SURGERY FOR ACQUIRED HEART DISEASE

EARLY OUTCOME OF MITRAL VALVE RECONSTRUCTION IN PATIENTS WITH END-STAGE CARDIOMYOPATHY

Uncontrollable severe mitral regurgitation is a frequent complication of end-stage cardiomyopathy, significantly contributing to heart failure in these patients, and predicts a poor survival. Although elimination of mitral valve regurgitation could be most beneficial in this group, corrective mitral valve surgery has not been routinely undertaken in these very ill patients because of the presumed prohibitive operative mortality. We studied the early outcome of mitral valve reconstruction in 16 consecutive patients with cardiomyopathy and severe, refractory mitral regurgitation operated on between June 1993 and April 1994. There were 11 men and five women, aged 44 to 78 years (64 ± 8 years) with left ventricular ejection fractions of 9% to 25% ($16\% \pm 5\%$). Preoperatively all patients were in New York Heart Association class IV, had severe mitral regurgitation (graded 0 to 4+ according to color flow Doppler transesophageal echocardiography) and two were listed for transplantation. Operatively, a flexible annuloplasty ring was implanted in all patients. Four patients also had single coronary bypass grafting for incidental coronary disease. In four patients the operation was performed through a right thoracotomy because of prior coronary bypass grafting, and four patients also underwent tricuspid valve reconstruction for severe tricuspid regurgitation. No patient required support with an intraaortic balloon pump. There were no operative or hospital deaths and mean hospital stay was 10 days. There were three late deaths at 2, 6, and 7 months after mitral valve reconstruction, and the 1-year actuarial survival has been 75%. At a mean follow-up of 8 months, all remaining patients are in New York Heart Association class I or II, with a mean postoperative ejection fraction of $25\% \pm 10\%$. There have been no hospitalizations for congestive heart failure, and a decrease in medications required has been noted. For patients with cardiomyopathy and severe mitral regurgitation, mitral valve reconstruction as opposed to replacement can be accomplished with low operative and early mortality. Although longer term follow-up is mandatory, mitral valve reconstruction may allow new strategies for patients with end-stage cardiomyopathy and severe mitral regurgitation, yielding improvement in symptomatic status and survival. (J THORAC CARDIOVASC SURG 1995;109:676-83)

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Read at the Twentieth Annual Meeting of The Western Thoracic Surgical Association, Olympic Valley, Calif., June 22-25, 1994.

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0022-5223/95 \$3.00 + 0 12/6/62477

Mitral regurgitation is a significant complication of end-stage cardiomyopathy, thought to be due to dilation of the mitral annular-ventricular apparatus^{1,2} with altered ventricular geometry³ or due to ischemic papillary muscle dysfunction.² Mitral regurgitation leads to a cycle of more volume overload of the already dilated left ventricle (LV),⁴ with progressive annular dilation, worsened mitral regurgitation, and congestive heart failure. Finally,

uncontrollable severe mitral regurgitation, refractory to medical therapy, is a late complication of cardiomyopathy and predicts a poor survival.⁵ Although cardiac transplantation has encouraging results for these patients with end-stage cardiomyopathy, transplantation is hindered by donor organ shortage and its limited applicability to older patients and patients with concurrent disease. As an alternative, surgical correction of mitral valve regurgitation could be most beneficial in this group. However, mitral valve replacement has not been routinely contemplated because of the presumed prohibitive operative mortality in these very ill patients.⁶⁻⁸ Mitral valve replacement is associated with a postoperative loss of LV systolic function^{9,10} because of interruption of annular–chordal–papillary muscle continuity.^{11,12} This loss of ventricular function, poorly afforded in patients with cardiomyopathy, may be responsible for the high operative mortality and poor late survival¹³⁻¹⁵ in patients with LV dysfunction undergoing mitral valve replacement. Conversely, reconstruction of the mitral valve with preservation of annular–chordal–papillary muscle continuity¹⁶ results in maintenance of LV systolic function, lower LV volumes, and less end-systolic wall stress^{17,18} and has a lower mortality than mitral valve replacement.^{19,20} Hypothetically, worsening heart failure in patients with dilated cardiomyopathy, resulting from annular dilation and mitral regurgitation, could be stabilized or improved by correction of mitral regurgitation, and preserving mitral annulus–chordal integrity by mitral reconstruction could yield acceptable operative and postoperative mortality. This present study was designed to assess the feasibility of mitral valve reconstruction for mitral regurgitation caused by dilated cardiomyopathy and to monitor the early outcome of mitral valve reconstruction in terms of survival, impact on heart failure, and LV function.

