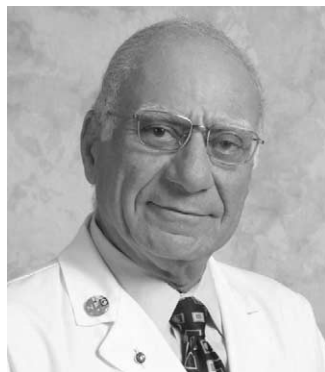


Factors affecting late survival after surgical remodeling of left ventricular aneurysms

Hooshang Bolooki, MD,^a Eduardo DeMarchena, MD,^b Stephen M. Mallon, MD,^b Kushagra Katariya, MD,^a Michael Barron, MD,^c H. Michael Bolooki, BS,^a Richard J. Thurer, MD,^a Stana Novak,^a and Robert C. Duncan, PhD^d



Dr Bolooki

See related editorial on page 323.

From the Divisions of Thoracic and Cardiovascular Surgery^a and Cardiology^b and Departments of Anesthesiology,^c Epidemiology, and Public Health,^d University of Miami School of Medicine/Jackson Memorial Hospital, Miami, Fla.

Supported in part by the Thomas Curtis Research Grant.

Read at the Twenty-eighth Annual Meeting of The Western Thoracic Surgical Association, Big Sky, Mont, June 19-22, 2002.

Received for publication July 15, 2002; revisions requested Sept 3, 2002; revisions received Oct 11, 2002; accepted for publication Oct 28, 2002.

Address for reprints: Hooshang Bolooki, MD, FRCS (C), University of Miami/Jackson Memorial Hospital, PO Box 016960 (R-114), Miami, FL 33101 (E-mail: hblooki@med.miami.edu).

J Thorac Cardiovasc Surg 2003;126:374-85

Copyright © 2003 by The American Association for Thoracic Surgery

0022-5223/2003 \$30.00 + 0

doi:10.1016/S0022-5223(03)00023-0

Objectives: Surgical remodeling of the left ventricle has involved various techniques of volume reduction. This study evaluates factors that influence long-term survival results with 3 operative methods.

Methods: From 1979 to 2000, 157 patients (134 men, mean age 61 years) underwent operations for class III or IV congestive heart failure, angina, ventricular tachyarrhythmia, and sudden death after anteroseptal myocardial infarction. The preoperative ejection fraction was $28\% \pm 0.9\%$ (mean \pm standard error), and the pulmonary artery occlusive pressure was 15 ± 0.07 mm Hg. Cardiogenic shock was present in 26 patients (16%), and an intra-aortic balloon pump was used in 48 patients (30%). The type of procedure depended on the extent of endocardial disease and was aimed at maintaining the ellipsoid shape of the left ventricle cavity. In group I patients ($n = 65$), radical aneurysm resection and linear closure were performed. In group II patients ($n = 70$), septal dyskinesia was reinforced with a patch (septoplasty). In group III patients ($n = 22$), ventriculotomy closure was performed with an intracavitary oval patch.

Results: Hospital mortality was 16% (25/157) and was similar among the groups. Actuarial survival up to 18 years was better with a preoperative ejection fraction of 26% or greater ($P = .004$) and a pulmonary artery occlusive pressure of 17 mm Hg or less ($P = .05$). Survival was worse in patients who had intra-aortic balloon pump support ($P = .03$). Five-year survival for all patients in group III was higher than for patients in group II (67% vs 47%, $P = .04$).

Conclusions: Factors that improved long-term survival after left ventricular surgical remodeling were intraventricular patch repair, preoperative ejection fraction of 26% or greater, and pulmonary artery occlusive pressure of 17 mm Hg or less without the need for balloon pump assist.

Surgical methods to restore the volume and shape of the left ventricle (LV), after an extensive myocardial infarction (MI) that has resulted in cavity remodeling (LV aneurysm), have evolved over the years. Present techniques attempt to reconstruct the natural ellipsoid shape of the LV cavity, which offers the most favorable geometry for LV performance and patient survival.¹⁻³ The long-term surgical results have been reported as an observational experience by various centers, most recently by the RESTORE group.^{4,5} This study evaluates the factors affecting the long-term survival of patients who underwent 3 methods of LV cavity restoration.

Patients and Methods

Patient Selection

A retrospective review was performed of 157 consecutive patients who underwent LV volume reduction surgery by 1 of 3 surgical techniques from 1979 to 2000. All procedures were performed by the senior author (H.B.). Table 1 summarizes the characteristics of all patients. The age range of the patients was from 30 to 85 years (mean 61 ± 0.08 years SEM). There were 134 male patients (85%). All patients had had an anteroseptal MI in the past, and

TABLE 1. Preoperative patient characteristics

Variable	All patients (n = 157)	Group I (n = 65)	Group II (n = 70)	Group III (n = 22)	P value
Age, y ± SEM (range)	61 ± 0.8 (30–85)	62 ± 1.25 (39–85)	60 ± 1.1 (30–78)	62.5 ± 2.2 (40–84)	.731
Male/female	134/23	57/8	58/12	19/3	.722
CHF, n (%)	117 (74)	40 (61)	59 (88)	15 (68)	.032*†
Class III and IV	84 (53)	27 (41)	42 (60)	15 (68)	.04*†
Angina, n (%)	103 (65)	43 (66)	44 (62)	16 (72)	.691
VT, n (%)	82 (51)	16 (25)	56 (80)	10 (45)	.001*
SD, n (%)	60 (38)	17 (26)	38 (54)	5 (22)	.001*‡
CS, n (%)	26 (16)	8 (12)	17 (24)	1 (4.5)	.03*‡
Recent MI, n (%)	66 (42)	27 (41)	25 (35)	14 (63)	.068

CHF, Congestive heart failure; VT, ventricular tachyarrhythmia; SD, sudden death; CS, cardiogenic shock; MI, myocardial infarction (within 30 d of admission).

*Comparing groups I and II.
 †Comparing groups I and III.
 ‡Comparing groups II and III.

TABLE 2. Preoperative hemodynamic findings

Variable n	All patients 157	Group I 65	Group II 70	Group III 22	P value
EF % ± SEM (n = 131) (range)	28.8 ± 0.9 (7–56)	32 ± 1.2 (15–49)	27.8 ± 1.3 (7–56)	22.7 ± 2.7 (10–40)	.002*†
CI (L · min ⁻¹ · m ⁻²) n = 121 (range)	2.5 ± 0.05 (1.3–4.4)	2.5 ± 0.07 (1.5–3.9)	2.5 ± 0.08 (1.3–4.4)	1.99 ± 0.15 (1.3–2.4)	.04†‡
PAO (mm Hg) n = 127 (range)	15.7 ± 0.75 (3–40)	14.3 ± 1.1 (3–38)	16.4 ± 1.0 (4–40)	20.1 ± 3.1 (7–33)	.141
EDP (mm Hg) n = 53 (range)	20 ± 1 (7–38)	17.4 ± 1.5 (11–30)	20.2 ± 1.3 (7–30)	23 ± 2.7 (11–38)	.131

EF, Ejection fraction; CI, cardiac index; PAO, pulmonary artery occlusive (wedge) pressure; EDP, left ventricular end-diastolic pressure; SEM, standard error of mean.

