Surgery for Post-Myocardial Infarct Ventricular Septal Defect

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Forty-three patients (mean age 62 ± 1 years) were treated for ventricular septal defect (VSD) secondary to myocardial infarction. Whenever possible, operation was postponed until six weeks post-onset chest pain. However, hemodynamic instability, evidenced by cardiogenic shock, refractory pulmonary edema, or a rising blood urea nitrogen (BUN) forced operation in 21 patients within 21 days post-infarct (Group I). In seven patients operation was performed three to six weeks post-infarct (Group II). In only eight patients could operation be delayed beyond six weeks post-infarct (Group III). Clinical deterioration, once begun, progressed rapidly, and could be reversed only temporarily by intra-aortic balloon pumping, used in 26 patients for safe conduct of cardiac catheterization and for peri-operative hemodynamic support. Hospital survival was achieved in 24 of the 36 operated patients (66%). In Group I patients, ten of 21 survived. In Group II, six of seven survived. In Group III, eight of eight patients survived. There have been five late deaths with a mean follow-up of 41 months in survivors. Improved survival has been achieved recently by the greater use of prosthetic material to replace necrotic muscle and by a transinfarct incision regardless of infarct location. Operative mortality before 1973 was 47%; mortality after 1973 was only 18%, with a concomitant reduction of mortality (30%) even in Group I patients.

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RUPTURE OF THE VENTRICULAR septum following myocardial infarction is an uncommon, but usually catastrophic, event. The mortality of this complication is very high: 50% of these patients die within one week and about 85% within two months.[18,19] The dismal prognosis of this subgroup of patients with myocardial infarction has elicited aggressive surgical intervention. Subsequent to the initial report of Cooley in 1957,[3] numerous cardiac surgeons have contributed to the development of surgical principles leading to successful early closure of ventricular septal defects which complicate acute myocardial infarction.[1,6,8,10–12,16,20,21] Extensive reviews of early approaches to this problem have been reported elsewhere.[12,16,21] This study is designed to supplement these reports by presenting our total experience with this complication of infarction, including early therapeutic failures, technical considerations, patients in whom operative intervention was not used and late results.

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Clinical Material

Between June of 1968 and March of 1977, 43 patients were treated at the Massachusetts General Hospital with a diagnosis of ventricular septal defect secondary to myocardial infarction. Most of these patients were referred to our hospital because of acute or chronic cardiac failure refractory to conventional medical therapy. The mean age of this group of patients was 62 ± 1 (SEM) years. There were 24 males and 19 females. A tabulation of these patients is presented in Table 1.

Preoperative Management

In every patient, an effort was made to postpone operation until at least six weeks after the onset of chest pain.[16] But hemodynamic instability (as evidenced by cardiogenic shock, refractory pulmonary edema, rising BUN or prolonged requirement for parenteral catechol-amines) forced operative intervention in 21 patients within three weeks after infarction. In seven patients operation was performed between three weeks and six weeks after infarction. In only eight patients could operation be postponed beyond six weeks after infarction.

Clinical deterioration, once initiated, usually progressed rapidly in these patients. This progression could be *temporarily* reversed by insertion of an intra-

TABLE 1.

Patient #	Age	Date of Operation	Days from Chest Pain to Operation	Location Infarction	Patch	Other Procedures	Hospital Survival	Current NYHA Functional Classification
1	69	8/68	20	posterior	yes	—	no	—
2	59	2/69	240	posterior	yes	—	yes	III
3	70	3/69	11	posterior	yes	—	no	—
4	70	9/69	13	apical	no	—	yes	II
5	60	11/69	120	posterior	yes	—	yes	I
6	60	1/70	26	anterior	no	—	yes	died 1/74
7	55	5/70	7	anterior	no	—	yes	died 11/74
8	67	none	—	anterior	—	—	no	—
9	73	3/71	40	apical	no	—	no	—
10	65	5/71	98	posterior	no	MVR*	yes	died 6/72
11	63	7/71	7	anterior	no	*	no	—
12	58	none	—	posterior	—	—	no	—
13	62	10/71	8	posterior	no	CABG	no	—
14	56	10/71	49	posterior	no	CABG	yes	I
15	62	3/72	38	posterior	no	*	yes	I
16	54	3/72	11	anterior	yes	—	yes	II
17	65	4/72	7	posterior	no	CABG	no	—
18	63	9/72	71	anterior	yes	CABG	yes	I
19	60	none	—	anterior	—	—	no	—
20	61	12/72	9	posterior	no	*	no	—
21	65	1/73	1	anterior	no	—	no	—
22	55	5/73	5	posterior	no	CABG	no	—
23	48	9/73	7	apical	no	—	yes	I
24	50	10/73	24	anterior	no	CABG	yes	II
25	62	12/73	5	posterior	yes	—	yes	died 1/77
26	73	none	—	posterior	—	—	no	—
27	61	1/74	6	anterior	no	—	no	—
28	66	4/74	12	posterior	yes	MVR*	yes	II
29	66	5/74	16	anterior	no	CABG	yes	I
30	60	none	—	anterior	—	—	no	—
31	66	none	—	anterior	—	—	no	—
32	63	11/74	61	anterior	no	—	yes	I
33	65	none	—	anterior	—	—	no	—
34	69	1/75	7	anterior	no	—	no	—
35	54	1/75	34	apical	no	*	yes	II
36	60	3/75	16	posterior	yes	—	yes	II
37	52	3/75	22	anterior	yes	—	yes	I
38	73	9/75	110	posterior	no	—	yes	II
39	66	10/76	49	posterior	yes	CABG	yes	II
40	44	11/76	30	anterior	no	—	yes	died 12/76
41	67	12/76	12	posterior	no	—	no	died 2/77
42	70	12/76	12	anterior	no	—	yes	II
43	63	2/77	6	apical	no	CABG	yes	II

CABG = coronary artery bypass graft.
MVR = mitral valve replacement.
* A significant coronary occlusion in an area other than that of the primary infarct was not bypassed.
Coronary angiography was not performed in cases 1, 9, 19, 21, 26, 30, 31 and 33.

aortic balloon pump (Fig. 1). The balloon provided hemodynamic stability during the time necessary for cardiac catheterization, but further deterioration of the patient's clinical status usually ensued. The balloon pump, then, was important, not as definitive therapy, but as an adjunct which allowed cardiac pathology to be safely ascertained before operation and which provided hemodynamic support in the peri-operative period. After March of 1971, the intra-aortic balloon pump was used for this purpose in 26 patients.