Methods

Between June 1993 and April 1994, 16 consecutive, nonrandomized patients at the University of Michigan Medical Center, with end-stage dilated cardiomyopathy and refractory severe mitral regurgitation, were studied prospectively. All patients had New York Heart Association (NYHA) class III or IV congestive heart failure, despite receiving maximized medical regimens, and had severe LV systolic dysfunction defined as an ejection fraction of less than 25% on LV angiography or radionuclide angiography. Patients were excluded from this study if they had evidence of primary mitral valvular disease, if significant mitral regurgitation preceded cardiomyopathy, if surgical intervention included revascularization of via-

ble myocardium, or if they had significant aortic insufficiency.

Echocardiographic imaging. All preoperative and postoperative echocardiographic imaging was performed with standard transthoracic windows with commercially available equipment, including Acuson 128XP/10 (Acuson, Inc., Mountain View, Calif.) and Hewlett-Packard Sonos 1000 (Hewlett-Packard Company, Waltham, Mass.) echocardiographs. Intraoperative echocardiography was performed with a Hewlett-Packard Sonos 1500 echocardiograph and biplane transesophageal probe. All images were recorded on standard VHS videotape. Quantitative analysis was performed by a single experienced echocardiographer blinded at the time of analysis to clinical data. Mitral regurgitation was assessed by color flow Doppler echocardiography; severity was graded as mild, moderate, or severe.²¹ Quantitative echocardiographic and Doppler measurements were performed off-line on a digital workstation (Freeland/Prism Imaging, Broomfield, Colo.). Measurements of LV, mitral annular, and outflow tract diameters were made from two-dimensional echocardiographic images in parasternal long-axis views. LV volumes and ejection fraction were calculated by means of Simpson's method with two apical views.^{22,23} Stroke volume was calculated as the difference between the diastolic and systolic volumes and ejection fraction as the ratio of stroke volume to end-diastolic volume. Flow was calculated from an echocardiographically determined inflow or outflow diameter (*d*), pulsed Doppler-derived velocity time integral (VTI) at that location, and heart rate (HR): Flow (cm^3/min) = $\pi (d/2)^2 \times \text{VTI (cm)} \times \text{HR (min}^{-1})$.²¹

Forward cardiac output was determined from the LV outflow tract diameter measured in the parasternal long-axis view in mid-systole, and the pulsed Doppler velocity time integral was measured from the apical five-chamber view. Mitral inflow was calculated from the annular diameter measured at the site of leaflet insertion in the parasternal view in mid-diastole, and the velocity time integral was measured from the apical four-chamber view. Averages of at least five cardiac cycles were used for all measurements of diameter and velocity time integral; ten cardiac cycles were averaged in the setting of atrial fibrillation. Heart rate was determined from an average of cardiac cycle lengths (R-R intervals) for the measured Doppler envelopes. Regurgitant volume was calculated as the difference between mitral inflow and forward cardiac output, and the regurgitant fraction was calculated as the ratio of the regurgitant volume to mitral inflow volume.²⁴ Intraoperative and postoperative mitral gradients were calculated by continuous wave Doppler echocardiography.

Operation. Mitral valve reconstruction was performed via a median sternotomy with standard cardiopulmonary bypass (CPB) and hypothermic, blood cardioplegic arrest in 12 patients. Mitral valve reconstruction was performed via a right thoracotomy with CPB and cold fibrillatory arrest in four patients who had had a previous cardiac operation (prior coronary bypass grafting). All operations were performed with institutional approval and informed consent.