*Comparing groups I and II.
 †Comparing groups I and III.
 ‡Comparing groups II and III.

TABLE 3. Operative procedures: Early and late mortality and follow-up

Variable	All patients (n = 157)	Group I (n = 65)	Group II (n = 70)	Group III (n = 22)	P value
CABG	126 (80)	55 (85)	56 (80)	15 (68)	.398
LITA graft	43 (34)	18 (32)	11 (19)	14 (93)	.002*†
Endarterectomy	12 (10)	6 (11)	4 (17)	2 (13)	.716
Grafts/patients (mean ± SE)	2.96 ± .11	3.03 ± .17	2.87 ± .17	3.0 ± .42	.493
Perioperative IABP	48(30)	14 (21)	26 (37)	8 (36)	.133
Elective/emergency	13/35	3/11	10/16	0/8	.089
Mapping/cryo	81 (51)	16 (25)	56 (80)	9 (41)	.001*
AICD	16 (10)	3 (5)	11 (15)	2 (9)	.101
Emergency operation	74 (47)	26 (40)	46 (65)	2 (9)	.003*†‡
CPB, min (mean ± SE) (range)	173 ± 6.2 (50–442)	159 ± 10 (52–442)	186 ± 8 (50–324)	168 ± 18 (67–267)	.113
ACC, min (mean ± SE) (range)	101 ± 3.8 (20–251)	96 ± 6.3 (20–251)	106 ± 5.2 (24–198)	102 ± 12.5 (29–174)	.503
Other procedures (valves)	5 (3%)	3	0	1/1(ASD)	.127
Mortality (hospital/30 d)	25 (16)	10 (15)	12 (17)	3 (13)	.460
Follow-up, mo mean ± SE	61.9 ± 5.3	67.9 ± 9.6	63.8 ± 7.6	37.9 ± 6.6	.185
Mortality (follow-up), n	76	37	38	1	
Lost to follow-up	20 (14.7)	9 (16.3)	9 (14.5)	2 (9)	
Presently surviving (3/2002)	36	9	11	16	
>70 y old	14	5	4	5	

CABG, Coronary artery bypass graft; LITA, left internal thoracic artery; IABP, intra-aortic balloon pump; Cryo, cryoablation; AICD, automatic implantable cardioverter-defibrillator; CPB, cardiopulmonary bypass; ACC, aortic crossclamp; ASD, atrial septal defect.

*Comparing groups I and II.
 †Comparing groups I and III.
 ‡Comparing groups II and III.

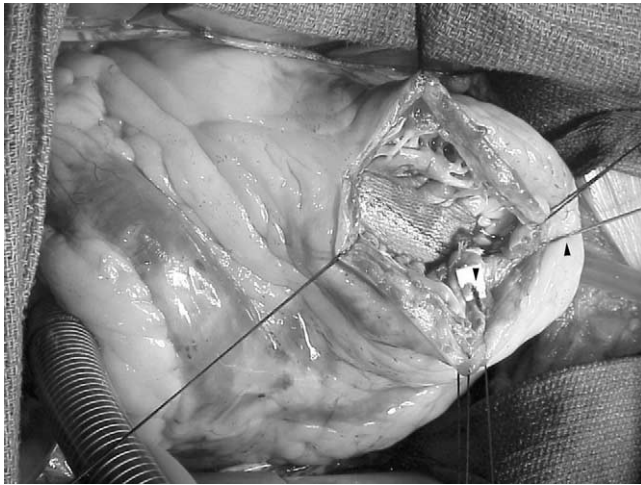


Figure 1. Surgeon's view of the anterior wall of the heart after LV volume restoration operation with an oval endoventricular Dacron patch. The black arrowheads point to the sutures that secure the patch and the purse-string suture.

66 patients (42%) had had an MI within the month before admission. All patients had a large anterior wall LV dyskinetic aneurysm. The indications for operation were congestive heart failure, cardiogenic shock (systolic blood pressure < 80 mm Hg and cardiac index < $1.8 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$), angina pectoris, ventricular tachyarrhythmia (VT), and sudden death. The preoperative data on LV systolic function and pulmonary artery occlusive (PAO) pressure are shown in Table 2. Patients with isolated lateral or posterior wall aneurysm or with LV dysfunction and predominant mitral valve insufficiency were not included in this survey.

Complete myocardial revascularization was performed in 126 patients (80%) by using saphenous vein segments and the left internal thoracic artery (Table 3). The left anterior descending artery was bypassed whenever possible. If the left anterior descending artery or any other major coronary artery had an extensively narrowed lumen, endarterectomy was performed to establish proximal blood flow, especially to the septal perforators. In 3 patients, the right internal thoracic artery was used as a free graft to bypass the left coronary artery branches. Sequential coronary grafts were performed whenever possible.⁶

The operations were performed on an emergency or urgent basis (within 12 to 24 hours of admission) in 74 (47%) of the patients. Additional procedures included mitral valve repair or replacement in 3 patients, aortic valve replacement in 1 patient, and atrial septal defect closure in 1 patient. An automatic implantable cardioverter-defibrillator was implanted in 16 patients. In our early experience, intrapericardial automatic implantable cardioverter-defibrillator patches were placed in 44 patients during surgery.

Surgical Techniques

The details of techniques used in our center have been published.^{7,8} In brief, normothermic cardiopulmonary bypass was established, and the heart was maintained beating, empty, and in sinus rhythm. The blood pressure was kept at 70 to 80 mm Hg.

Left ventriculotomy was made parallel and lateral to the course of the left anterior descending artery. Endocardial mapping was performed in all patients with VT, and cryoablation or subendocardial scar resection was performed according to our VT protocol.⁹ Thereafter, under mild hypothermia (33°C - 35°C), the aorta was crossclamped and antegrade cold-blood (4°C) cardioplegic arrest was induced.

Aneurysm resection and LV volume restoration were performed using 3 methods. The type of operation was selected depending on the extent of endocardial disease. The goal was to restore an elliptical LV cavity and a cone-shaped apex.

In patients with minimal septal dyskinesia (group I, $n = 65$), radical aneurysmectomy was performed.⁷ This involved resection of the entire scarred aneurysm wall, leaving a narrow rim near the contracting endocardium laterally and medially, avoiding the left anterior descending artery injury and entry into the right ventricular cavity. A linear anterior ventriculotomy closure was performed using interrupted sutures with Teflon (DuPont, Wilmington, Del) pledget reinforcement with minimal plication of the lateral wall. Attempts were made to reconstruct the LV apex without creating mid-ventricular narrowing.

In patients with a large dyskinetic septal scar (group II, $n = 70$), septal plication was performed by using interrupted sutures reinforced with Teflon pledgets. A Teflon patch (1 mm thick) was cut to an oval shape (with an average size of $3 \times 5 \text{ cm}$) to support the septum (septoplasty).⁸ The ventriculotomy was closed by including the supported septum that was sewn to the junction of the visible scar tissue and the myocardium (septal inclusion).