Coronary angiography was not performed in those patients seen prior to March of 1971, but right heart catheterization was used to confirm the diagnosis of ventricular septal defect. After March of 1971, the diagnosis of ventricular septal defect was often confirmed in the coronary care unit by using a Swan-Ganz balloon tip catheter to perform right heart catheterization. Coronary angiography was then performed in 28 of the 34 patients treated after March of 1971. Of the six patients in whom angiography was not performed, one was rushed to the operating room without catheterization (Table 1, #21) and the other five were not offered operation for the reasons listed in Table 2. Angiography invariably revealed occlusive disease

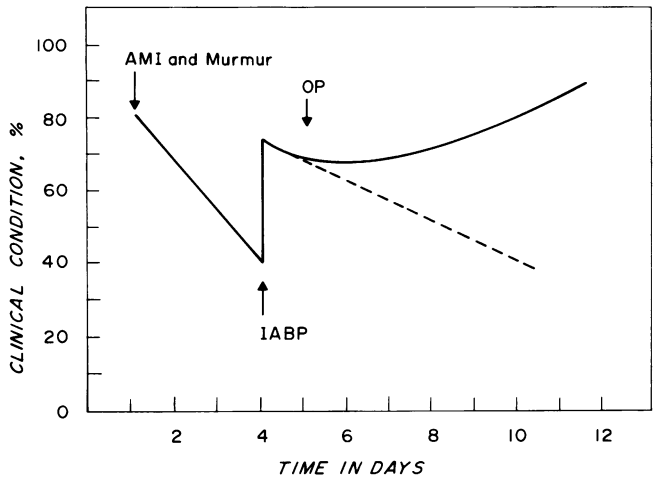

FIG. 1. Diagrammatic illustration of the author's clinical impression of the course followed by many patients after the development of a ventricular septal defect (VSD) as a complication of acute myocardial infarction (AMI). In such patients who have shown deteriorating hemodynamics, the institution of intra-aortic balloon pumping (IABP) will *temporarily* reverse the patient's declining hemodynamic condition. Persistence with intra-aortic balloon pumping as the sole therapeutic mode, however, is associated with continued clinical deterioration as indicated by the dashed line. Thus in such patients it is imperative that confirmatory diagnostic procedures such as cardiac catheterization be followed promptly by urgent operative correction of the mechanical defect in order to take maximum advantage of the temporary restoration of good hemodynamics provided by IABP.

of the anterior descending artery in patients with anterior infarction with an anterior or apical septal defect, and of the posterior descending system in patients with inferior infarction with a posterior septal defect. But while significant occlusive disease of both the anterior descending and posterior descending arteries was present in a majority of the patients, it was *not* a prerequisite for septal perforation after infarction. In eight of 14 patients with anterior infarction, the posterior descending artery was not significantly obstructed at angiography. In four of 14 patients with posterior infarction, the anterior descending artery was widely patent.

TABLE 2. *Clinical Condition of Seven Patients Who were not Offered Operation*

Patient #	Condition
8	Metastatic malignant melanoma
12	Progressive failure on IABP*, very poor contraction in residual myocardium with vessels poorly suitable for grafting
19	Rapidly progressive failure on IABP with massive catecholamine support; death in less than 24 hours
26	Anuric with systolic blood pressure 50 mm Hg on IABP and Levophed
30	Massive small bowel infarction at laparotomy, ? emboli, ? IABP dissection (no post-mortem examination)
31	Neurologically unresponsive on admission, no improvement with IABP and Levophed; systolic blood pressure less than 70 mm Hg
33	Severe oliguria on IABP, Levophed, Dopamine; BUN elevated to 170 mg%

* IABP = intra-aortic balloon pump.

TABLE 3. *Causes of Postoperative Deaths*

Patient Number	Condition Leading to Death
1, 9, 17	Pre-op azotemia, post-op intractable renal failure
3, 21	Progressive pump failure, died in less than one day after operation
11	Pre-op azotemia, inadequate functioning myocardium after infarctectomy, died in operating room
13	Emergency operation for acute deterioration, would not come off cardiopulmonary bypass
20	Sutures would not hold in OR, death in OR
22	Persistent VSD noted in OR after repair, low output postoperatively
27	Pre-op azotemia, persistent VSD, low output and renal failure postoperatively
34	Recurrent aspiration, poor wound healing, sepsis
43	Rerupture of VSD seven weeks after operation

Seven patients in this series were not offered operation. A brief statement of the clinical condition of each patient is listed in Table 2. Patients #26 and #33 with cardiac failure and concomitant severe renal failure were evaluated but not operated in the last three years of the series. Operation was not attempted in these two

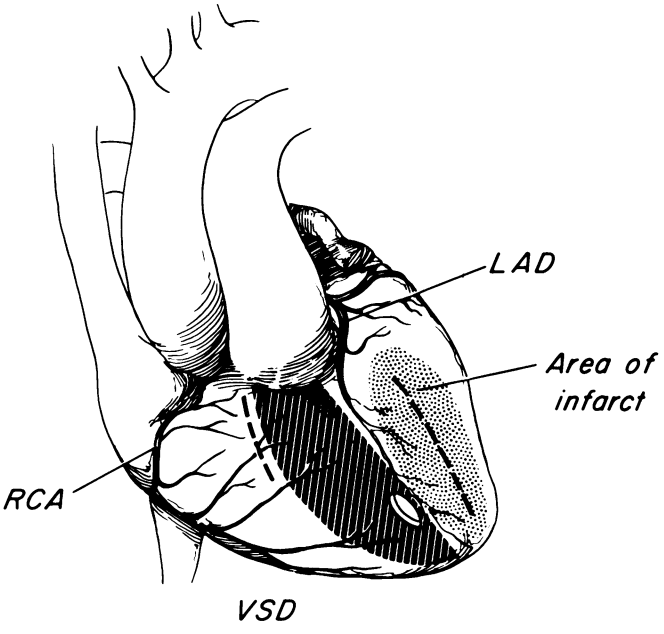

FIG. 2. Illustration of heart with anterior interventricular septal rupture complicating acute anterior myocardial infarction (stippled area). The dashed line on the left indicating an incision in the right ventricular outflow tract, used for repair of congenital ventricular septal defect (VSD) is inappropriate for post-infarct VSD (see text). A transinfarct incision (dashed line in center of infarct), with infarctectomy, gives the best exposure and most complete correction of the pathology. RCA = Right Coronary Artery. LAD = Anterior Descending Branch of Left Coronary Artery.

