

Surgical Repair of Postinfarction Ventricular Septal Defect

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Thirty-one patients underwent repair of postinfarction ventricular septal defect (VSD) from 1980 to 1989. All patients were in New York Heart Association functional class IV, and 15 of them were in cardiogenic shock when operated on. Coronary arteriography was performed in all patients before surgery: nine had one-vessel, 11 had two-vessel, and 11 had three-vessel disease. The VSD was anterior in 15 patients and posterior in 16. The operative technique evolved over the years from a fairly extensive infarctectomy and reconstruction of the septum and right and left ventricular walls with a double Dacron patch, to minimal or no infarctectomy and closure of the VSD by excluding the infarcted muscle from the left ventricular cavity. This is accomplished by suturing a single patch of bovine pericardium to healthy endocardium surrounding the infarcted muscle. The right ventricle is left intact. Overall mortality was 10%, with three operative deaths. All deaths occurred in patients in cardiogenic shock who had three-vessel coronary artery disease. Thus, the mortality for patients in shock was 20%, and the mortality for patients with three-vessel disease was 27%. The operative mortality for patients with posterior VSD was twice as high as in patients with anterior VSD. However, univariate analysis of various clinical, hemodynamic, and operative variables indicated that only three-vessel disease was predictive of operative mortality. Because the number of patients was small and the overall operative mortality relatively low, the results of this analysis may not be valid. Other investigators have shown that right ventricular dysfunction is an important determinant in the development of cardiogenic shock as well as in the outcome of these patients. Therefore, we believe that our current operative technique of closing the VSD by excluding the ventricular cavity with a single patch of bovine pericardium without resecting part of the right ventricle is physiologically sound and should improve surgical results. (*Circulation* 1990;82(suppl IV):IV-243-IV-247)

The first successful operation for postinfarction ventricular septal defect (VSD) was reported by Cooley and associates in 1957¹; the repair was performed 9 weeks after the diagnosis of septal rupture. Although rupture of the interventricular septum complicates 1–2% of all patients who suffer myocardial infarction,^{2,3} the surgical treatment of this disease remains a major challenge. Few heart operations can be as humbling as repair of an acute rupture of the interventricular septum in a patient in cardiogenic shock with an acute posterior myocardial infarction. Surgeons continue to search for new approaches to improve the outcome of treatment.^{4–8} Although the operative mortality has decreased during the past three decades, it is still relatively high. This study reviews our experience with surgery for postinfarction VSD during the past decade.

Methods

Patients

Thirty-three patients were referred to one of us (T.E.D.) for surgical treatment of postinfarction VSD from January 1980 to December 1989. Two patients were not accepted for surgery because of advanced age. Thirty-one underwent surgery and are the subject of this study.

There were 16 women and 15 men whose mean age was 66 years (range, 48–77). Every patient had a documented myocardial infarction; the mean interval between the infarction and the operation was 13 days (range, 1–35 days in 28 patients). Three patients had infarction at 2, 6, and 8 months before the operation. All patients were in New York Heart Association functional class IV when referred for treatment. Fifteen patients developed cardiogenic shock (systolic blood pressure less than 80 mm Hg, anuria or oliguria, elevated creatinine, and cool or clammy skin) before operation and were managed initially with an intra-aortic balloon pump. Fourteen of these 15 patients also required dopamine or dobutamine to

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maintain adequate blood pressure. Preoperatively, an intra-aortic balloon pump was inserted in five other patients because of pulmonary edema. All 20 patients who received a preoperative intra-aortic balloon pump underwent surgery the same day that this device was inserted. The remaining patients were operated on urgently (eight patients) or electively (three patients).

Twenty patients had a history of systemic hypertension, six were diabetic, and six had a history of an old myocardial infarction. Two patients had had myocardial revascularization.

All 31 patients underwent coronary angiography before surgery, and 28 also had contrast left ventriculography. Nine patients had one-vessel, 11 had two-vessel, and 11 had three-vessel coronary artery disease. The VSD was anterior in 15 patients and posterior in 16. Contrast ventriculography, echocardiography, or both revealed mild impairment of left ventricular function in 18 patients, moderate in 11, and severe in three. It also was possible to estimate right ventricular function by echocardiography or contrast ventriculography in 28 patients: nine had mild, 14 had moderate, and five had severe impairment of right ventricular function. The mean pulmonary artery pressure ranged from 10 to 41 mm Hg (mean \pm SD, 27 \pm 7 mm Hg). The calculated shunt ranged from 1.6:1 to 5.0:1 (mean \pm SD, 2.9 \pm 0.8:1). Of the 15 patients in cardiogenic shock, eight had posterior and seven had anterior VSD.