In group III ($n = 22$), there was minimal involvement of the septal wall. The borders of the scarred and viable (contracting) myocardium were identified, and the ventriculotomy opening was used to size a teardrop-shaped (or oval) patch of Dacron (Hemashield; Boston Scientific Corporation, Natick, Mass) to reconstruct the anterior wall of the LV. Moderate plication of the LV opening was achieved by an encircling purse-string suture or spacing of the sutures to secure the patch (Figure 1). Attempts were made to avoid oversizing the patch (the size averaged $2 \times 4 \text{ cm}$). It was sewn in place with 1 row of continuous sutures ensuring an ellipsoid LV cavity and a cone-shaped apex. Intraoperative transesophageal echocardiography was used extensively to assess the LV cavity size before and after the operation (Figure 2). The medial (septal) and the lateral remnants of the scarred aneurysm tissues were then trimmed, and the ventriculotomy was closed over the patch (septal exclusion).

Follow-up

Patients were followed up by direct contact, telephone interviews, and outpatient clinic visits. Follow-up intervals ranged from 15 months to 22 years (group I, 260 months; group II, 186 months; group III, 104 months). Follow-up management was performed by the primary care physicians. Patients were maintained on an optimized regimen for treatment of congestive heart failure that included use of β blockers and angiotensin-converting enzyme inhibitors. Follow-up cardiac catheterization was performed if the patients became symptomatic. Twenty patients (14%) were lost to follow-up because of tertiary referrals, patient migration, and unknown reasons.

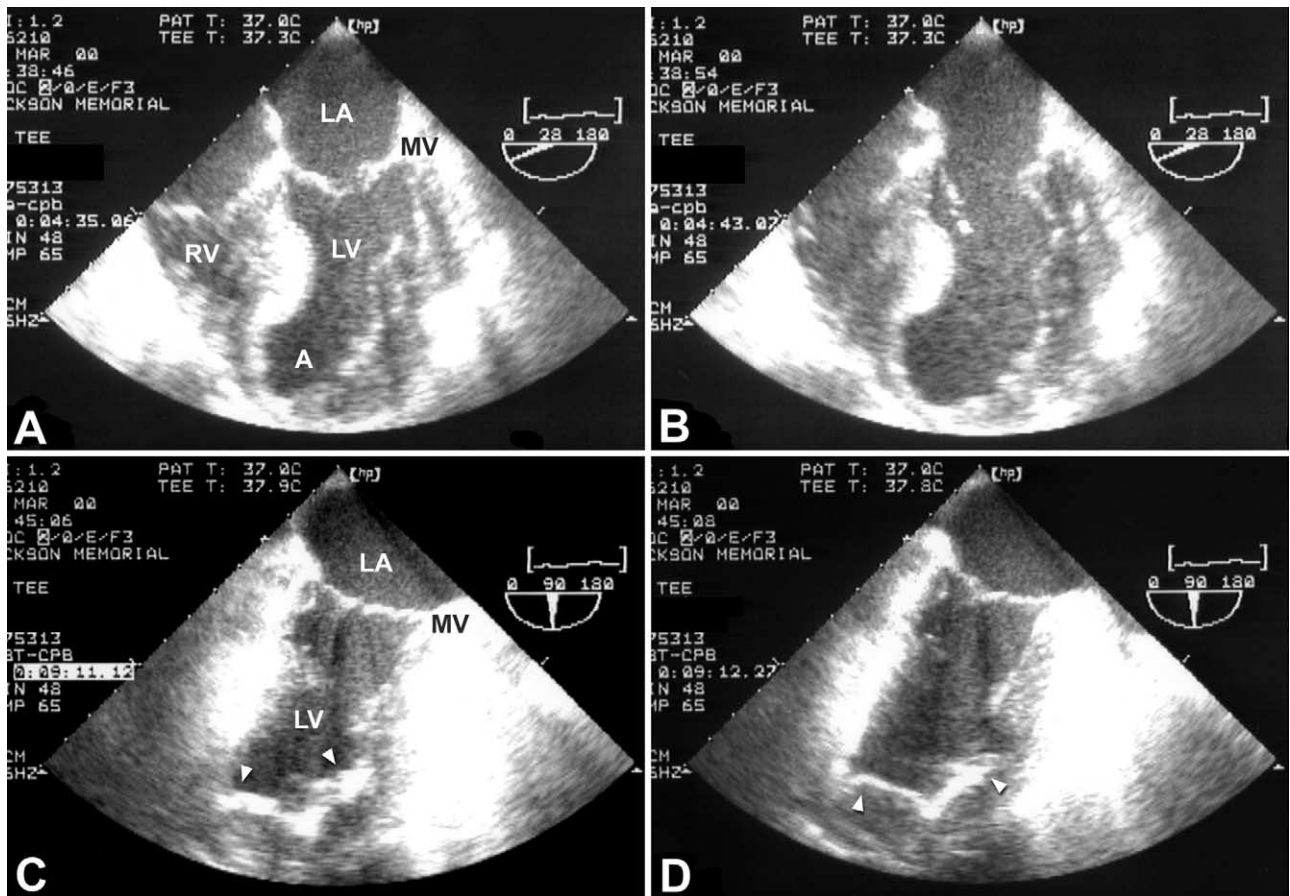


Figure 2. Intraoperative transesophageal echocardiogram. The views are in systole (A, C) and diastole (B, D) before (A, B) and after (C, D) volume reduction surgery in a patient in group III. Left atrium (LA), left ventricle (LV), mitral valve (MV), aneurysm (A), and right ventricle (RV) are identified. The arrowheads show the extent of the anterior endoventricular patch in systole (C) and diastole (D). Note the extent of LV volume reduction and apical restoration.

Statistical Analysis

Data were analyzed using the SAS System (SAS Institute Inc, Cary, NC). Continuous variables were expressed as mean ± SEM and categorical variables as frequency and percent. Univariable analysis of categorical determinants of mortality was performed by the χ^2 test and the Fisher exact test. Continuous variables were analyzed using the analysis of variance and Student *t* tests. Actuarial survival curves (Kaplan-Meier) were used to assess differential mortality among the 3 groups. Multivariable regression analysis (Cox model) was used to assess the effect of independent variables on survival. Wilcoxon and log-rank statistical analyses were applied to evaluate the survival curves.

Results

The operative procedures were performed from 1979 to 2000. Initially (1979-1992), most patients underwent radical aneurysmectomy with linear closure (group I, n = 47) or septal inclusion (group II, n = 57). Septal exclusion with endocardial patch was the technique used from 1992 to 2000 (group III, n = 22), whereas other operative tech-

niques (group I, n = 18; group II, n = 13) were used according to the pathologic findings as previously described. One patient underwent septoplasty in 1977 for acute MI, LV false aneurysm, and cardiogenic shock. He lived 80 months.