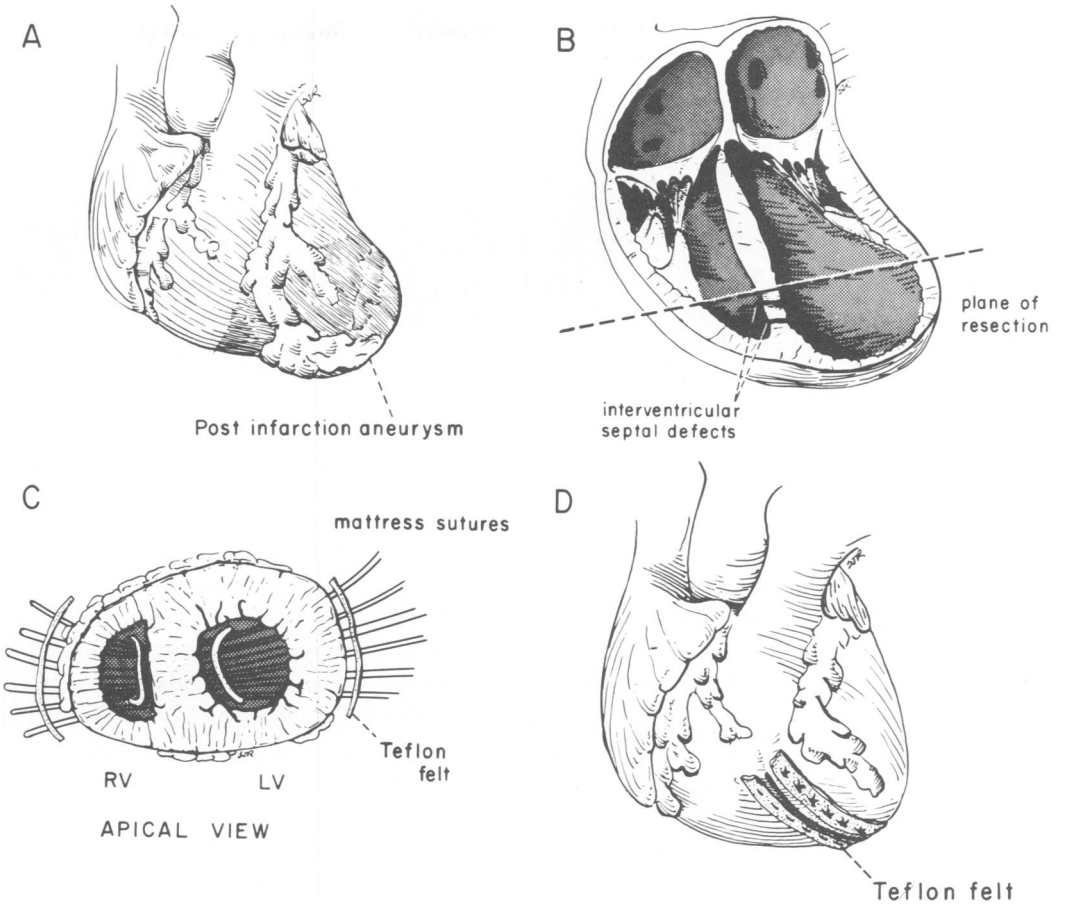

FIG. 3. Artist's representation of operation performed for apical ventricular septal defect complicating acute anterior myocardial infarction. Operation performed in patient 12 days following acute anterior infarction was that of apical amputation. This figure describes the operation which was reported in N. Engl. J. Med. 283:1507, 1970, and is reprinted with the permission of the N. Engl. J. Med. Anterior surface of the heart (A and B), showing the infarct and area of resection, and apical view of the heart (C and D), showing the approximation of right ventricular wall, septum and left ventricle with mattress sutures buttressed by teflon felt.

patients because in previous years all five patients with similar severe renal failure (BUN greater than 100 mg% or profound oliguria) had not survived after operation (Table 3).

Operative Procedures

Since this series includes all patients treated surgically at this hospital for ventricular septal defect after myocardial infarction, the operative techniques used were modified with time. Four of the first five patients in the series underwent patch closure of the septal defect through an anterior right ventriculotomy, as one might repair a congenital ventricular septal defect (Fig. 2). But in 1969 it became apparent that both exposure and postoperative ventricular function were better maintained if ventriculotomy were carried out through the area of infarction[6,16,21] in the anterior or apical aspect of the left ventricle (Fig. 3). Identification of the borders of the infarct is facilitated by placing suction on the left ventricular vent after the institution of cardiopulmonary bypass. This maneuver causes the flaccid musculature of the fresh infarct or the thinned-out scar of the old infarct to pucker, demarcating the limits of the incision.

After ventriculotomy, the extent of the septal defect is determined. If the septal defect is small and particularly if the infarct is anterior and apical, simple amputation of the infarct with exclusion of the ventricular septal defect may be an adequate operation.[6] Interrupted horizontal mattress sutures are placed sequentially through a teflon felt strip, the left ventricle, a second strip of felt, the septum, a third strip of felt, the right ventricle and a fourth teflon felt strip. Necrotic muscle in either ventricular wall or the septum is trimmed away as necessary to allow placement of the suture line at the edge of viable muscle. All sutures are placed before any are tied down. For anterior defects higher up along the septum, the repair was modified to include buttressing of the defect closure with sutures through the right ventricular free wall (Fig. 4). This method had the additional advantages of sparing viable muscle in the distal septum and of preserving left ventricular volume by transposing the interventricular septum to the right.[2,5,12] These techniques were our primary method of repair from 1970 until the middle of 1973. Of 15 operations performed in the interval, only two simple cloth patches were used for closure of large anterior septal defects in conjunction with infarctectomy.

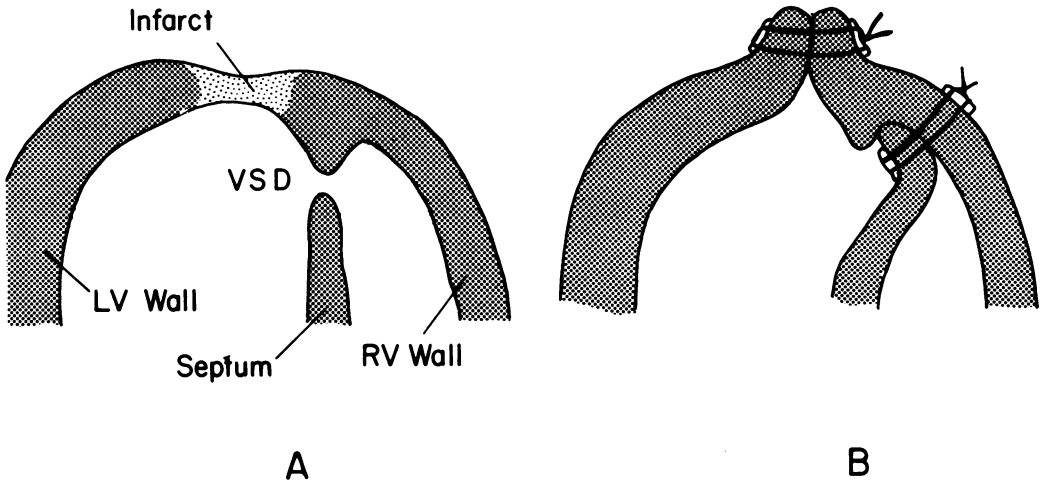

FIG. 4. Illustration of method of repair for ventricular septal defect (VSD) occurring higher up along the anterior aspect of ventricular septum after anterior myocardial infarction. This method has the advantages of sparing viable muscle in the distal septum and of preserving left ventricular volume.