Follow-up. All patients were contacted by telephone and personal office visit for follow-up. A standardized

questionnaire was used for symptomatic evaluation, in which physical activity was quantitated by self-report and compared with preoperative functional status. The patients' personal physicians were also contacted in follow-up. Echocardiographic studies were generally performed at the University of Michigan. In the case of echocardiograms performed elsewhere, original videotapes were sent for quantitative analysis at the University of Michigan. All data are presented as mean $\pm$ standard deviation. Comparison between echocardiographic and angiographic ejection fraction was made by means of a Pearson correlation. Comparisons between preoperative and postoperative NYHA class and LV size, volume, ejection fraction, regurgitant fraction, and forward cardiac output were made by means of paired Student's *t* tests. Actuarial survival was performed by the Kaplan-Meier method. Differences were considered significant at a 95% confidence limit, $p < 0.05$ (two-tailed).

Results

Sixteen patients (11 men and 5 women) were included in the study. Patient ages ranged from 44 to 78 years (mean 65 ± 11 years). Twelve patients had nonischemic dilated cardiomyopathy. Four had end-stage ischemic cardiomyopathy, without ongoing ischemia, as defined by negative dobutamine echocardiographic functional testing and negative positron emission tomography scanning for viability. Preoperatively, 14 patients had NYHA class IV congestive heart failure and two had class III failure. The mean duration of documented cardiomyopathy or symptomatic congestive heart failure was 6.6 ± 6.1 years (range 1 to 16 years). In the 6 months before the operation these patients had one to nine hospital admissions for congestive heart failure. Four patients had been previously intubated for pulmonary edema related to congestive heart failure. Two patients were listed for heart transplantation and five others were involved in the preliminary workup for heart transplantation. All patients were receiving maximized medical therapy for congestive heart failure, including digoxin, diuretics, and afterload reducers. Afterload reduction was accomplished with angiotensin-converting enzyme inhibitors in all patients, with either additional nitroglycerin or hydralazine in two patients each.

Preoperative ejection fraction from left ventriculography ($n = 14$) or radionuclide angiography ($n = 2$) ranged from 9% to 25% (mean $16\% \pm 5\%$). Preoperative echocardiography was performed on all patients. There was good correlation between echocardiographically and angiographically determined ejection fractions ($R = 0.85$, $p = 0.01$). On coronary angiography, four patients with nonischemic dilated cardiomyopathy were found to have

incidental single-vessel coronary artery disease. All patients with ischemic cardiomyopathy had significant coronary artery disease, with prior coronary artery bypassing, but no ongoing ischemia on functional testing. One patient with ischemic cardiomyopathy had a history of recurrent ventricular tachycardia. One patient with nonischemic cardiomyopathy had hypereosinophilia syndrome and was receiving interferon therapy. This patient was believed not to be a candidate for transplantation.

At operation all patients underwent mitral reconstruction via remodeling ring annuloplasty, with implantation of a flexible remodeling ring (Duran ring, Medtronic, Inc., Minneapolis, Minn., or Physio ring, Baxter, Inc., Irvine, Calif.). No patient required a complex repair (i.e., leaflet resection, leaflet/chordal turnover) at the time of the operation, inasmuch as all patients were believed to have only annular dilation and/or papillary muscle dysfunction. The average cardiopulmonary bypass time was 149 ± 24 minutes (range 93 to 198 minutes), with an aortic crossclamp time of 92 ± 11 minutes (range 81 to 123 minutes). No patients with irreparable defects were entered in this study. Single saphenous vein coronary artery bypass grafts were placed in four patients with incidental coronary artery disease. Additional procedures at the time of the operation included De Vega tricuspid annuloplasty in four patients and placement of epicardial implantable cardioverter defibrillator patches in one patient.