Hospital mortality included all deaths within the same hospitalization or within 30 days. There were 25 early postoperative deaths (16%). The major causes of deaths were persistent congestive failure, recurrent ventricular arrhythmia, and multiorgan failure. There was no significant difference in early deaths among the 3 groups of patients (Table 3). For the entire group, the risk factors associated with early mortality included class III and IV (New York Heart Association) congestive failure (*P* = .04), cardiogenic shock (*P* < .0001), use of an intra-aortic balloon pump (IABP) (*P* = .0001), and emergency or urgent operations (*P* = .0003).

For the entire group (including early mortality), the actuarial survival at 5, 10, and 15 years was 53%, 30%, and

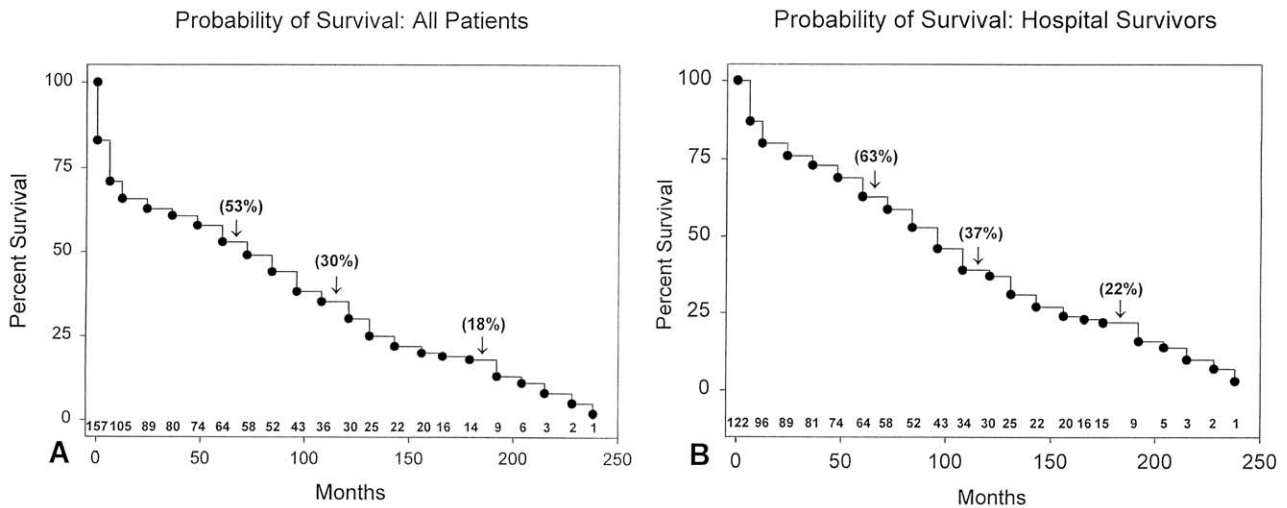


Figure 3. Actuarial survival for all patients at risk including (A) and excluding (B) early (operative) mortality.

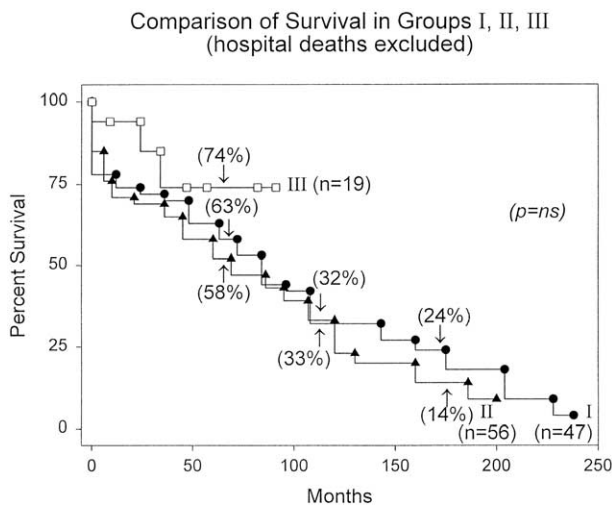


Figure 4. Comparison of actuarial survival in the 3 patient groups after LV volume reduction surgery: group I, radical resection; group II, septoplasty; group III, endoventricular patch. $P = .229$.

18%, respectively (Figure 3, A). Excluding the early death, the actuarial survival for the same intervals was 63%, 37%, and 22%, respectively (Figure 3, B). A comparison of the actuarial survival for the hospital survivors among the 3 groups is shown in Figure 4. At 5 years, 74% of group III patients (with endoventricular patch) were alive in comparison with 63% of group I and 58% of group II ($P = .229$). Comparison of actuarial survival for all patients at risk in group I (radical resection) and patients in group III showed no significant difference (Figure 5, A), whereas there was a significant difference in survival comparing all patients at risk in group III with those in group II (septoplasty). Survival at 5 and 7 years was 47% and 37% for group II,

respectively, compared with 67% and 67% for group III, respectively ($P = .04$) (Figure 5, B).

There were significant differences in the preoperative incidence of cardiogenic shock and of urgent or emergency operations and use of IABP among the groups. Group III patients had the lowest number of urgent or emergency operations 9% (2/22) compared with 40% (26/65) and 65% (46/70) in groups I and II, respectively ($P = .003$) (Table 3). There was a significant difference in survival among all patients undergoing operations electively ($n = 83$) compared with those undergoing emergency or urgent operations ($n = 74$). Survival was 64% versus 41% at 5 years and 53% versus 34% at 7 years for elective versus urgent or emergency operations, respectively ($P = .0006$, Wilcoxon; $P < .02$ log-rank). However, the difference in long-term survival among hospital survivors was not significant ($P = .31$) (Figure 6, F).

The early mortality for patients with cardiogenic shock in groups I, II, and III was 50% (4/8), 47% (8/17), and 0% (0/1), respectively. There was significantly lower survival (36% at 3 years) in all patients with cardiogenic shock than in patients without cardiogenic shock (68% at 3 years, $P = .0003$ by the log-rank test). However, among hospital survivors, patients with preoperative cardiogenic shock demonstrated 42% survival at 5 years versus 66% in patients without cardiogenic shock ($P = .137$).

The incidence of myocardial revascularization and the number of coronary bypass grafts per patient were similar in the 3 groups ($P = .398$) (Table 3). For the entire group, among 98 hospital survivors, the survival at 5 and 10 years was higher in patients with arterial grafts (74% and 35%) than in patients with venous grafts only (60% and 32%), respectively ($P = .360$).