In 1973 review of our results (Table 1) suggested need for restoration of the geometry of the extensively damaged heart. This consideration, and the principle of avoiding tension on friable suture lines, led to more frequent use of woven teflon or dacron to replace portions of resected muscle. In particular, a modification of the two-layer patch described by Iben et al.[13] has allowed great flexibility in restoration of ventricular geometry. Using horizontal mattress sutures with teflon felt pledgets, two layers of woven teflon cloth are sewn to the edge of the ventricular septal defect as shown in Figure 5. The patch may then be trimmed to allow partial replacement of either left or right ventricular musculature as is necessary. Patch closures have been used in five of the last 15 operations.

Recognition that the septal defect which complicates inferior or posterior myocardial infarction requires different techniques than those most applicable to the anterior or apical defect has led to better understanding of the principles underlying the successful repair of all such defects.[7] In contrast to the septal defect which complicates anterior myocardial infarction, the defect following inferior or inferoposterior myocardial infarction is posteriorly located and higher up along the septum. In our view, an incision in the anterior aspect of the left ventricle is as inappropriate for this type of defect as a right ventricular outflow tract incision was for the defect which complicates anterior myocardial infarction. After inferior myocardial infarction, the posteriorly located defect is best approached by dislocating the heart anteriorly out of the pericardium (Fig. 6), as one would do for construction of a posterior descending artery by-pass. The infarct may be located in the diaphragmatic surface of the right ventricle, directly over the posterior descending artery and underlying septum or involve more of the left ventricle, depending on the coronary anatomy and disease in a given patient. In addition to a posterior aproach to the posterior defect, the anatomic placement of the ventriculotomy is important.

From our experience a myocardial incision eccen-

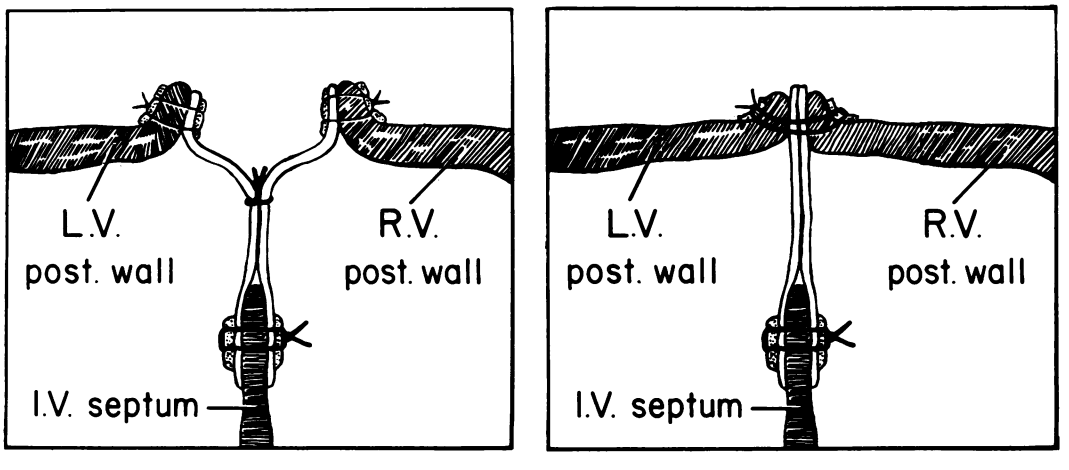

FIG. 5. Modifications of Iben's "double patch" repair, illustrated here for repair of *posterior* ventricular septal rupture; repair with patch (shown in right panel) was used in two patients (#25 and #36, Table 1), both hospital survivors (Case Report #2). Repair shown in left panel has not been used yet, but is presented as a logical hypothetical extension of right panel repair, for the patient with more extensive ventricular free wall muscle necrosis posteriorly. The increasing use of prosthetic material to replace necrotic muscle has improved results, particularly in patients with posterior defects. L.V. = Left Ventricle; R.V. = Right Ventricle. I.V. septum = Interventricular Septum.

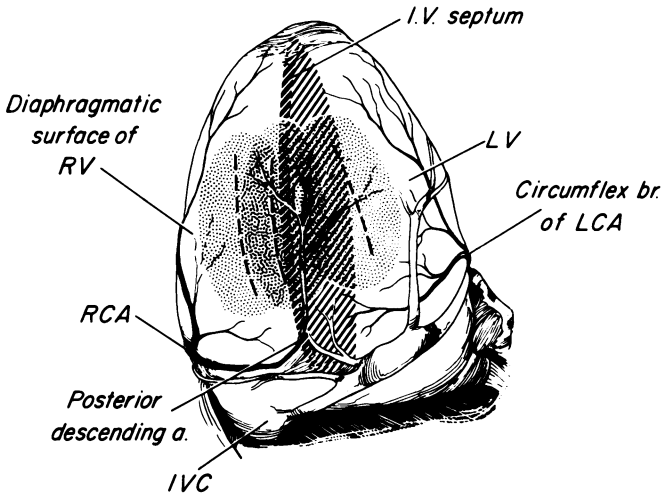

FIG. 6. Illustration of heart dislocated out of pericardial cavity, anteriorly and cephalad, for best exposure of posterior VSD, as one would do to perform posterior descending artery bypass. As the three stippled zones show, the infarct may be located in the diaphragmatic surface of the right ventricle (RV), directly over the posterior descending artery and underlying septum (striped area), or involve more of the posterior free wall of the left ventricle (LV). In addition to the posterior approach, as suggested by Shumacker[20], it is important to place ventriculotomy in center of infarct (dashed line for each zone of infarct) to void tearing out of sutures in friable myocardium (Case Reports #1, #2, and #3, also patients #28, #36, and #39 in Table 1). IVC = Inferior Vena Cava.

tric to the infarct almost invariably results in tearing out of sutures and either uncontrollable hemorrhage or recurrence of the VSD. Thus, it would seem that the concept which embodies a left-sided approach to the defect is less appropriate than the principle of operating directly through the infarct. Incision directly through the soft center of the infarct invariably leads one to the defect, and exposure can be improved by excision of surrounding necrotic ventricular muscle (Fig. 7a).

Associated procedures were carried out in 11 patients: mitral valve replacement in two and coronary artery vein bypass graft in nine. In six patients significantly narrowed or occluded coronary arteries to areas other than that of the primary infarct were not bypassed at the time of operation. The following cases illustrate our current management of ventricuilar septal defect after myocardial infarction.

Case Reports

Case 1. F. G. is a 66-year-old male (Patient #28 in Table 1) who was well until 3/21/74, when, after jogging, he had severe substernal chest pain. He was admitted to an outside hospital. His electrocardiogram demonstrated S-T segment elevation in leads 2, 3, AVF, V5 and V6. On his second hospital day, he was noted to have an apical holosystolic murmur consistent with mitral regurgitation. He developed congestive failure and was transferred to the Massachusetts General Hospital with continuing evidence of papillary muscle dysfunction.

