Operative Risks and Long-Term Results of Operation for Left Ventricular Aneurysm

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A review of 336 consecutive patients who underwent repair of left ventricular aneurysm from 1978 to 1989 disclosed that partial resection of the aneurysm and conventional closure of the ventriculotomy was performed in 281 patients, inverted T closure in 17, and endocardial patch in 38. These two latter techniques were developed in an attempt to restore normal left ventricular geometry. The operative mortality was 6.8% (23 patients). A stepwise logistic regression analysis of various preoperative clinical, hemodynamic, and angiographic variables revealed that left ventricular ejection fraction of 0.20 or less, age greater than 60 years, previous myocardial revascularization, lack of angina, and New York Heart Association functional class IV were independent predictors of operative mortality. The technique of repair was not a predictor of outcome, but when patients with poor left ventricular function were analyzed separately, the operative mortality was reduced from 12.5% to 6.5% when newer techniques were employed. Patients were followed up during a mean of 6.3 years. There have been 51 late deaths, 45 cardiac. Cox regression analysis indicated that poor left ventricular function and left main coronary artery stenosis were the only two predictors of late mortality. The actuarial survival at 10 years was 63% ± 4%. Most patients (88%) are in New York Heart Association class I or II. These data indicate excellent long-term results after repair of left ventricular aneurysm. Newer techniques of repair are valuable in patients with poor left ventricular function.

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Surgical repair of left ventricular aneurysm (LVA) was first performed by Charles Bailey in 1955 [1]. A large clamp was applied to the neck of the aneurysm and, after its base was sutured, a segment measuring 5 by 7 cm was resected [1]. The first resection under cardiopulmonary bypass was reported by Cooley and associates in 1958 [2]. The operative technique for resection of LVA remained unchanged for almost three decades. As surgeons recognized the importance of left ventricular geometry in left ventricular function, attention was turned to new methods of repairing the aneurysm to improve surgical results. In his Honored Guest's Address to the American Association for Thoracic Surgery in 1984, Adib Jatene from Brazil introduced the concept of left ventricular "reconstruction" for patients with chronic LVAs [3]. That was a timely presentation because many surgeons were looking at different methods to repair the left ventricle in such patients. It is now generally accepted that whatever technique one employs to repair an LVA, restoration of left ventricular geometry is important to optimize left ventricular function.

This report is a review of our experience with different methods of repairing LVAs, the operative risks associated with them, and the long-term results.

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Material and Methods

Patients and Operative Technique

From January 1978 to December 1989, 365 patients underwent operation for LVA. A careful review of the operative notes and left ventricular angiographic reports indicated that 29 patients had had small apical LVAs that were repaired by simple plication with a few sutures. These patients were excluded from this study. The remaining 336 patients had resection of part of the aneurysm and repair of the ventricular wall by one of the following methods: conventional closure of the ventriculotomy (281 patients), inverted T closure (17 patients), or endocardial patch with Dacron graft or glutaraldehyde-preserved bovine pericardium (38 patients) as illustrated in Figure 1. The inverted T closure of the ventriculotomy and the endocardial patch techniques were developed in an attempt to restore normal left ventricular geometry. We started to employ these two techniques in 1982 and in 1984, respectively.

There were 278 men and 58 women whose mean age was 55.7 years (range, 25 to 83 years). Table 1 summarizes pertinent clinical, hemodynamic, and angiographic data in these patients. All patients underwent heart catheterization and coronary arteriography before operation. Left ventricular ejection fraction was assessed by contrast ventriculography in 317 patients and by radionuclide angiography or echocardiography in 19. The LVA was anteroseptal in 321 patients and posterior in 15.

All operations were performed on cardiopulmonary bypass and mild systemic hypothermia. Aortic cross-