All patients survived the mitral reconstruction procedure, despite their poor preoperative ventricular function. All patients were weaned from CPB with a phosphodiesterase inhibitor, amrinone (Sanofi-Winthrop, Newark, N.J.) and norepinephrine. No patient required intraaortic balloon pumping or mechanical support. Intraoperative transesophageal echocardiography after annuloplasty and the discontinuation of CPB revealed no mitral regurgitation in 12 patients and trivial to mild regurgitation in four. The mean transmitral gradient identified by intraoperative transesophageal echocardiography was 3 ± 1 mm Hg (range 2 to 6 mm Hg). After the mitral reconstruction, three patients required longer than 24 hours of mechanical ventilator support but only one patient required longer than 48 hours. One late superficial wound infection developed. No operative or hospital deaths occurred, and the mean duration of hospitalization after the operation was 10 ± 4 days (range 5 to 17 days).

Follow-up was available for all patients. The

Table I. Mean LV size, volume, and flow before and after mitral annuloplasty

	Preop.	Postop.	Δ^*	% $\Delta^\dagger$	<i>p</i> Value
LVIDD (mm)	72 $\pm$ 9	70 $\pm$ 8	-2 $\pm$ 5	-3 $\pm$ 7	0.29
End-diastolic volume (ml)	335 $\pm$ 107	307 $\pm$ 103	-28 $\pm$ 33	-8 $\pm$ 9	0.06
End-systolic volume (ml)	277 $\pm$ 101	237 $\pm$ 98	-40 $\pm$ 39	-15 $\pm$ 14	0.03
Stroke volume (ml)	58 $\pm$ 13	70 $\pm$ 21	+12 $\pm$ 10	+18 $\pm$ 14	0.02
Ejection fraction (%)	18 $\pm$ 5	24 $\pm$ 10	+6 $\pm$ 6	+31 $\pm$ 24	0.03
Mitral inflow (L/min)	12.4 $\pm$ 5.3	5.4 $\pm$ 0.5	-6.9 $\pm$ 5.0	-49 $\pm$ 22	0.02
Forward cardiac output (L/min)	3.2 $\pm$ 1.0	4.7 $\pm$ 0.9	+1.5 $\pm$ 1.0	+52 $\pm$ 38	0.01
Regurgitant volume (L/min)	9.2 $\pm$ 5.4	0.8 $\pm$ 0.6	-8.4 $\pm$ 5.4	-88.6 $\pm$ 10.1	0.01
Regurgitant fraction (%)	70 $\pm$ 14	15 $\pm$ 11	-55 $\pm$ 15	-79 $\pm$ 15	<0.001

Matched preoperative and 4- to 6-month postoperative echocardiographic measurements of LV function and Doppler-derived flow and volume (mean $\pm$ standard deviation). The NYHA class for this group fell significantly from 3.9 $\pm$ 0.4 to 1.8 $\pm$ 0.5 ($p < 0.001$). LVIDD, Left ventricular internal diameter in diastole.

* Δ , Change from preoperative study.

$\dagger$ % Δ , Percent change from preoperative study.

duration of follow-up in these patients has been 1 to 13 months, with a 1-year actuarial survival of 75%. At a mean follow-up of 8 months, all remaining patients are in NYHA class I or II, with a mean postoperative ejection fraction of 25%, significantly better than before the operation. In the 6 months before the operation these patients had one to nine hospital admissions for congestive heart failure. Postoperatively, there have been no hospitalizations for congestive heart failure. All patients have continued to receive medical therapy for congestive heart failure, consisting of digoxin, diuretics, and afterload-reducing agents. However, drug dosages tended to be lower in the postoperative period. In no case was a patient receiving a higher dose of any medication for heart failure in the postoperative period than in the preoperative regimen. Finally, the diuretic dose required has been reduced by more than twofold. All patients have remained in their preoperative rhythm, 11 in normal sinus rhythm and five in atrial fibrillation.

All patients reported subjective improvement in their functional status after the operation, citing activities routinely performed for which exertional dyspnea was previously prohibitive. The NYHA failure class significantly decreased for every patient individually and from a mean of 3.9 $\pm$ 0.2 to 1.8 $\pm$ 0.4 for the group.