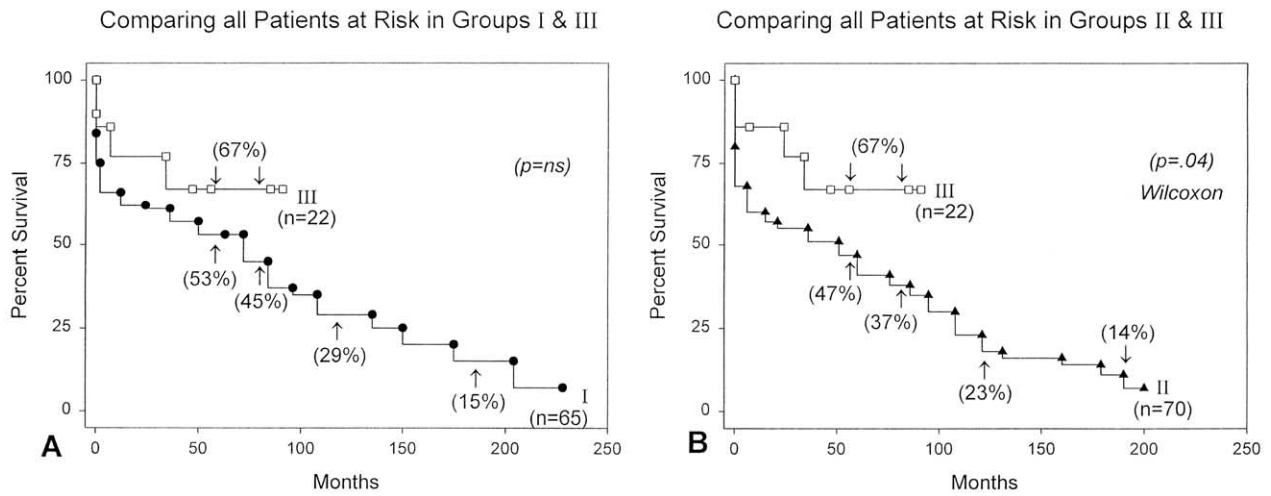


Figure 5. Actuarial survival including the early mortality comparing the results for the 3 operative techniques. A, All patients at risk in groups I and III are compared. *P* = .1591. B, All patients at risk in groups II and III are compared.

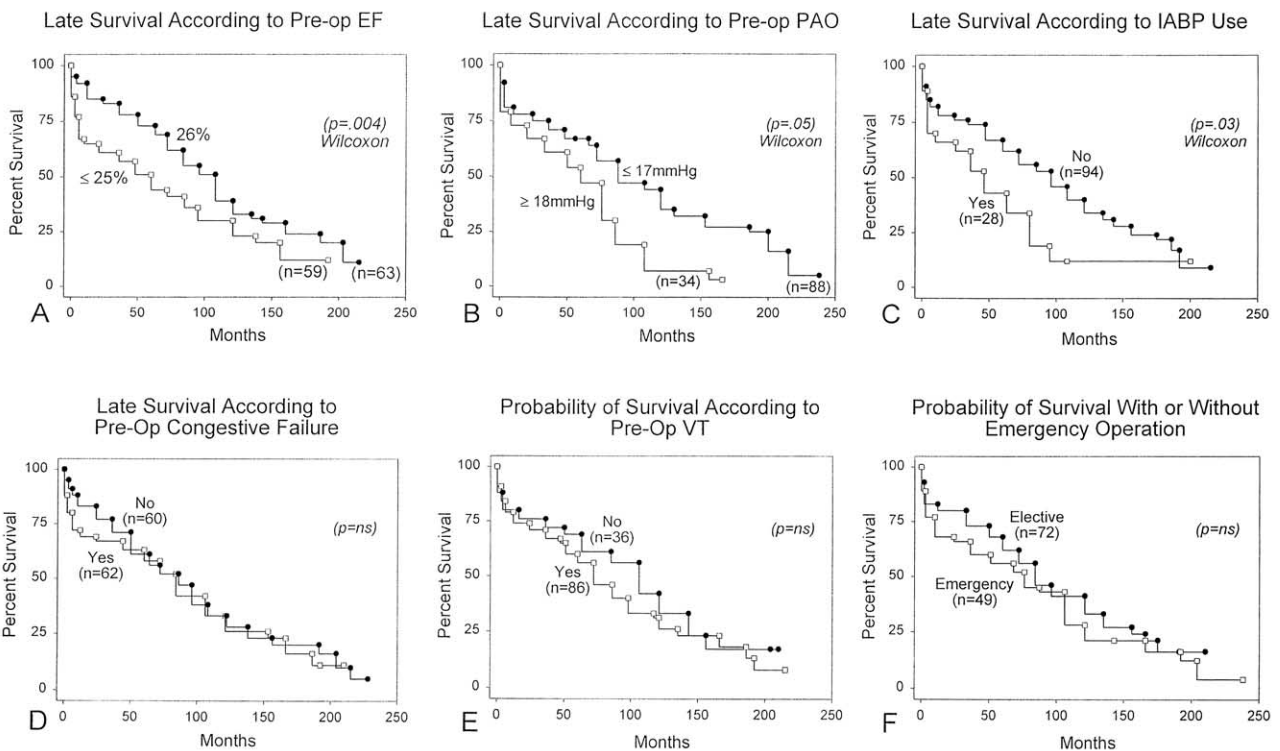


Figure 6. Actuarial late survival excluding early death according to preoperative variables: ejection fraction (EF), pulmonary artery occlusive (PAO) pressure, intra-aortic balloon pump (IABP), and ventricular tachyarrhythmia (VT). D, *P* = .3356; E, *P* = .2427; F, *P* = .3184.

Multivariate analysis was performed to identify the risk factor(s) that affected the late mortality. A total of 24 factors were considered including the following: operative technique, age, ejection fraction (EF), PAO pressure, cardiac

index, VT, cryoablation, congestive heart failure, cardiogenic shock, recent MI, use of arterial grafts, urgency of operation, and need for IABP. Preoperative factors that significantly affected the survival of all patients at risk and

TABLE 4. Cox regression analysis of multivariable predictors of late mortality in all hospital survivors after left ventricular volume reduction surgery

Variable	Hazard ratio	95 % CI	χ^2	P value
IABP	2.27	1.47–3.5	13.8	.0002
EF	1.49	1.01–2.20	3.9	.045
PAO	1.54	1.08–2.21	4.0	.044

CI, Confidence interval.

TABLE 5. Causes of late death

Cause	All patients (n = 76)	Group I (n = 37)	Group II (n = 38)	Group III (n = 1)
CHF	31	11	19	1
SD	14	5	9	—
MI	12	6	6	—
Cancer/sepsis	8	4	4	—
Unknown/other	11	11	—	—

hospital survivors included EF ($\geq 26\%$), PAO pressure (≤ 17 mm Hg), and no IABP (Table 4 and Figure 6, A-C). Factors that influenced early death but had no significant effect on long-term survival included preoperative congestive failure, VT, cardiogenic shock, recent MI, and emergency versus elective operations (Figure 6, D-F).

There were 76 deaths during the follow-up period (Table 5). Congestive heart failure was the most frequent cause. Cause of death was unknown in 11 instances. The hearts from 8 patients (in groups I and II) who died 6 months to 3 years postoperatively were examined by pathologists. The position and the extent of healing of the septal patch were impressive (Figure 7).

One patient in group III underwent cardiac transplantation and is doing well. On their last visit, the 36 surviving patients were in New York Heart Association symptom class I or II. Fourteen of these patients were older than 72 years and lived independently. Late cardiac catheterization data (5–16 years postoperatively) from 7 patients were reviewed. The LV angiogram in 5 patients showed decreased LV end-systolic and end-diastolic volumes and a slightly improved EF (Figure 8).