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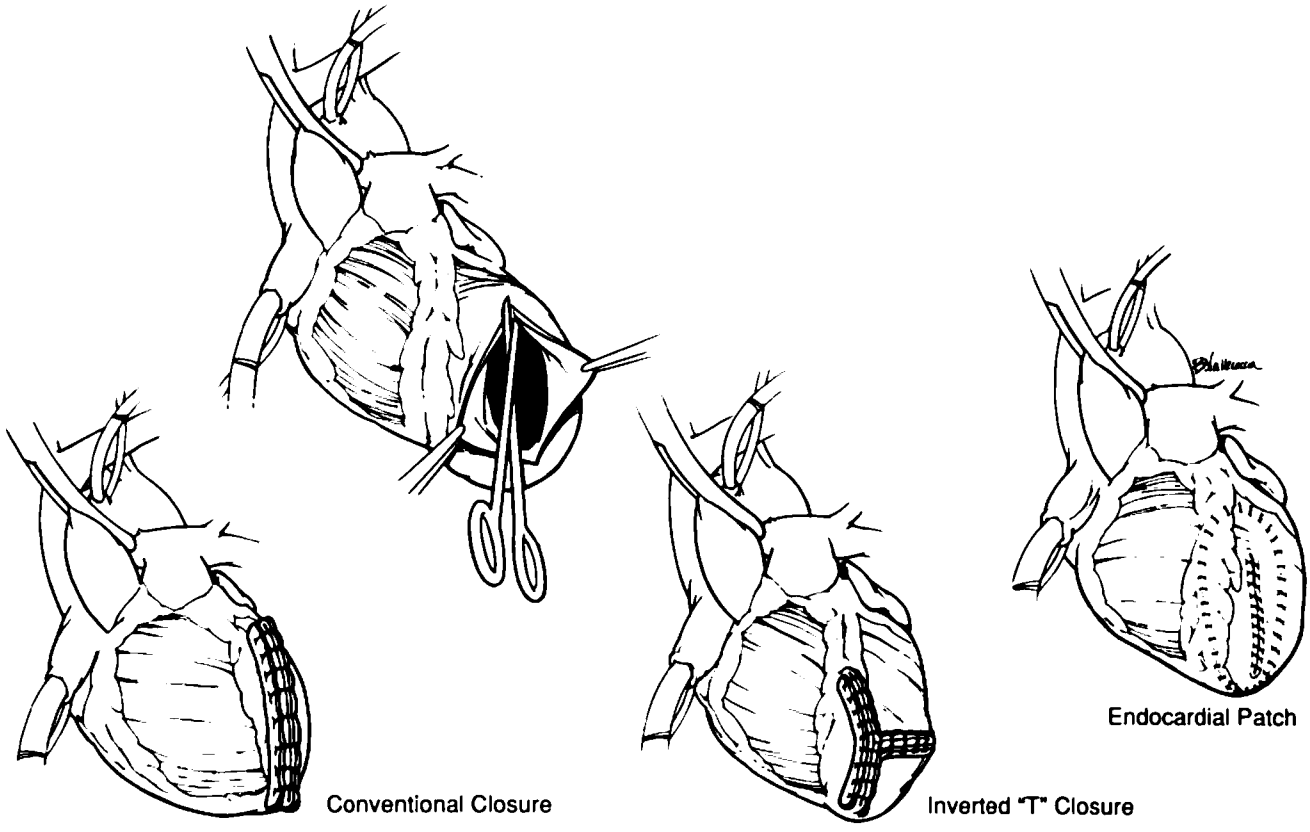

Fig 1. Operative techniques.

clamping and cold cardioplegia were employed in all patients. Crystalloid cardioplegia was used in the first 207 patients and blood cardioplegia in the last 129. Since we have begun to use blood cardioplegia, the repair of the LVA and construction of all distal and proximal anastomoses were performed with the aorta cross-clamped. When crystalloid cardioplegia was used, only the distal anastomoses were performed with the aorta cross-clamped.

At operation, 325 aneurysms were chronic and 11 were acute due to recent transmural myocardial infarction; 326 aneurysms were true aneurysms and 13 were false. Thrombus was present in 64 aneurysms. Twelve patients who had documented preoperative ventricular tachycardia or fibrillation had resection of the aneurysm without intraoperative electrophysiologic studies. All these 12 patients were operated on before 1984.

Coronary artery bypass grafts were performed in all but 17 patients. The average number of coronary arteries grafted was 2.6 per patient (range, 1 to 5). One internal mammary artery was used for grafting in 38 patients, and two arteries in 6. Endarterectomy of the right coronary artery was performed in 10 patients. In patients in whom the left anterior descending artery was ligated during repair of the LVA and the right coronary artery was totally occluded, an effort was made to bypass at least one marginal branch supplying the right ventricular wall. Bypass grafting to one marginal branch of the right coronary artery was performed in 28 patients.

The aortic cross-clamping time was 45.7 ± 18 minutes, and the cardiopulmonary bypass time was 87 ± 29 minutes.

Intraaortic balloon pump was inserted preoperatively in 27 patients because of intractable angina, heart failure, or cardiogenic shock, and postoperatively in 61 patients because of low cardiac output.

Statistical Analysis

Statistical analysis was performed with the SAS (Cary, NC) and BMDP (Los Angeles, CA) statistical programs. Clinical, hemodynamic, and angiographic data were tested to determine their value in predicting operative mortality, postoperative low cardiac output syndrome, and late mortality. Statistically significant variables were entered in a stepwise logistic regression analysis to determine their independent value. Actuarial survival curves were constructed using the Kaplan-Meier method.