On admission, because of relative hypotension (systolic blood pressure 90 mm Hg), pre-renal azotemia (BUN 40) and clinical evidence of pulmonary edema, an intra-aortic balloon pump was inserted. Swan-Ganz catheterization revealed large V waves in the pulmonary capillary wedge pressure tracing and a significant oxygen step-up between the superior vena cava and the pulmonary artery. Mean pulmonary capillary wedge pressure was 20 to 25 mm Hg, rising to 26 to 30 mm Hg when balloon augmentation was temporarily halted. Coronary angiography, performed on the day after admission, revealed complete occlusion of the right coronary artery and stenosis of a short left anterior descending coronary artery. The circumflex artery was small with scattered non-occlusive disease. The left ventriculogram showed excellent anterolateral contraction, but akinetic inferior wall, early opacification of the right ventricle and pulmonary artery and moderate mitral regurgitation.

On the third hopsital day, the patient remained hemodynamically unstable and showed balloon pump dependence. When balloon agumentation was temporarily stopped, systolic blood pressure fell from 110 to 85 mm Hg, pulmonary capillary wedge pressure rose from 22 to 27 mm Hg, and central venous oxygen saturation fell from 42 to 37%. The patient was then taken to the operating room on April 1, 1974. A large posterior left ventricular infarct was present with minimal extension into the right ventricle. An incision through the posterior left ventricular infarct revealed a posterior ventricular septal defect and a ruptured posterior papillary muscle. The septal defect was closed by mattress sutures buttressed by teflon felt (Fig. 7a). The large defect in the left ventricle after infarctectomy could not be closed primarily, and this defect was replaced with a four by six centimeter oval patch of tightly woven teflon (Figs. 7b and c). The mitral valve was then replaced via the left atrium with a Bjork-Shiley disc prosthesis. Subsequently, the patient was taken off bypass with minimal pressor and continued balloon pump support.

His postoperative course was complicated by pneumonia, requiring long-term ventilatory support via tracheostomy. A gastrotomy for alimentation was performed at the time of tracheostomy. A bacteremia, presumably from a superficial abscess in his gastrotomy incision, required four weeks of parenteral antibiotics. Seven weeks after operation, right and left heart catheterization was per-

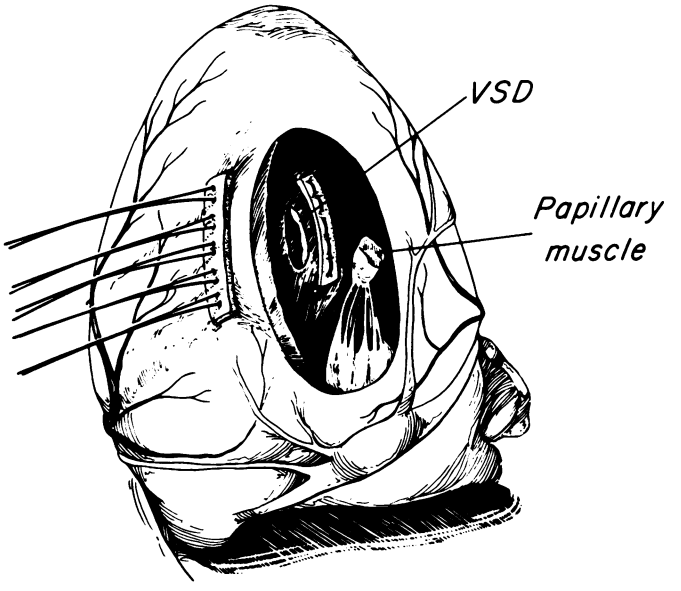

FIG. 7a. Illustration of pathology, as seen intra-operatively, in patient F.G. (Case Report #1 and patient #28 in Table 1) with ventricular septal defect (VSD), closed primarily with teflon felt buttressing; infarctectomy of posterior left ventricular free wall and ruptured posterior papillary muscle are also shown.

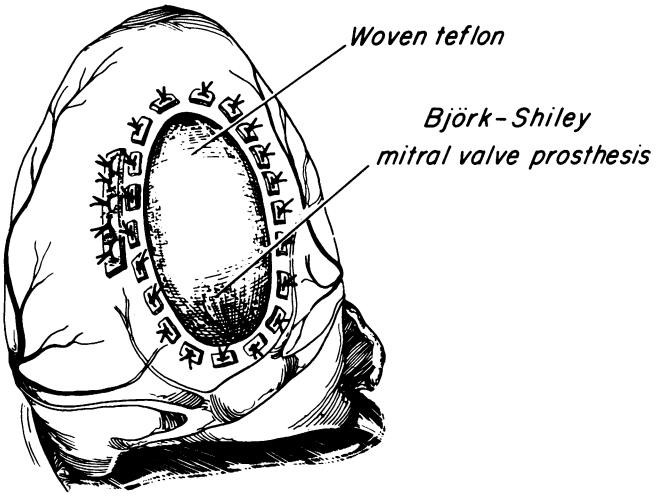

FIG. 7b. Illustration of replacement of posterior left ventricular free wall with woven teflon graft fabric after infarctectomy and VSD closure in patient F.G. (Case Report #1 and patient #28 in Table 1). Mitral regurgitation due to papillary muscle rupture corrected by mitral valve replacement with Bjork-Shiley prosthesis. Patient is living and well (NYHA Functional Class II) three years after operation.

formed, which revealed good left ventricular function and a very small persistent ventricular septal defect. The patient was discharged on May 29, 1974 on digoxin, Coumadin and Aldactazide. He is currently well, three years after operation, and able to engage in all but the most strenuous activities.

Case 2. W. F. is a 66-year-old male (Patient #36 in Table 1) who began coughing persistently on 3/12/75. On 3/15/75 he had a single brief episode of chest pain. He was admitted to another hospital, and after eight days of persistent pulmonary edema and intermittent atrial fibrillation, he was transferred to our hospital on 3/25/75. On admission his electrocardiogram revealed marked sinus tachycardia and small Q waves in leads 2, 3, AVF, V5 and V6. He was judged clinically to be in pulmonary edema with a grade 4/6 holosystolic murmur at the apex. After two days treatment of refractory pulmonary edema, oliguria and a systolic blood pressure of 90 mm Hg, an intra-aortic balloon pump was inserted. His urine output improved, his blood pressure on the baloon pump was 110 mm Hg, and his pulmonary edema improved sufficiently to allow him to sleep flat in bed.

Cardiac catheterization was performed three days after admission and revealed occlusion of the right coronary artery, a 40% stenosis of the left anterior descending artery, a pulmonary capillary wedge pressure of 27 mm Hg, and a ventricular septal defect with a 4:1 left-to-right shunt. Operation was planned for 3/31/75, but clinical deterioration forced emergency operation on 3/30/75. At operation, the infarct lay directly over the posterior desending vessels and underlying posterior septum. A transinfarct incision directly over the septum exposed the posterior septal defect. Necrotic septal muscle was debrided and two woven dacron patches were sewn to either side of the septum. The free edge of right ventricle, the patches and free edge of left ventricle were closed as one suture line with felt buttressing (Fig. 4 right panel).