Discussion

LV remodeling is a natural consequence of transmural MI.^{10,11} It is enhanced by the extent of infarction and is a sequela of infarct expansion and wall stretch. These anatomic changes initially ameliorate cardiac dynamics in the form of an increase in blood pressure and cardiac output and a decrease in end-diastolic pressure, although at the expense of increase in LV volume, wall stress, and wall tension (La Place law).

Surgical methods of eliminating the noncontractile areas of the LV have been used since 1958.¹² The procedure is universally accepted. It improves cardiac systolic performance and in conjunction with myocardial revascularization improves long-term survival results.^{13–15} In recent years, a combination of medical management using percutaneous methods of revascularization followed by afterload reduction and β -blocker therapy has played a highly successful role in the prevention of LV aneurysm formation and remodeling.¹⁶

The intracavitary repair of LV aneurysm is a technique that has evolved as the result of the observation of a thin-walled cardiac septum that could not be resected and was frequently associated with a patent anterior descending artery suitable for bypass grafting.^{17–20} Few reports have been published that compare the late survival results (>5 years) with various methods of LV volume reduction.^{18,21} The early results have shown similar survival among the patient groups. No randomized trials have been performed.

A survey of changes in cardiac function and volume after the 3 types of repair, as discussed here, has been reported and is beyond the intent of this work.^{1,2,7,8,21} The incidence of preoperative congestive heart failure, angina, and recent MI in our study was similar in the 3 groups of patients and matches the reports by other investigators.^{5,13,14} The larger number of patients with VT in our study was possibly the result of referrals to our Sudden Death Center during the 1980s when catheter ablative therapy was not available. In recent years, the incidence of ventricular aneurysm surgery in general and the referral rate for surgical treatment of VT in particular have substantially declined.^{1,15}

In this study, a comparison of preoperative hemodynamic factors shows a difference in EF, cardiac index, and end-diastolic pressure in the 3 patient groups. The difference was significant in group III who had lower EF (22%) and higher pulmonary occlusive pressure (20 mm Hg) and end-diastolic pressure (23 mm Hg) values compared with groups I and II (Table 3). This difference may have negatively influenced the operative mortality (13%) and long-term survival of patients in group III. Despite a more depressed cardiac function, group III patients had a higher survival at 5 and 7 years compared with group II patients (67% vs 47% $P = .04$) (Figure 4). However, other factors, such as newer therapeutic regimen (eg, angiotensin-converting enzyme inhibitors and β blockers), may have contributed to the improved survival in group III as well.

A significant decrease in pulmonary occlusive (wedge) pressure after aneurysm resection has been reported by a number of investigators.^{15,17,21} Similarly, an elevated LV end-diastolic pressure markedly decreases postoperatively.^{4,13,14} For these reasons, we considered an elevated preoperative PAO pressure as a possible factor that may prognosticate long-term survival after the operation. The

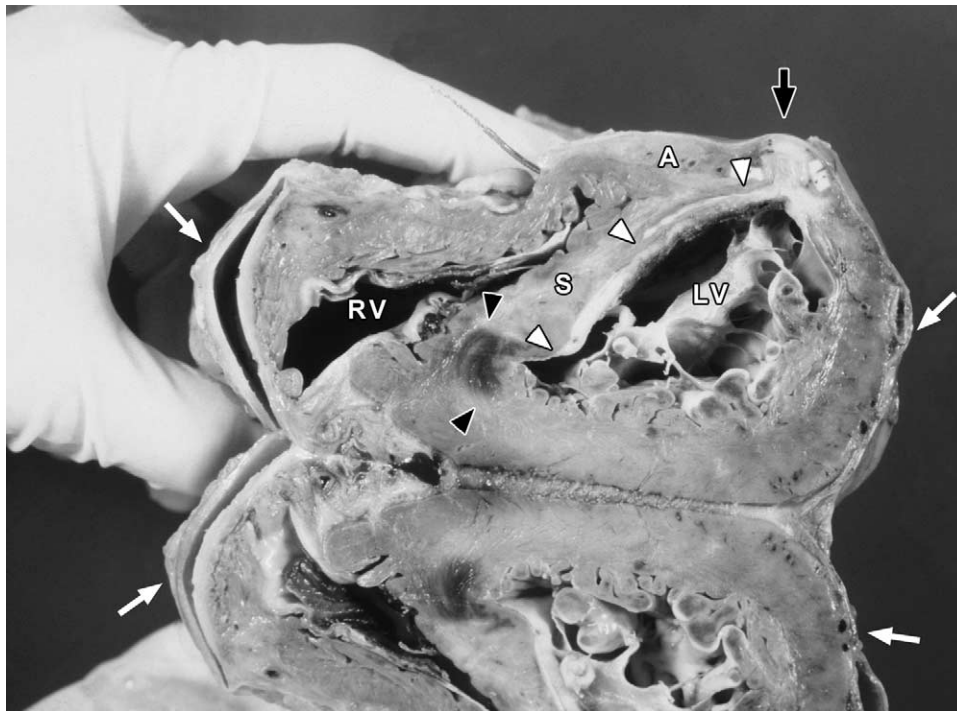


Figure 7. Heart specimen showing mid-ventricular section 6 months after volume reduction surgery with septoplasty technique (group II). The septal patch (white arrowheads), right ventricle (RV), left ventricle (LV), septum (S), and patent anterior descending artery (A) are shown. This patient died suddenly of cardiac dysrhythmia probably caused by a posterior septal infarction (black arrowheads). White arrows indicate 2 implanted defibrillator patches. Note the suture line (black arrow), the thick wall LV cavity, and the septal wall with the Teflon patch.

accepted maximum normal value of 18 mm Hg was considered to be the cutoff point. There was a significant difference in survival ($P = .05$) among hospital survivors with preoperative PAO pressure of 17 mm Hg or less and those with PAO pressure of 18 mm Hg or greater. This finding is not supported by previous reports, probably because of the lack of survival analysis considering this factor or use of mean pulmonary artery pressure readings.^{5,21,22} Mickleborough and colleagues¹⁵ have considered severe pulmonary hypertension as a contraindication to this procedure. We believe that patients with a high intracardiac diastolic pressure and congestive failure in association with preserved LV systolic function have diastolic dysfunction.²³ This condition may not improve with LV volume reduction surgery, especially in patients with a mostly akinetic LV aneurysm. Instead, these patients may benefit from circulatory assist devices aiming for cardiac transplantation.

The incidence of cardiogenic shock and the use of an IABP were higher in this study compared with other reports.^{14,15,21} As shown in Table 4, the use of an IABP was associated with a high probability of poor early and late outcomes. Factors that showed no significant effect on long-term survival, if the initial operative mortality was excluded, were congestive heart failure, preoperative VT, the

use of arterial grafts for coronary bypass, and whether the operation was urgent, emergency, or elective (Figure 6). In this regard, the results of our study do not conform to some of the previous reports that involved follow-up times of 2 to 5 years.^{3,15,24} A longer follow-up may be necessary to clarify these points.