Results

The operative mortality was 6.8% (23 patients). The principal cause of death was low cardiac output syndrome in 19 patients, ventricular arrhythmias in 1, stroke in 1, mesenteric artery occlusion in 1, and sepsis in 1 (Appendix Table 1A). Table 2 summarizes all serious postoperative complications.

Postoperative Low Cardiac Output Syndrome

This was the most common cause of death. A stepwise logistic regression analysis of all preoperative clinical,

angiographic, and hemodynamic data indicated that the following variables were independent predictors of postoperative low cardiac output syndrome: left ventricular ejection fraction of 0.20 or less, previous myocardial revascularization, New York Heart Association functional class IV, and age greater than 60 years (Appendix Table 2A).

Operative Mortality

A stepwise logistic regression analysis indicated that left ventricular ejection fraction less than 0.20, age greater than 60 years, previous myocardial revascularization, lack of angina before operation, and New York Heart Association class IV were independent predictors of operative mortality (Appendix Table 3A).

Table 1. Clinical, Hemodynamic, and Angiographic Data in 336 Patients

Parameter	Number[a]
Previous myocardial revascularization	25 (7.4)
Angina pectoris:	
None	35 (10.4)
Stable angina	181 (53.8)
Unstable angina	120 (35.7)
Congestive heart failure	161 (47.9)
Systemic thromboembolism	18 (5.3)
Ventricular tachycardia/fibrillation	12 (3.5)
Myocardial infarction <30 days	
Subendocardial	9 (2.6)
Transmural	23 (6.8)
NYHA functional classification	
I	8 (2.3)
II	50 (14.8)
III	124 (36.9)
IV	154 (45.8)
Left ventricular end-diastolic pressure	
<25 mm Hg	201 (59.8)
>25 mm Hg	135 (40.1)
Left ventricular ejection fraction	
>0.54	3 (0.8)
0.35–0.54	44 (13.0)
0.21–0.34	140 (41.6)
<0.21	149 (44.3)
Coronary artery disease	
Left main stenosis	41 (12.2)
Triple-vessel	213 (63.3)
Double-vessel	63 (18.7)
Single-vessel	19 (5.6)
Indications for operation	
Angina pectoris	158 (47.0)
Congestive heart failure	29 (8.6)
Angina + heart failure	132 (39.2)
Other	17 (5.0)

[a] Numbers in parentheses are percentages.

NYHA = New York Heart Association.

Table 2. Postoperative Complications

Complication	Number[a]
Reexploration of the mediastinum	
For bleeding	14 (4.1)
For resuscitation	4 (1.1)
Low cardiac output syndrome	131 (38.9)
Intraaortic balloon pump[b]	61 (18.1)
Thromboembolism	
Cerebral (stroke)	11 (3.2)
Mesenteric	1 (0.3)
Arrhythmias	
Atrial fibrillation/flutter	32 (9.5)
Premature ventricular beats	114 (33.9)
Ventricular tachycardia/fibrillation	29 (8.6)
Renal failure	14 (4.1)
Respiratory failure	12 (3.5)

[a] Numbers in parentheses are percentages. [b] All because of low cardiac output syndrome.

Operative Technique

The method of repair of the LVA did not affect the overall operative mortality. However, when patients with severely impaired left ventricular function (ejection fraction of 0.20 or less) were analyzed separately, the operative mortality in those who had conventional repair was 12.6% (13 deaths among 103 patients), and in those who had inverted T closure or endocardial patch it was 6.5% (3 deaths among 46 patients). This difference was statistically significant ($p < 0.01$).

Follow-up

Patients were followed up from 1 to 13 years (mean, 6.3 years). Twenty-two patients (6.6%) were lost to follow-up. There have been 51 late deaths, 45 of which were caused by congestive heart failure, myocardial infarction, or sudden death. Cox regression analysis revealed that only left ventricular ejection fraction of 0.20 or less and left main coronary artery stenosis were independent preoperative predictors of late mortality (Appendix Table 4A). Figure 2 shows the actuarial survival of the entire group of patients. The 10-year survival was 63% ± 4%. Figure 3 shows the actuarial survival curves according to preoperative left ventricular ejection fraction, and Figure 4 shows the actuarial survival curves for patients with and without left main disease. At the latest follow-up 158 patients (66%) were in New York Heart Association class I, 50 (21%) in class II, 27 (11%) in class III, and 5 (2%) in class IV. Nine patients have suffered strokes. Eleven patients have undergone repeat myocardial revascularization.