The patient's convalescence was remarkably event-free. The balloon pump was weaned and removed three days after operation. The patient was discharged from the hospital 16 days after operation. Although retired, he remains well and active two years after operation.

Case 3. M. S. is a 66-year-old nun (Patient #39 in Table 1) who presented to an outside hospital in Septemberof 1976 after a 20 minute episode of chest pain following two weeks of increasing angina. She had no past history of cardiac disease. She was found to have a recent inferior myocardial infarction by electrocardiogram and a III/VI systolic murmur. Because of congestive heart failure unresponsive to medical therapy, she underwent cardiac catheterization and angiography, which revealed occlusion of the distal right coronary artery, high-grade stenosis of the anterior descending and a large diagonal branch, 2.3:1 left-to-right shunt via a posterior septal defect, a small posterior left ventricular aneurysm and biventricular failure. She was transferred to our institution six weeks after the onset of chest pain, still very limited by shortness of breath. A few days after admission, she was taken to the operating room.

At operation, saphenous vein bypass grafts were constructed to both the anterior descending artery and its diagonal branch. The scarred area of the infarct was easily seen posteriorly, involving principally the diaphragmatic surface of the right ventricle with little involvement of the posterior left ventricle. A transinfarct incision was made in the diaphragmatic surface of the right ventricle and a two centimeter septal defect was thereby exposed. The edges of the defect were scarred. The defect was closed with a woven dacron patch buttressed with teflon felt on the left and right ventricular side of the septum. The right ventriculotomy was closed primarily with felt-buttressed sutures. The patient was easily weaned from cardiopulmonary bypass without inotropic support.

Soon after arrival in the intensive care unit, an episode of atrial flutter led to mild hypotension and oliguria.She required pressors after this episode and an intra-aortic balloon pump was inserted. Her hemodynamic situation quickly stabilized, and the balloon was removed on the second postoperative day. Her recovery was thereafter slow but uneventful, and she was discharged five weeks after operation on digoxin, diuretics and Coumadin. She has since resumed her work at the convent, and is NYHA functional class II, five months after operation.

Case 4. (J. S.) Six months prior to admission, this 62-year-old female (Patient #43 in Table 1) had an inferior myocardial infarction complicated by transient complete heart block. On January 29, 1977, she presented to an outside hospital with severe chest pain. An episode of ventricular fibrillation required cardioversion in the emergency room, with restoration of normal sinus rhythm. Her electrocardiogram revealed Q waves and new S-T segment elevation in leads 2, 3 and AVF, as well as S-T segment elevation in leads V1–V5. Five days after admission, she developed a loud systolic murmur, severe chest pain and profound hypotension. She was transferred

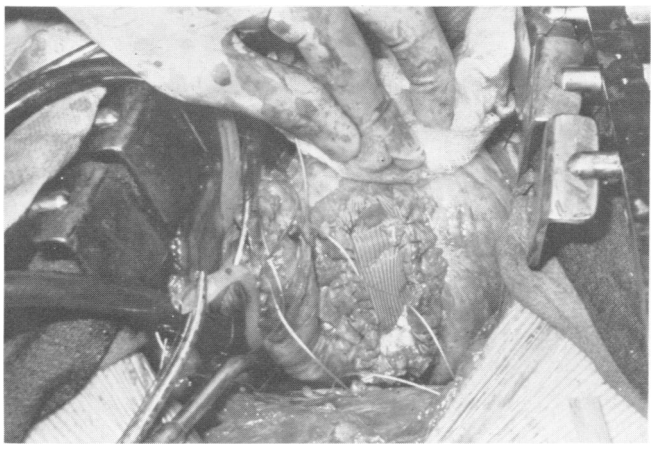

FIG. 7c. Intra-operative photograph of posterior left ventricular free wall replacement with woven teflon graft fabric after infarctectomy as illustrated in Figure 7b. For orientation, patient's feet are below and head above.

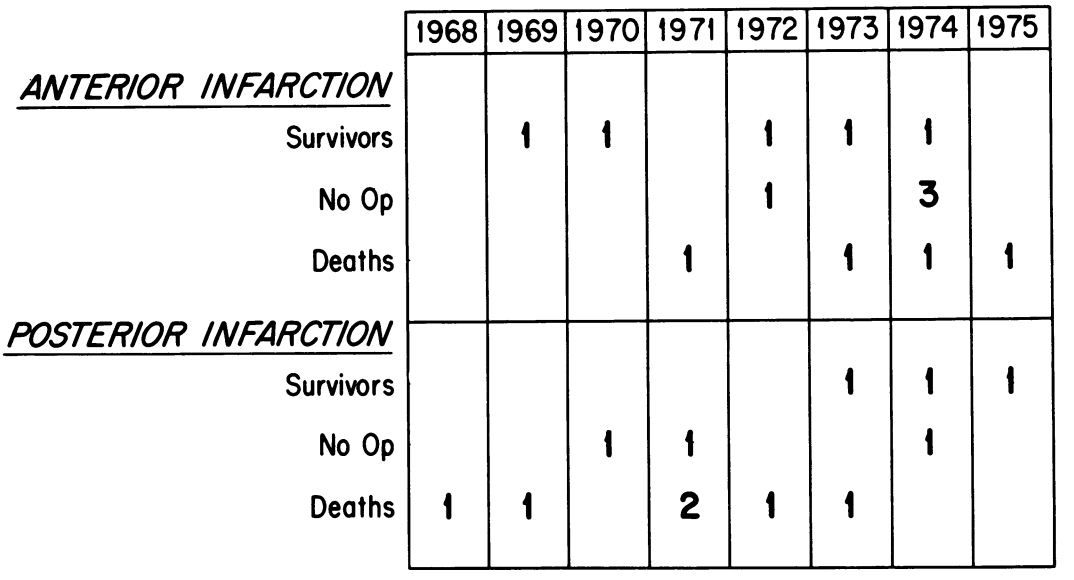

FIG. 8. Group I patients who were treated less than 21 days after myocardial infarct by year. Initial successes were in patients with anterior defects after anterior myocardial infarction. More recently, results in patients with posterior VSD after inferior or posterior myocardial infarction, have improved with transinfarct ventriculotomy and more liberal use of prosthetic material to replace necrotic muscle. (See text for discussion.)

to our hospital with a systolic pressure of 80 on norepinephrine and dopamine. Soon after arrival, her blood pressure was unobtainable by palpation, and an intra-aortic balloon pump was inserted. A 5:1 left-to-right shunt was documented by Swan-Ganz catheter prior to balloon pump insertion. With intra-aortic balloon pumping the pressors were discontinued. The patient's blood pressure was 80/50 without the balloon pump and 150/75 with the balloon pump. The following day she underwent cardiac catheterization, which revealed total occlusion of the right coronary artery and its posterior descending branch, 90% occlusion of the left anterior descending artery proximally, but a widely patent distal anterior descending artery, and a 40% narrowing of the circumflex artery. Left ventricular angiogram revealed a large apical septal defect and hypokinesis of the inferior and diaphragmatic segments of the left ventricle. Two days after admission, she was taken to the operting room.