The following variables were analyzed to determine their value as predictive of operative mortality: age, sex, diabetes, systemic hypertension, cardiogenic shock, length of time between myocardial infarction and operation, coronary artery disease, left and right ventricular function, mean pulmonary artery pressure, left-to-right shunt, VSD location, operative technique, patch material, cardioplegia, myocardial revascularization, aortic crossclamping, and cardiopulmonary bypass times. Each variable was assessed by univariate analysis using Fisher's exact test or unpaired *t* test. The life-table method was used to calculate actuarial survival.

Operations

All operations were performed by one surgeon. Moderate systemic hypothermia was used during cardiopulmonary bypass. Crystalloid cardioplegia was used until 1984; thereafter, cold blood cardioplegia was used. Myocardial revascularization was performed in 22 patients who had two- or three-vessel coronary artery disease. Only saphenous veins were used, and both the distal and proximal anastomoses were performed before repair of the VSD for better myocardial protection. The mean number of grafts was 1.8 \pm 0.8 per patient (range, 1-4). The mean aortic cross-clamping time was 62 \pm 24 minutes, and mean cardiopulmonary bypass time was 91 \pm 26 minutes.

Several modifications were made in the operative technique for VSD repair during the decade of this study. Until 1985, a technique similar to that de-

scribed by Daggett and associates⁴ was used in 10 patients. Most of the infarcted myocardium was resected and the septum was reconstructed with two Dacron patches, one on each side of the septum, which were then sewn to the free wall of the right and left ventricles. A third patch sometimes was used in posterior VSD.⁴ Because of problems with bleeding through the suture lines, the patch material was changed to autologous or bovine pericardium, and we also began to resect less infarcted myocardium.⁶ This technique was used in 13 patients.

During the past 2 years, in an attempt to preserve as much right ventricle as possible,² a single patch of bovine pericardium with no resection of the infarcted myocardium has been used. This technique was used in eight patients. The repair is done through a left ventriculotomy in the infarcted area, parallel to the interventricular artery. A properly tailored bovine pericardial patch is sutured to healthy endocardium around the infarct in the septum and lateral ventricular wall using a running 4-0 polypropylene suture in such a way as to exclude the VSD and the infarcted muscle from the left ventricular cavity. The size and shape of this patch must be such as to preserve the normal shape of the left ventricle. The operation for anterior or posterior VSD is essentially the same. In posterior VSD, the patch may have to be sutured to the mitral annulus when the defect is very proximal in the septum. It may also have to be sutured to the base of the posterior papillary muscle, which is frequently infarcted. The ventriculotomy is closed with large buttressed sutures, and nothing is done about the VSD itself (Figures 1 and 2). We refer to this technique as endocardial repair of the ventricular wall.^{6,9}

Results

Three patients died after surgery, for an overall operative mortality of 10%. The cause of death was a technical problem (uncontrollable bleeding) in one patient with posterior VSD, low cardiac output in one with anterior VSD, and a postoperative stroke (40 days after surgery) in one with posterior VSD. Table 1 shows the operative mortality in some subgroups of patients. Univariate analysis indicated that only three-vessel coronary artery disease was statistically significant as a predictor of operative mortality. Because the number of patients was small and the operative mortality relatively low, the results of this analysis may not be valid.

Postoperative complications were common. Twenty patients had an intra-aortic balloon pump inserted before surgery, and seven additional patients required this device after surgery because of low cardiac output. The intra-aortic balloon pump was removed after a mean of 1.9 \pm 0.8 days. Twenty patients required dopamine or dobutamine after surgery for 2.4 \pm 1.0 days; five patients required peritoneal or hemodialysis; 12 patients needed assisted ventilation for more than 48 hours; seven patients had ventricular dysrhythmias. The mean stay in the intensive care unit was 6.2 \pm 8.0 days (range, 2-40).