Comment

Operation for LVA began in the late 1950s, and coronary artery bypass in the late 1960s. The addition of myocardial revascularization in patients with LVA and multivessel coronary artery disease reduced the operative mortality

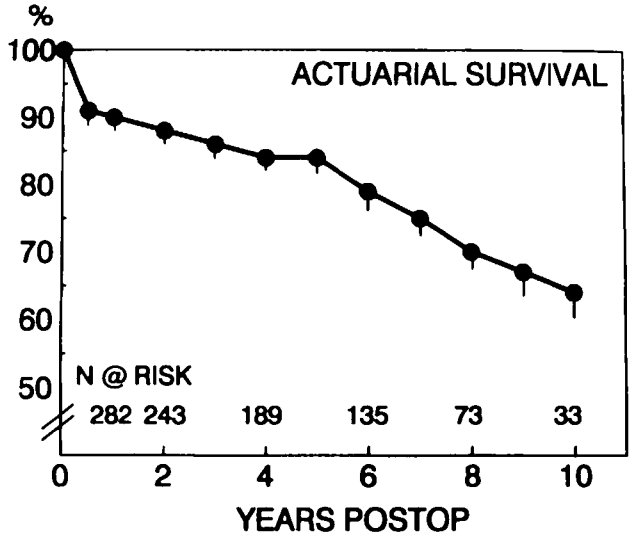

Fig 2. Overall actuarial survival of patients who had repair of left ventricular aneurysm.

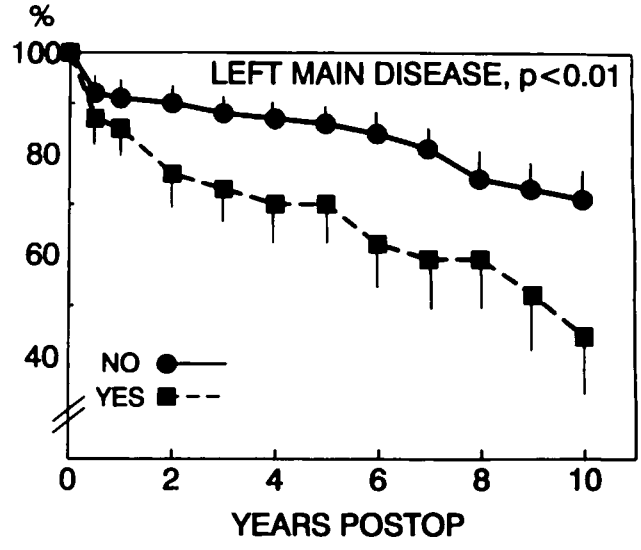

Fig 4. Effect of left main coronary artery stenosis on actuarial survival.

and increased long-term survival [4–6]. The results of operation for LVA continued to improve in the 1970s with the development of the intraaortic balloon pump and cardioplegic solutions, but by 1980 the operative mortality was yet relatively high in comparison with other cardiac operations. This fact, plus the lack of objective evidence that resection of the aneurysm improved resting left ventricular function [7], prompted many surgeons to develop alternate methods of repairing LVAs.

Jatene [3] pointed out the importance of restoring left ventricular geometry during repair of LVA to reduce operative mortality and improve long-term survival. His method of left ventricular reconstruction consisted in opening the aneurysm in the empty, beating heart and passing one or two pursestring sutures in and out of the ventricle at the transition of normal and fibrous tissue. Pulling on these sutures would restore the left ventricular cavity to its original shape. Depending on the size of the original myocardial infarction, the ventriculotomy could be closed primarily or by a Dacron patch. Jatene reported a reduction in operative mortality from 12.6% to 3.5% using this technique [3].

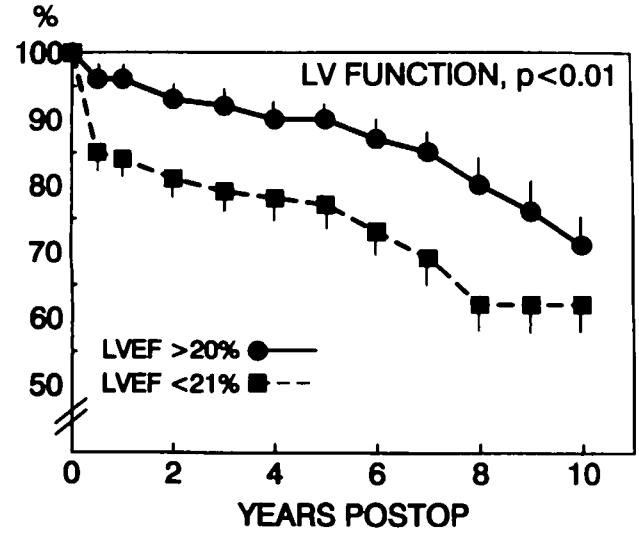

Fig 3. Effect of left ventricular (LV) function on actuarial survival.

