presence of an overlooked defect, or to the development of a new septal rupture during the early postoperative period. These recurrent defects should be closed when they cause symptoms or signs of heart failure or when the calculated shunt fraction is large (Qp/Qs > 2.0). When they are small (Qp/Qs < 2.0) and either asymptomatic or controlled with minimal diuretic therapy, a conservative approach is reasonable and late spontaneous closure can occur. Intervention in the catheterization laboratory with a clam-shell device may be useful in closing symptomatic residual or recurrent defects postoperatively.
REFERENCES


From the Cardiac Surgical Unit, Massachusetts General Hospital, Boston, MA. Address reprint requests to Joren Madsen, MD, DPhil, Cardiac Surgical Unit, Massachusetts General Hospital, Boston, MA 02114-2996. Copyright © 1997 by W.B. Saunders Company 1085-5637/97/0202-0006$5.00.