At operation, a large apical infarct was easily identified. A coronary artery bypass graft was constructed to the distal left anterior descending artery. An infarctectomy of the apex was performed. The septal defect was closed by felt buttressed sutures passed through the septum and out the right ventricular wall. The edges of the left ventricle after infarctectomy were then closed by similarly reinforced sutures passed through the left ventricular free wall, through the septum and through the right ventricle. The patient was weaned from cardiopulmonary bypass with the aid of epinephrine and balloon pumping. She was rapidly weaned from pressors in the intensive care unit and the balloon was removed on the second post-operative day. Her subsequent postoperative course was uneventful, and she was discharged on the seventeenth postoperative day on digoxin and Coumadin. She is currently well and has resumed doing her housework, six weeks after operation.

Results of Operation

Operative Mortality

Operation was carried out in 36 of 43 patients. Twenty-four of these patients survived the initial hospitalization (i.e., 66% of the operated patients or 56% of the total series). In patients with anterior infarction in whom operation could be deferred longer than 21 days, seven of eight patients were operative survivors. Similar results were obtained in patients with posterior infarction: seven patients survived of the seven in whom operation for a posterior ventricular septal defect was deferred greater than 21 days. Operative mortality for patients undergoing operation greater than 21 days after infarction was only 7%.

If operation was required prior to 21 days, the mortality was substantially higher (Fig. 8). Seven of eleven such patients with anterior infarction survived and only three of ten patients with posterior infarction survived. Operative mortality, then, in this subgroup was 52%.

In patients who had associated procedures, the two patients who required mitral valve replacement were both hospital survivors (Table 4); of the nine patients in whom saphenous vein aortocoronary bypasses were done, six were hospital survivors. Of the six patients who had a significant coronary occlusion in an area other than that of the primary infarct, that was not bypassed, four were hospital survivors.

TABLE 4. *Associated Procedures and Lesions*

	No.	Hospital Survival	Late Survivors
MVR	2	2	1
CABG	9	6	6
Occluded vessel not bypassed, other than the primary one to infarct	6	4	3

Of the patients who had associated procedures, in addition to VSD closure, and were hospital survivors, only one died late, of lymphoma, a year after operation. This same patient also was the only late death among patients who had an occluded vessel, other than the primary one to the infarct, that was not bypassed.

MVR = Mitral Valve Replacement.

CABG = Coronary Artery Bypass Graft.

Overall operative mortality in this series is 33% (12 of 36 patients). But current operative techniques were instituted in 1973 with the more liberal use of woven teflon to replace resected muscle. Operative mortality prior to mid 1973 was 47% (nine deaths in 19 patients). Mortality after mid 1973 was only 18% (three deaths in 17 patients). Even in those patients in whom operation was necessary prior to 21 days after infarction, operative mortality in the last 44 months has been only 30% (three deaths in ten patients).

Postoperative Ventricular Septal Defect

Six patients were thought to have a postoperative ventricular septal defect. Patient #2, a long-term survivor, had a postoperative parasystolic apical murmur consistent with ventricular septal defect after a posterior septal defect was closed with difficulty through an anterior right ventriculotomy. Patient #7 underwent infarctectomy with suture closure and exclusion of an anterior septal defect. A murmur was noted in the early postoperative period, but recovery was uneventful, with good cardiac function for three years. In 1973 the patient developed progressive heart failure and a 2.5:1 shunt was documented at cardiac catheterization in June of 1974. The patient died in November of 1974. Patient #22 had attempted direct suture exclusion of a posterior ventricular septal defect. A persistent septal defect was noted at operation and a 1.8:1 shunt was demonstrated by Swan-Ganz catheterization postoperatively. The patient died 17 days after operation. Patient #27 was noted to have a pansystolic murmur after direct suture exclusion of an anterior septal defect. He died five days after operation of low cardiac output. Patient #28 was found to have a small left-to-right shunt as an incidental finding at postoperative catheterization. Patient #41 died of catastrophic rerupture of the ventricular septum seven weeks after operation.

Long-term Results

There were five late deaths in this series, and no patients have been lost to follow-up. Mean duration of survival of these five patients was 26 months (range: 1–42 months). Patient #6 died suddenly, having been in good health for four years after operation. Patient #7 died after three years of progressive cardiac failure as described above. Patient #10 died one year postoperatively while undergoing treatment for disseminated lymphoblastic lymphoma. Patient #25 died three years after operation of intracerebral hemorrhage, having been in good health since her operation. Patient #40 died suddenly ten days after hospital discharge.

Of the 19 other long-term survivors, eight are NYHA functional class I, ten are NYHA functional class II, and one is NYHA functional class III. Mean length of follow-up of currently surviving patients is 41 months (range: 1–96 months).

Discussion

This series of patients reflects the evolution of our therapeutic approach to septal perforation after myocardial infarction. Early patients were treated as we treated congenital ventricular septal defects, with attempted patch closure through a standard anterior right ventriculotomy. The two patients thus treated with acute infarction died, and one of the two patients with a chronic septal defect had a persistent murmur postoperatively.

Infarctectomy with felt buttressed suture closure of the ventricle and exclusion of the septal defect led to some success, but, in addition to this basic change in technique, several other alterations allowed better management of these patients: 1) Ventriculotomy was positioned in the center of the infarcted area. 2) The intra-aortic balloon pump was used to support the patient hemodynamically while coronary angiography and left ventriculography defined the pathology and to support the patient in the perioperative period. 3) Associated coronary or mitral valve deficiencies were corrected if necessary.

Ventriculotomy through the right ventricular outflow tract is clearly inappropriate for ventricular septal rupture after infarction. This incision gives poor exposure to the defect, it injures normal muscle, it interrupts potential collateral channels and it does not allow correction of often-present paradoxical pulsation of the infarcted myocardium. Iben et al. altered the usual ventriculotomy by placing the incision in the right ventricle over the site of greatest thrill, allowing better exposure of the defect.[13] Kay and colleagues[16] and Javid et al.[14] advocated a left-sided approach to post-infarction septal defects, with the incision through an area of post-infarction scar or thinning. Shumacker[20] pointed out that an anterior left ventriculotomy is entirely inappropriate for a posterior septal defect and suggested that incision through the posterior right ventricle affords excellent exposure of the defect. We have felt for some time[7] that ventriculotomy is best performed through the center of the infarcted area, whether that site be anterior or posterior, right ventricular, left ventricular or directly over the septum (Figs. 2 and 6). An incision eccentric to a fresh infarct will sacrifice functioning muscle on the non-infarcted side of the incision to allow adequate margin for suturing and may lead to sutures tearing out of friable muscle on the infarcted side of the incision.