Our attempt at restoring left ventricular geometry during repair of LVA consisted of closing the ventriculotomy in an inverted T fashion (see Fig 1). This type of closure restores the left ventricle to its conical shape. The drawback of this method of closing the ventriculotomy as well as the one described by Jatene is that they cannot be satisfactorily applied in patients with extensive fibrosis of the interventricular septum. In these cases, we believe that reconstruction of the left ventricular cavity with a Dacron patch sutured directly to the endocardium at the transition zone between scarred and viable myocardium is preferable. The size and shape of the patch should resemble those of the original myocardial infarction. This is not always easy to do in patients with chronic aneurysms because of the distortion in the anatomy of the left ventricle. It is simpler in patients with acute LVA. We prefer to use glutaraldehyde-preserved bovine pericardium to patch the endocardium in patients with acute LVA because it is easier to sew to healthy muscle than is Dacron graft.

Our data demonstrated that these newer techniques of repair of LVA are valuable in patients with severely impaired left ventricular function. The operative mortality was reduced from 12.6% when conventional repair was used to 6.5% when inverted T closure or endocardial patch was used. The endocardial patch technique is also valuable in patients with acute aneurysms who need

operation because of shock or intractable heart failure. We had no operative deaths among 8 patients with acute LVA who were treated by this method. We have also used this technique in patients with postinfarction ventricular septal defect with most impressive results [8]. This newer technique of repair of LVA does not seem to be important in patients with only moderate impairment of left ventricular function.

The impairment of left ventricular function in patients with LVA may be due to distortion of left ventricular geometry by the aneurysm or by fibrosis of other segments of the ventricular wall due to extensive coronary artery disease [3, 6, 9]. The importance of complete revascularization should not be overlooked [5, 6, 9]. We believe that this should include bypassing the proximal third of the left anterior descending artery or the first septal perforator branch if the proximal part of the septum is found not to be scarred at operation. Another detail during revascularization is that the distal half of the anterior descending artery is often ligated during closure of the ventriculotomy [4], and it may have been the principal source of collaterals to the marginal branches of an occluded right coronary artery. Although postoperative right ventricular dysfunction is common after repair of LVA [10], it can be an extremely serious [11] and sometimes fatal complication. We believe that at least one marginal branch of the right coronary artery should be bypassed in this subset of patients with LVA.

A stepwise logistic regression analysis of multiple clinical, hemodynamic, and angiographic variables in our patients identified poor left ventricular function, age greater than 60 years, previous myocardial infarction, lack of angina pectoris, and New York Heart Association functional class IV as independent predictors of operative mortality. Other investigators have come to similar conclusions [12–15]. Repair of LVA alone should not be performed in patients with ventricular tachycardia or fibrillation. Early in our experience we had a high mortality in this group of patients. The operative risk is decreased by intraoperative electrophysiologic mapping to guide resection or ablation of the arrhythmogenic focus [16, 17].

The long-term survival after repair of LVA in our series is comparable with that of other reports [6, 13]. Most investigators have identified poor left ventricular function as an important predictor of late mortality [9, 13–15]. The 10-year actuarial survival in our patients with moderate impairment of left ventricular function was 71% ± 7%; for patients with severe impairment it was 61% ± 6%. This difference in long-term survival was statistically significant. Patients with poor left ventricular function usually have symptoms of congestive heart failure before operation. Thus, heart failure has also been identified as a predictor of late mortality [6, 9, 13]. The best long-term results were obtained in patients who were operated on primarily for angina pectoris [6, 9, 13, 15]. In our series the presence of left main coronary artery stenosis was also a predictor of late mortality after repair of LVA.

Our data indicate good long-term results after repair of LVA. Techniques aimed to restore normal left ventricular geometry have a favorable effect in operative mortality in patients with poor left ventricular function. They may also improve long-term survival.