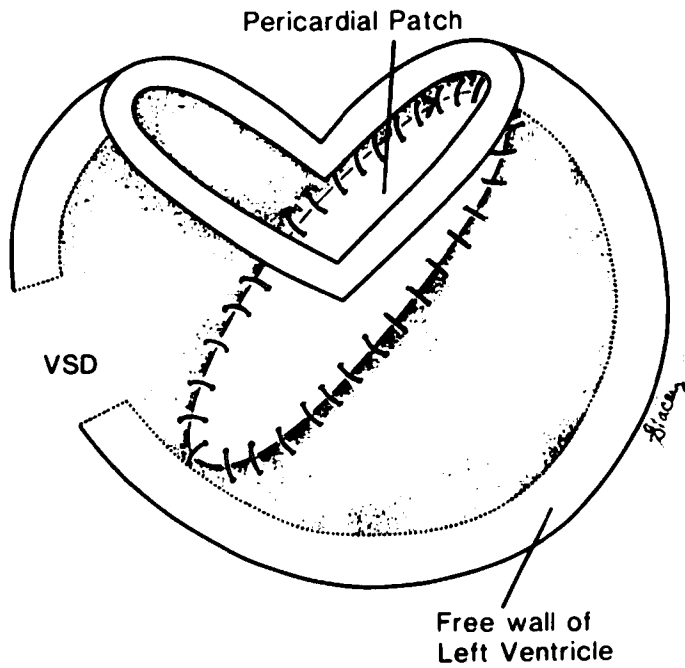


FIGURE 1. Operative techniques used to repair postinfarction ventricular septal defect (VSD). The bovine pericardial patch initially is sutured to healthy endocardium of the septum and then to the free wall of the left ventricle, excluding the VSD and the infarcted muscle from the left ventricular cavity.

The mean hospital stay was 15.1 ± 8.8 days (range, 7–40).

Twenty-eight hospital survivors were followed up for 1–113 months (mean, 25). There was one late death at 13 months due to cancer of the stomach. The 5-year actuarial survival was $83 \pm 7\%$. Three patients suffered additional myocardial infarctions, and one of them developed a second postinfarction VSD 5 years after the initial operation. This patient underwent a second repair but developed a recurrence with a small shunt ($<1.5:1$). Although this patient is very disabled, no further surgery was performed because

he has severe left ventricular dysfunction. One patient suffered a stroke 41 months after surgery. The freedom from any complication was $53 \pm 13\%$ at 5 years. Twenty-four patients (89%) are functionally in New York Heart Association class I or II. Patients who had the VSD repaired by different methods have a similar postoperative functional class.

Discussion

One of the earliest series on surgical treatment of postinfarction VSD was published by Daggett et al.^{4,10} Their initial operative mortality was 47% but

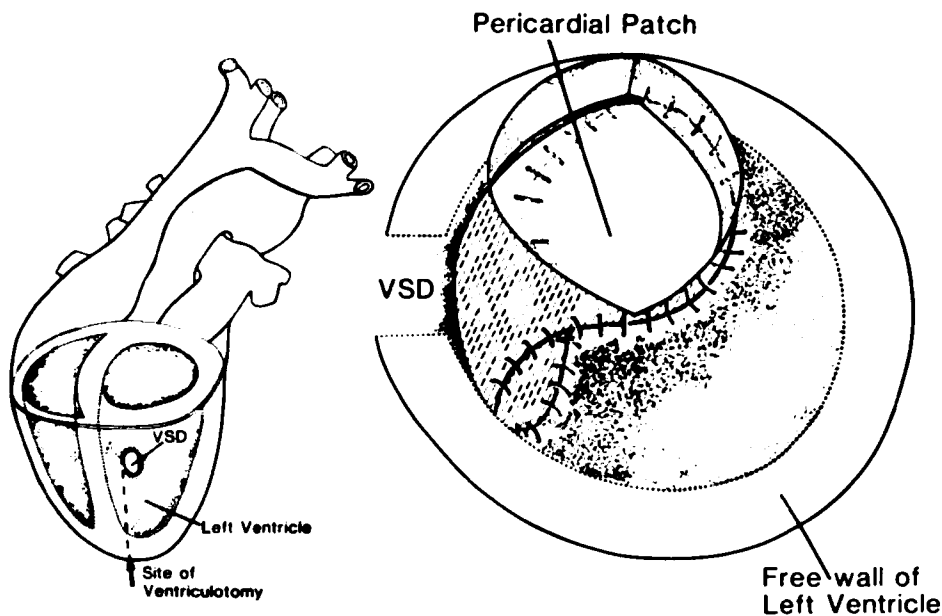


FIGURE 2. Ventricular septal defect (VSD) repair after completion. The shape of the left ventricular cavity is maintained after completion of the repair.