In the past, the goals of surgical management of patients with a large anteroseptal LV aneurysm involved the elimination of the dyskinetic segment with little regard to the restoration of LV cavity size and shape. In recent years, the functional significance and the effects on long-term survival of LV configuration have been realized.^{19,24-26} Our study shows that if these concerns are met, the survival results of surgical management of LV aneurysm will be satisfactory, especially with the endoventricular patch technique.

Recently, Athanasuleas and colleagues⁴ reported for the RESTORE group of surgeons on 439 patients who were studied prospectively and had received anterior ventricular patch repair. There was an in-hospital mortality rate of 6.6%, a balloon pump was used in 7% of patients, and the preoperative EF was $29\% \pm 10\%$ (SD). The overall early survival (at 18 months) was 84%. These early results are impressive and probably the result of proper patient selection and the experience of the entire group. In an elegant

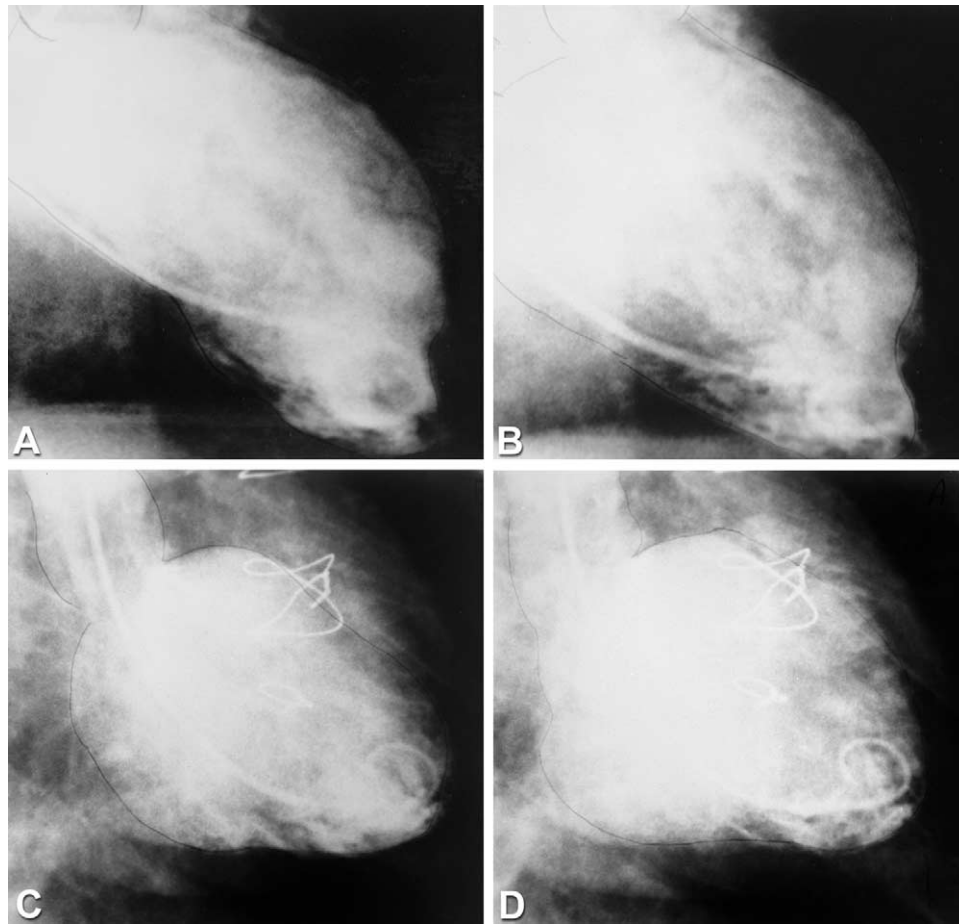


Figure 8. Preoperative (A, B) and postoperative (C, D) left ventriculograms in systole (A, C) and diastole (B, D) in a patient in group III. Preoperatively, the apex contained islands of clots. Note restoration of LV apex and improvement in systolic function.

monograph, Buckberg and colleagues²⁷ described in detail the basis for their concept in repairing the LV cavity considering the oblique anterior ventricular wall fiber orientation. The reconstruction of the anterior wall and the apex cone was accomplished with a 2 × 3-cm patch.²⁸ We used a larger patch with an average size of 2 × 4 cm (in group III) that was cut to an ovoid shape to accommodate the LV outflow and construct the LV apex. It is possible that downsizing the patch would play a role in patient survival by decreasing the LV volume. The LV end-systolic volume index has been shown to significantly affect patient survival and freedom from cardiac morbidity.⁵

With recent improvement in medical and surgical treatment of ischemic heart disease, the indications for volume restoration surgery have expanded to include patients with small aneurysms and minimal symptoms.²⁹ The variability of indications for this operation necessitates a prospective randomized trial to identify the method of treatment that consistently improves the long-term survival of these patients.

Drs Gerald Kaiser, Richard Perryman, Michael Horowitz, Kenneth Herskowitz, and George Palatianos (presently director of Cardiac Surgery Service at Onassis Cardiac Center in Athens, Greece) collaborated in the early part of this study. I am grateful to Mrs Joanne Bolooki for typing and editing this manuscript.

References

1. Dor V, Sabatier M, Di Donato M, Montiglio F, Toso A, Maioli M. Efficacy of endoventricular patch plasty in large postinfarction akinetic scar and severe left ventricular dysfunction: comparison with a series of large dyskinetic scars. *J Thorac Cardiovasc Surg.* 1998;116:50-9.
2. Elefteriades JA, Solomon LW, Salazar AM, Batsford WP, Baldwin JC, Kopf GS. Linear left ventricular aneurysmectomy: modern imaging studies reveal improved morphology and function. *Ann Thorac Surg.* 1993;56:242-52.
3. Kawata T, Kitamura S, Kawachi K, Morita R, Yoshida Y, Hasegawa J. Systolic and diastolic function after patch reconstruction of left ventricular aneurysms. *Ann Thorac Surg.* 1995;59:403-7.
4. Athanasuleas CL, Stanley AWH Jr, Buckberg GD, Dor V, Di Donato M, Blackstone EH, and the RESTORE group. Surgical anterior ventricular endocardial restoration (SAVER) in dilated remodeled ventricle after anterior myocardial infarction. *J Am Coll Cardiol.* 2001;37:1199-209.
5. Di Donato M, Toso A, Maioli M, Sabatier M, Stanley AWH Jr, Dor V,