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Appendix

Table 1A. Operative Mortality in Various Subsets of Patients With Left Ventricular Aneurysm

Variable	No. of Patients	No. of Deaths[a]	*p* Value[b]
Sex			0.246
Male	278	17 (6.1)	
Female	52	6 (10.3)	
Age			0.060
60 y or less	221	11 (5.0)	
>60 y	115	12 (10.4)	
Previous myocardial revascularization			0.048
Yes	26	4 (15.3)	
No	339	19 (5.6)	
Angina pectoris			0.183
None	35	5 (14.3)	
Stable	181	11 (6.1)	
Unstable	113	7 (5.3)	
NYHA functional class			0.042
I	8	0 (0)	
II	53	1 (1.9)	
III	138	5 (3.6)	
IV	165	17 (10.2)	
Acute myocardial infarction			0.174
None	298	19 (6.4)	
Subendocardial	9	2 (22.2)	
Transmural	23	2 (8.7)	
Preoperative embolism			0.461
No	318	21 (6.6)	
Yes	18	2 (11.1)	
LV ejection fraction			0.010
>0.20	195	7 (3.6)	
<0.21	152	16 (10.5)	
Preop ventricular tachycardia[c]			0.000
Yes	12	6 (50)	
No	353	17 (4.8)	
Predominant symptoms			0.000
Angina alone	158	4 (2.5)	
Angina + CHF	132	10 (7.6)	
CHF alone	29	3 (10.3)	
Multiple	17	6 (35.3)	
Coronary artery disease			0.207
One-vessel	19	0 (0)	
Two-vessel	56	7 (11.1)	
Three-vessel	246	16 (6.5)	
Left main disease			0.167
Yes	41	5 (12.2)	
No	285	18 (6.3)	
Number of occluded coronary arteries			0.281
None	40	9 (0)	
One	189	15 (7.9)	
Two	81	7 (8.6)	
Three	7	1 (12.5)	
Thrombus in the aneurysm			0.025
Yes	61	8 (13.1)	
No	275	15 (5.5)	
Right coronary endarterectomy			0.101
Yes	10	2 (20)	
No	319	21 (6.6)	
Location of aneurysm			0.538
Anteroseptal	321	21 (6.5)	
Posterior	15	2 (13.3)	
Pathology of aneurysm			0.662
Acute	11	1 (9.0)	
Chronic	325	21 (6.4)	
Method of repair			0.954
Conventional	281	19 (6.8)	
Inverted T closure	17	1 (5.9)	
Endocardial patch	38	2 (7.9)	
Aortic cross-clamping:			0.015
≤50 min	221	8 (3.6)	
>50 min	143	15 (10.5)	
Postop low output syndrome			0.000
Yes	137	20 (14.6)	
No	225	3 (1.3)	

[a] Numbers in parentheses are percentages. [b] The *p* values were calculated by univariate analysis. [c] Not entered into stepwise logistic regression analysis (no cases operated on since 1984).

CHF = congestive heart failure; LV = left ventricular; NYHA = New York Heart Association.

Table 2A. Low Cardiac Output Syndrome: Results of Stepwise Logistic Regression Analysis

Variable	χ^2	*p* Value
Left ventricular ejection fraction <0.21	28,201	0.000
Previous myocardial revascularization	13,994	0.000
NYHA functional class IV	12,406	0.000
Age > 60 years	6,242	0.012

NYHA = New York Heart Association.

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1992;53:22-9

Table 3A. Operative Mortality: Stepwise Logistic Regression Analysis[a]

Variable	χ^2	p Value
Left ventricular ejection fraction <0.21	5,280	0.022
Age > 60 years	3,712	0.054
Previous myocardial revascularization	3,374	0.064
Lack of angina pectoris	3,374	0.066
NYHA functional class IV	3,053	0.081

[a] $p < 0.1$ is considered significant.

NYHA = New York Heart Association.

Table 4A. Late Mortality: Stepwise Logistic Regression Analysis

Variable	χ^2	p Value
Left ventricular ejection fraction <0.20	8,207	0.004
Left main coronary artery stenosis	8,022	0.005

DISCUSSION

DR WILLIAM S. STONEY, JR (Nashville, TN): I congratulate Dr Komeda and his associates on a fine presentation, and also for their excellent results. They have correctly placed emphasis on ventricular aneurysm repair rather than simple excision, and attribute a reduced overall mortality in high-risk patients to this type of repair. I would agree with this conclusion, with the reservation that some part of the reduced mortality could be attributed to better myocardial protection and other advances and improvements in the conduct of cardiac surgical procedures.

I would like to make a point about ejection fraction. The ejection fraction is the standard gauge of operative risk for most cardiac operations. In patients with very large ventricular aneurysms, the ejection fraction will be quite low, suggesting high risk, but if the nonaneurysmal portion of the ventricle contracts well, the operative risk actually can be quite reasonable. Therefore, we, like others, have tried to evaluate risk by looking at the function of the nonaneurysmal portion of the left ventricle.