TABLE 1. Operative Mortality in Subgroups of Patients

Variable	No. of patients	No. of deaths (%)	<i>p</i>
Three-vessel disease			
No	20	0 (0)	
Yes	11	3 (27)	0.035
Cardiogenic shock			
No	16	0 (0)	
Yes	15	3 (20)	NS
Location of VSD			
Anterior	15	1 (6.6)	
Posterior	16	2 (13)	NS
Time interval from infarction to operation			
<7 days	11	2 (18)	
>7 days	20	1 (5)	NS
Right ventricular dysfunction (28 patients)			
Mild	9	1 (10)	
Moderate	14	1 (7)	
Severe	5	0 (0)	NS
Operative technique			
Two-patch Dacron	10	1 (10)	
Two-patch pericardium	13	2 (17)	
One-patch pericardium	8	0 (0)	NS

NS, not significant; VSD, ventricular septal defect.

fell to 25% after 1975. Other groups reported similar results.¹¹⁻¹⁶

Cardiogenic shock has been identified as one of the most important determinants of operative mortality.^{2,11-15} Cummings and associates,¹⁵ using a logistic regression analysis in 33 patients with postinfarction VSD, found that cardiogenic shock was an independent predictor of operative mortality. The operative mortality in patients in cardiogenic shock in most reports ranged from 47% to 88%.^{2,11-15} Our operative mortality in 15 patients in cardiogenic shock was 20%. We had no operative mortality in 16 hemodynamically stable patients. The difference between these two groups of patients did not reach statistical significance because of the small number of patients and the relatively low operative mortality. It is interesting to note that only one of our patients died because of low cardiac output; the other two died from noncardiac causes.

Another important determinant of operative mortality has been the site of the VSD. Repair of posterior VSD consistently has been associated with a higher operative mortality than repair of anterior VSD.^{2,10-16} Several investigators have found in postmortem examinations that the extent of right ventricular infarction in patients with posterior VSD was significantly greater than in patients with anterior VSD.^{2,11,15} In a detailed review of 25 patients with postinfarction VSD, Moore and associates² found that nonsurvivors had greater impairment of right ventricular function and greater elevation of right ventricular and atrial pressures when compared with survivors. Shock was associated with a 91% mortality and was more common after inferior infarction.²

Mortality for inferior VSD was 73%, compared with 30% for anterior VSD.² Fananapazir and colleagues¹⁷ reported a 20% mortality in patients with good right ventricular function, compared with 76% mortality in patients with poor right ventricular function. Our operative mortality in patients with posterior VSD was twice as high as in patients with anterior VSD. We found no correlation between operative mortality and right ventricular dysfunction. Indeed, neither of the two deaths in posterior VSD was due to heart failure. One patient died because of technical problems with the operation and the other suffered a postoperative stroke. Repair of posterior VSD is, however, more difficult than repair of anterior VSD. This also may contribute to the higher operative mortality in patients with posterior VSD.

The time interval between the myocardial infarction and the operation also has been found to be important in determining the outcome of treatment.^{10,12,13} The shorter this interval, the worse the outcome of surgery.^{10,12,13,18} In a recent review by Hill and Stiles¹⁸ of 48 consecutive patients operated on within 30 days from the myocardial infarction at multiple centers, the operative mortality was 67%, and it was not affected by age, location of the VSD, or concomitant myocardial revascularization. Delay of repair for 6-8 weeks results in an improved surgical outcome, but at the cost of the death of many patients who might otherwise be saved by early operation.^{2,12-15} We share the view of those surgeons who believe that patients with postinfarction VSD should undergo surgery soon after the diagnosis is established.¹⁴

The extent of coronary artery disease did not seem to affect the outcome of patients with postinfarction VSD in other series.^{2,11-18} However, three-vessel coronary artery disease was the only statistically significant variable in our experience. Obviously, because of the small number of patients, this finding may not be valid.

Our improved results with surgery for postinfarction VSD could be due to the fact that most of our patients underwent surgery in the past 5 years. Most previous reports covered the experience of two or three decades.¹⁰⁻¹⁶ The operative technique that evolved in our institution was directed to correct problems that we encountered as our surgical experience increased. We first modified the patch material. Autologous or bovine pericardium was used because of technical problems with Dacron graft, particularly in posterior VSD. Bleeding along the suture lines was practically eliminated when we began to use pericardium.⁶ Next, we limited the resection of infarcted myocardium, particularly of the right ventricle, because of reports indicating the importance of the right ventricle in these patients.^{2,11,15-17} Finally, to avoid any further trauma to the right ventricle, we changed the patching technique and performed the whole operation on the left side of the heart by excluding the VSD and the infarcted myocardium from the left ventricle through an endocardial repair with a properly tailored pericardial patch. Because the right ventricle appears to be an important determinant in the outcome of this operation,^{2,11,14,15} our single-patch technique should improve the results of surgery for postinfarction VSD. Our last eight patients underwent this method, and there were no deaths or serious postoperative complications despite the fact that four patients were in cardiogenic shock when surgery took place.

Note added in proof. Since the submission of this manuscript, we have successfully operated on four additional patients with post-myocardial infarction VSD using the pericardial patch technique. Three patients were in cardiogenic shock and two had posterior VSD. All four are doing well.

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KEY WORDS • interventricular septum • myocardial infarction