The intra-aortic balloon pump (IABP) has been an

important part of our management of myocardial ischemia with cardiac failure.[17] Patient #30 in this series (Tables 1 and 2) died as a result of balloon insertion, but such fatal complications are rare (six of over 750 balloon insertions), and the use of the balloon pump greatly facilitates coronary angiography and operation. Moreover, by virtue of systolic unloading of the left ventricle, use of the balloon pump invariably leads to a decrease in the size of the left-to-right shunt in this particular complication of infarction.[9] Use of IABP, however, in a manner which delays needed urgent operation, once a patient has demonstrated deteriorating hemodynamics, is *not* recommended.

Associated coronary and mitral valve deficiencies should always be identified prior to operation. We do not usually perform a bypass graft to the artery supplying the area of infarction, since the mass of viable muscle supplied by this artery is too small to justify this procedure, and closure of the ventricular septal defect often precludes a vein bypass to this area on technical grounds. Crosby et al.[4] and Hill et al.[12] suggest that significant obstructions in other arteries should always be bypassed. Conversely, Kaplan and co-workers do not.[15] Bypass grafting was carried out in nine of 15 patients with potentially bypassable lesions in this series. Each patient presents a different situation with regard to muscle mass that might be revascularized, and we have certainly not felt compelled to perform bypass grafts in every instance. Significant mitral regurgitation has required valve replacement in two patients, both of whom survived hospitalization; one died of lymphoma a year after operation.

Through mid 1973, our results with septal defects requiring operation after three weeks were excellent, and results with anterior septal defects in fresh (<3 weeks old) infarction were also good (Fig. 8). But our success with septal defect after fresh *inferior* or posterior infarction was not, with six deaths in six attempts. Patients #20 and #22 (Tables 1 and 3) both died because sutures tore out of the attempted repair. This experience suggested that inferior or posterior infarction with septal rupture was not always amenable to the primary suture technique of repair (although Crosby et al.[4] report success with the technique in two of three patients). The subsequent three patients with acute inferior infarction were treated with patch closure of the septal defect or prosthetic replacement of the ventricular free wall. All three patients survived with excellent hemodynamic results (Fig. 8). A modification of the Iben double-patch technique is proposed to allow flexibility in extension of prosthetic replacement to either the right or left ventricular free wall and to prevent undue tension at any part of the suture line (Fig. 5).

In a recent patient (Patient #41 in Table 1), an acute inferior ventricular septal defect was repaired without a patch. Seven weeks postoperatively, the patient died when the septum reruptured, reopening the septal defect. This experience reinforces our opinion that the acute posterior septal defect requires patch closure of the defect and/or of the ventricular free wall.

In this series, seven patients were not offered operation. Of the five patients from whom operation was withheld for cardiac reasons, four were in profound cardiogenic shock unresponsive to pressors or intra-aortic balloon pumping. The fifth patient (#12) had severe diffuse ventricular hypokinesis and vessels unsuitable for grafting in addition to acute infarction with a posterior septal defect. Operation has been withheld more frequently in recent years (Fig. 8), but this circumstance seems to be related to the recent availability of mobile support units which allow inter-hospital transfer of patients, some of whom are in advanced cardiogenic shock.

Hospital survival after operation is 66%, a result similar to that of Hill et al.,[12] and the 18% operative mortality in the last three and a half years is indeed encouraging. The quality of life obtained after operative salvage has been gratifying in this series. Eighteen of the 19 surviving patients are either NYHA functional class I or II.

Our current management of this uncommon catastrophe includes: 1) deferring operation, if possible, until three weeks after infarction, 2) cardiac support with intra-aortic balloon pump insertion to allow preoperative definition of coronary and ventricular anatomy of patients with hemodynamic deterioration, and 3) a transinfarct incision with prosthetic replacement of excised ventricular free wall or septum, if necessary, and 4) possible repair of associated coronary or mitral valve pathology. With aggressive surgical intervention, we believe that considerable salvage is possible in patients with ventricular septal rupture after myocardial infarction.

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Discussion

Dr. Harris B. Shumacker, Jr. (Indianapolis, Indiana): This presentation is extraordinary in several aspects. Firstly, it comprises a very large number of cases. On most services, even when hundreds of patients are treated operatively for coronary artery disease each year, the number referred for post-infarction ventricular defects is comparatively small. When I reviewed the literature five years ago, I was able to find only 91 patients who had been operated upon for this condition with the aid of cardiopulmonary bypass. Dr. Daggett's group has been able to present a series from one institution more than a third that large.

The report is especially valuable since a substantial number of these patients have been treated recently, when we have had available assist devices for support such as the intra-aortic balloon and, more importantly, technical methods of repair which permit firm and lasting closure of the defect early or late after infarction, a situation which did not exist a few years ago.

The operative suggestions which have been made by them and a number of others of us have converted this problem to the lesser of the two important ones. The one of far greater significance, and the one which I believe is the chief determinant in the outcome of treatment, concerns the extent and degree of stabilization of the infarction. This is the reason that the earlier the operation must be done, the greater the risk; and the longer it can be deferred, the smaller.

Delay in treatment is advisable if the patient can be maintained in a reasonably satisfactory state, in order to permit demarcation of the infarct and to allow the cardiac damage to have reached as nearly a stable state as possible.

Unquestionably, in suitable cases coronary artery bypass grafting should accompany closure of the defect and remodeling of the cardiac chambers.

I find myself in substantial agreement with all Dr. Daggett and his associates have concluded, and I am quite certain that their large and excellent experience will help others to better treat this challenging complication of myocardial infarction.

Dr. Frank Gerbode (San Francisco, California): There are a number of points which they made which are now recognized generally as being important in the surgical treatment of infarction ventricular septal defect: access to the defect through the infarct, patch repair (at times on the right as well as the left side of the defect) and right lateral suture of an anterior septal defect.

Of special interest is their use of a cloth gusset to enlarge the left ventricle. It's remarkable to see how well this was tolerated, and how the patients who had it did in the long-term evaluation.

The improvement in risk of operation after three weeks results from natural patient selection as well as better tissue for repair. When it is not possible to delay operation, the operative mortality is much higher. In our hands in early emergency it is about the same as theirs; greater than 50%. The over-all mortality rate (early and late) generally for this defect is around 30%.

Balloon pump support is an important adjunct. Difficulty in obtaining a permanent complete repair is shown in their finding of six patients with small to large residual defects.

It would appear that bypass procedures did not improve the operative mortality rate. I would like the authors to comment on whether the long-term results indicated improvement over those who were not bypassed.

Dr. Frank Cole Spencer (New York, New York): I had not planned on discussing the paper, but the data are so significant I thought certain observations might be of interest.

Clearly, Dr. Daggett and his associates have pioneered the surgical treatment of ventricular septal defect following myocardial infarction. What you have just heard is probably the most substantial contribution that exists in the literature in the entire world today.