We divided our patients into three groups: those patients with good function of the nonaneurysmal portion of the left ventricle, those with very poor function of the nonaneurysmal left ventricle with one akinetic segment, and those in between with impaired function. Those patients with really good contractility had a low hospital mortality; those with some impairment had a higher operative mortality; and those with an akinetic segment and very poor function had a high operative mortality and a high late mortality. At the present time, we do not recommend aneurysm repair on this third group of patients. When the nonaneurysmal portion of the ventricle contracts well, you can expect the patient to do well and not to have a high operative risk, even if the overall ejection fraction is low.

Finally, I would like to ask one question. Pericardial patches have been used for many years in the right ventricle. Dr Komeda, do you think there is a risk for late aneurysmal formation or other problems with pericardial patches in the left ventricle?

DR LAWRENCE I. BONCHEK (Lancaster, PA): This has been another very insightful report from the Toronto group under Tirone David's leadership, and it prompted me to review our much more recent experience to assess the importance of patch repair of left ventricular aneurysms.

Beginning in 1983 in Lancaster, we had experience with 89 patients if we exclude those, as Komeda and associates did, with ventricular septal defects, as well as those who had mapping, endocardial resection, and cryoablation. If we added back those 20 patients, it would bring another death into this series. Eighty-six of the patients had associated coronary bypass. There were two mitral valve replacements.

There was one death in 89 patients. We define inotropic support very stringently. Any patient who leaves the operating room with inotropic support is counted as having received inotropic support. We do not use a 12-hour or 24-hour criterion. Thirty-five percent (31 patients) received inotropic support, and 5.6% of patients had a new intraaortic balloon pump inserted postoperatively.

We believe that myocardial protection is very important, and we generally try to provide optimal myocardial protection, which to us means not clamping the aorta. In 84 of the 89 patients we did not clamp the aorta while operating on the aneurysm, and in 13 patients we did not clamp the aorta at all, even while doing the coronary bypass grafts. Only 6 patients received blood cardioplegia, usually because of substantial aortic regurgitation. We closed the aneurysm directly in 85 patients. We occasionally will just plicate even a large aneurysm if the echocardiogram raises no concern about thrombus and there is some aortic regurgitation, and also we can thereby avoid any concern about air. We did use patch closure in only 4 patients.

So the results of this experience, to us, suggested that myocardial protection is crucial, and that patch closure has a role to play, but it is by no means necessary in a large number of patients. The point I wish to make is that a discipline as complex as cardiac surgery advances on many fronts, and results of operation are constantly improving. I would caution against concluding that a particular method of repair of left ventricular aneurysms is the reason for improved results and is necessary or even suitable for all patients.

I have two specific questions. Your review was a retrospective one. In which patients now do you select a nonconventional closure of the aneurysm? And second, how did you calculate ejection fraction? As Dr Stoney pointed out, the asymmetry imposed by an aneurysm invalidates the Dodge formula that is used to calculate a three-dimensional ejection fraction from the two-dimensional angiogram.

DR L. HENRY EDMUNDS, JR (Philadelphia, PA): I also compliment Dr Komeda and associates for a nice series. Dr Ed Savage and his colleagues at the University of Pennsylvania studied the effect of aneurysmal plication on left ventricular mechanics. To do this they used a sheep apical aneurysm model and a new technique of ventricular imaging called two-dimensional sonomicrometry imaging, which was developed by Dr Mark Ratcliffe and his colleagues.

In this technique, nine sonomicrometry crystals are placed on the epicardial surface in the midsagittal plane, and endocardial crystals are placed beneath six of the epicardial crystals. Using

this technique, the epicardial and endocardial contours of the ventricle are continuously imaged throughout the cardiac cycle. Assuming an axis of symmetry, which does not change after plication, and using Janz equations, regional wall stresses and strains in various sections of the myocardial wall can be calculated. Dr Savage found that plication does not change hemodynamics; neither cardiac output nor various chamber pressures change significantly.

However, the change in geometry fails to decrease anterior wall stress but does decrease posterior wall stress. It increases end-systolic elastance, which is a measure of contractility, but after plication this increase is due to a loss of diastolic compliance. There is no net increase in the force of contractility.

Using this and related technology, we believe that it will be possible to develop superior operations for left ventricular aneurysm. Possibilities include the Dor operation, or endocardial patch, and operations involving skeletal muscle cardiomyoplasty.

DR DENTON A. COOLEY (Houston, TX): I too enjoyed this excellent paper from Toronto. We first reported repair of postinfarction ventricular aneurysm with cardiopulmonary bypass 30 years ago. At that time, we adopted the same concept of excision and lateral repair that was used to repair sacciform aortic aneurysms. It seems that our minds were closed to changes in technique from that point forward, however, until just recently. After some 2,500 cases of ventricular aneurysm, we modified the technique and now repair the aneurysm internally by using a patch graft.