- and the RESTORE group. Intermediate survival and predictors of death after surgical ventricular restoration. *Semin Thorac Cardiovasc Surg.* 2001;13:468-75.
6. Palatianos GM, Bolooki H, Horowitz MD, Lowery MH, Rosenthal SP, Chandarlapaty SKC, et al. Sequential internal mammary artery grafts for coronary artery bypass. *Ann Thorac Surg.* 1993;56:1136-40.
 7. Palatianos GM, Craythorne CB, Schor JS, Bolooki H. Hemodynamic effects of radical left ventricular scar resection in patients with and without congestive failure. *J Surg Res.* 1988;44:690-5.
 8. Bolooki H, Palatianos GM, Zaman L, Thurer RJ, Luceri RM, Myerburg RJ. Surgical management of post-myocardial infarction ventricular tachyarrhythmia by myocardial debulking, septal isolation, and myocardial revascularization. *J Thorac Cardiovasc Surg.* 1986;92:716-25.
 9. Bolooki H, Horowitz MD, Interian A, Thurer RJ, Palatianos GM, De Marchena EJ, et al. Long-term surgical results in sudden death syndrome associated with cardiac dysfunction after myocardial infarction. *Ann Surg.* 1992;216:333-43.
 10. McKay RG, Pfeffer MA, Posternak RC, Markis JE, Come PC, Nakao S, et al. Left ventricular remodeling after myocardial infarction: a corollary to infarct expansion. *Circulation.* 1986;74:693-702.
 11. Pfeffer MA, Braunwald E. Ventricular remodeling after myocardial infarction. Experimental observations and clinical implications. *Circulation.* 1990;81:1161-72.
 12. Cooley DA, Henly WS, Ahmad KH, Chapman DW. Ventricular aneurysm following myocardial infarction: results of surgical treatment. *Ann Surg.* 1959;150:595-612.
 13. Pasini S, Gagliardotto P, Punta G, Del Ponte S, Serra M, Parisi F, et al. Early and late results after surgical therapy of post infarction left ventricular aneurysm. *J Cardiovasc Surg.* 1998;39:209-15.
 14. Couper GS, Bunton RW, Birjiniuk V, Disesa VJ, Fallon MP, Collins JJ Jr, et al. Relative risks of left ventricular aneurysmectomy in patients with akinetic scars versus true dyskinetic aneurysms. *Circulation.* 1990;82(Suppl IV):IV248-56.
 15. Mickleborough LL, Carson S, Ivanov J. Repair of dyskinetic or akinetic left ventricular aneurysm: results obtained with a modified linear closure. *J Thorac Cardiovasc Surg.* 2001;121:675-82.
 16. Pfeffer MA, Lamas GA, Vaughan DE, Parisi AF, Braunwald E. Effect of captopril on progressive ventricular dilatation after anterior myocardial infarction. *N Engl J Med.* 1988;319:80-6.
 17. Dor V, Bournon DF, Sabatier M, Grinneiser D, Montiglio F, Coste P, et al. La reconstruction du ventricule gauche par plastie circulaire endoventriculaire avec exclusion septale. *Arch Mal Coeur.* 1990;83:1687-94.
 18. Buckberg GD. Congestive heart failure: treat the disease, not the symptom—return to normalcy. *J Thorac Cardiovasc Surg.* 2001;121:628-37.
 19. Cooley DA. Ventricular endoaneurysmorrhaphy: simplified repair for extensive postinfarction aneurysm. *J Card Surg.* 1989;4:200-5.
 20. Cosgrove DM, Lytle BW, Taylor PC, Stewart RW, Golding LAR, Mahfood S, et al. Ventricular aneurysm resection. Trends in surgical risk. *Circulation.* 1989;79(Suppl I):I97-101.
 21. Shapira OM, Davidoff R, Hilkert RJ, Aldea GS, Fitzgerald CA, Shemin RJ. Repair of left ventricular aneurysm: long-term results of linear repair versus endoaneurysmorrhaphy. *Ann Thorac Surg.* 1997;63:701-5.
 22. Grossi EA, Chinitz LA, Galloway AC, Delianides J, Schwartz DS, McLoughlin DE, et al. Endoventricular remodeling of left ventricular aneurysm. Functional, clinical and electrophysiological results. *Circulation.* 1995;(Suppl II):II98-100.
 23. Vasan RS, Levy D. Defining diastolic heart failure. A call for standardized diagnostic criteria. *Circulation.* 2000;101:2118-21.
 24. White HD, Norris RM, Brown MA, Brandt PWT, Whitlock RML, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation.* 1987;76:44-51.
 25. Jatene AD. Left ventricular aneurysmectomy: resection or reconstruction. *J Thorac Cardiovasc Surg.* 1985;89:321-31.
 26. Mills NL, Everson CT, Hockmuth DR. Technical advances in the treatment of left ventricular aneurysm. *Ann Thorac Surg.* 1993;55:792-800.
 27. Buckberg GD, Coghlan HC, Torrent-Guasp F. The structure and function of the helical heart and its buttress wrapping. VI. Geometric concepts of heart failure and use for structural corrections. *Semin Thorac Cardiovasc Surg* 2001;13:386-401.
 28. Anthanasuleas CL, Buckberg GD, Menicanti L, Gharib Mand the, RESTORE Group. Optimizing ventricular shape in anterior restoration. *Semin Thorac Cardiovasc Surg.* 2001;13:459-67.
 29. Jones RH. Is it time for a randomized trial of surgical treatment of ischemic heart failure? *J Am Coll Cardiol.* 2001;37:1210-3.

Discussion

Dr Gerald D. Buckberg (*Los Angeles, Calif*). I would like to compliment Dr Bolooki for an important review of the geometric changes after anterior infarction that link the evolution of corrective measures with early and late results. Improvement followed changing from a linear resection to include the septum, to plicating the dyskinetic septum with a triangular patch as described in 1986, to more recently excluding the septum with an oval patch to restore an elliptical shape. The composite results may lead to some confusion because different reasons can explain why high-risk components of EF, pulmonary artery pressure, and the need for a balloon will raise mortality. From a mechanical standpoint, patch exclusion of the septum is a more complete procedure and was more successful in his presentation, despite the occurrence of the lowest EF of 22% and highest PA pressure of 21 mm Hg. Furthermore, adding patients with cardiogenic shock raises early mortality. Is their analysis without this data available?

Dr Bolooki. Yes. The article has more detailed data analysis. You are correct, patients with an intraventricular patch had overall higher survival results.

The composite slide (Figure 6) showed that by excluding the operative mortality, there is approximately a 10% better survivorship at 5 years, 7% at 10 years, and 4% at 15 years for the 3 patient groups. There were no statistically significant differences in overall survival among the 3 groups at 5 years. However, at 7 years of follow-up, the difference in survival was better for group III (patch exclusion of the septum) than for group II (septal inclusion). Preoperative use of a balloon pump, cardiogenic shock, and emergency operations had a significant effect on operative (in-hospital death) survival, but only balloon-pump use had an effect on long-term survival curves.

Dr Buckberg. I think that is correct, because a clearer picture emerges when you look at elective high-risk patients. We recently analyzed 662 patients, mostly in class III or IV heart failure, and the balloon was only needed in approximately 8%. Hospital mortality for restoration without repair was approximately 4%, so that goes along with what you are saying. I think that separating elective versus urgent operations will reduce the mortality as you describe, and you demonstrate that in your slide.

It seems that the consideration of methods of protection may suggest that the beating empty heart, rather than cardioplegia, may be useful for restoration when there is trabecular scar. This is because aneurysms are fairly rare today with the use of thrombolysis and percutaneous transluminal coronary angioplasty. However, the akinetic heart is relatively common. The thick akinetic heart is seen by echocardiography and ventriculography, and it does not collapse by venting. The junction between contracting and noncontracting muscle is found by palpation. I wonder how