We call this technique endoaneurysmorrhaphy, a term that Dr Rudolph Matas coined more than a century ago. No external or epicardial foreign bodies are applied in this repair. We are careful not to interfere with the left anterior descending coronary artery or the interventricular groove. We have found almost routinely that we can revascularize that essential artery with an internal mammary artery. Several surgeons, including Dor, Jatene, Komeda and associates, and ourselves, now use similar techniques. The drawings in this report by Komeda and associates reveal closure of the anterior descending artery by the repair, particularly the inverted T repair. I believe this to be improper. We should try to preserve the epicardium and, especially, the left anterior descending coronary artery at all costs.

During the past 2 years, we have used left ventricular endoaneurysmorrhaphy in 91 patients. Seven patients have died. A number of these patients were sent to us for cardiac transplantation. I must confess that we attempted this technique even though some of these patients had rather diffuse myocardiopathy rather than a localized aneurysm. Functional results, however, have been exceptionally good when compared with results obtained by former techniques.

Recently one of my colleagues, Dr Zvonimir Krajcer, reviewed our cases to determine whether postoperative ventricular function was better in patients who underwent endoaneurysmorrhaphy than in patients who underwent conventional repair. Dr Krajcer determined that the ejection fraction averaged 0.05 better, which was not particularly striking. The ejection fraction was better, however, in a comparable series of more advanced cases done by conventional repair.

In 1 patient, for example, nuclear ventriculograms using technetium revealed a striking improvement in ejection fraction (more than a twofold increase) in diastole and systole after left ventricular endoaneurysmorrhaphy.

The technique can be adapted easily to repair lesions resulting from acute infarction, ventricular septal defect, and even cardiac rupture.

DR C. E. ANAGNOSTOPOULOS (Stony Brook, NY): I echo the first two discussants', Dr Stoney's and Dr Bonchek's, general concerns, and in an effort to quantify the mystery of ejection fraction as a predictor in left ventricular aneurysms, I would like to remind Komeda and associates of a study by Kapelanski, Al-Sadir, and others that was published in *Circulation* just about the time their studies started. That study showed that regardless of overall ejection fraction, results, particularly in emergency aneurysms, were more related to the baseline, nonaneurysmal ejection fraction even if it was down to 0.30.

My question is, did they or could they analyze this baseline nonaneurysmal ejection fraction in the survivors versus the nonsurvivors in the high-risk group, and is it possible that this, rather than improved preservation or cardioplegia technique, is the reason for the excellent results?

DR DAVID: I would like to thank all the discussants. Dr Stoney and other discussants mentioned the problems in using global ejection fraction in patients with left ventricular aneurysm to assess operative risk, and we agree entirely. Segmental wall motion analysis, particularly of the basoconstrictor muscles, is more important than global ejection fraction in these patients. Unfortunately, our study was a retrospective one, and the preoperative data were collected from a computer databank where only global ejection fraction was recorded. Dr Komeda has promised us to review all preoperative angiograms to assess the relationships between preoperative segmental wall motion, global ejection fraction, and operative outcome.

Although we have used autologous pericardium to repair postinfarction ventricular septal defect and acute left ventricular aneurysm in numerous patients without a single case of patch aneurysm, we prefer glutaraldehyde-preserved bovine pericardium because it is readily available and easier to handle. We use Dacron graft in patients with chronic left ventricular aneurysms.

I have to congratulate Dr Bonchek for his outstanding results. I do not believe that the technique of myocardial protection you used is responsible for your results. I also close the ventriculotomy primarily in patients with small aneurysms. We use the endocardial patch technique in patients with large ventricular aneurysms and in those with impaired left ventricular function. In these patients, we believe that a repair that aims at restoration of left ventricular geometry will have a favorable effect on the surgical outcome. We have studied left ventricular function by pressure–volume relationships in a small number of patients before and after operation and found better systolic function in patients who had repair using these newer operative techniques.

We agree with Dr Edmunds' remarks. Those types of experiments will allow us to determine the best method of repair.

Dr Cooley, I am not certain that the left anterior descending artery has to be saved in all patients with ventricular aneurysm. If the proximal part of the interventricular septum is not scarred and the anterior descending coronary artery is occluded at its origin, it may be important to bypass its proximal third or the first septal perforator. In other cases, the anterior descending artery may be the principal source of collaterals to occluded marginal branches of the right coronary artery, and ligation of the anterior descending without grafting the marginal branches may result in right ventricular infarction. In most patients, however, ligation of the distal half of this artery does not seem to cause any harm.