Follow-up echocardiography at 1 week and 4 to 6 months was available for all patients. At 1 week 13 patients had no mitral regurgitation and three had mild regurgitation. The mean transmitral gradient on follow-up at 1 week was 3 $\pm$ 1 mm Hg (range 1 to 5 mm Hg). Matched preoperative and 4- to 6-month postoperative measurements of LV size

and Doppler-derived flow and volume are summarized in Table I. All patients had a marked reduction in regurgitant volume and regurgitant fraction and significantly improved LV ejection fraction, end-diastolic volume, and end-systolic volume.

Three late deaths have occurred in this group after mitral valve reconstruction. One late death occurred related to complications of cholecystectomy, 7 months after mitral reconstruction in a patient who was inadvertently removed from the majority of the heart failure medicines. The second late death occurred from continued ventricular failure 6 months after mitral valve reconstruction, despite no mitral regurgitation noted on numerous follow-up echocardiographic studies. The third late death occurred 2 months after the operation and was a witnessed sudden death, presumed to be from a ventricular arrhythmia in a patient who was doing well.

Discussion

Mitral regurgitation is a poor prognostic sign for patients with end-stage cardiomyopathy. In one study of 91 patients with dilated cardiomyopathy, those patients with mitral regurgitation (by two-dimensional color Doppler echocardiography) had a significantly decreased survival at 2 years of follow-up versus those patients without mitral regurgitation (20% versus 60%).⁵ This deleterious effect was most influential in patients with severe mitral regurgitation but was even evident in patients with mild mitral regurgitation. Additionally, in a recent study, long-term survival and prognostic factors for dilated cardiomyopathy were studied in 111 patients. Actuarial predictors of survival revealed that

mortality was related to ejection fraction less than 30%, episodes of heart failure, and increased LV end-diastolic volume.²⁵ In a further study of 130 patients awaiting transplantation, 1-year survival was only 46%. Significant independent predictors of death for these patients with idiopathic cardiomyopathy were low forward stroke volumes, an ejection fraction less than 25%, and mitral regurgitation.²⁶ All of these negative prognostic factors were present in our study population of cardiomyopathy with secondary mitral regurgitation and would predict a poor 1-year survival.

Surgical correction of mitral regurgitation and accompanying volume overload has been shown to be warranted for patients with mitral regurgitation and could be most beneficial in this group of patients with cardiomyopathy and secondary mitral regurgitation. In a study of 249 patients with mitral regurgitation who had angiographic assessment of LV function before the operation, survival of surgically treated patients was significantly better than that of medically treated patients. Those patients with mild to moderate impairment of ejection fraction were most responsible for the improved survival in surgically treated patients, which occurred despite greater functional and hemodynamic impairment in surgically treated patients before the operation.¹⁴

Unfortunately, many surgical studies have also denoted that greater functional and hemodynamic impairment and reduced ejection fraction equate with poor operative and in-hospital survival in patients operated on for mitral regurgitation by mitral valve replacement.^{8, 13-15, 27} The high operative mortality and poor outcome have inhibited the routine undertaking of surgical correction for mitral regurgitation in patients with extremely poor LV function. These findings are in accordance with those of many experimental laboratory studies demonstrating that chordal transection results in significant reductions in rest and exercise ejection fraction, caused by a significant increase in end-systolic circumferential wall stress and geometric distortion.⁹⁻¹² One study notes that loss of annular-chordal-papillary continuity immediately decreases LV systolic function by 30%, alters the twist mechanics of myocardial contraction, and results in a globular, inefficient geometry of the heart.⁹⁻¹²

However, many studies have shown that mitral reconstruction, as opposed to replacement, results in improved rest and exercise ejection indexes, primarily the result of a marked reduction in end-systolic stress and maintenance of a more ellipsoidal

chamber geometry.^{28, 29} Many clinical studies have compared the results of mitral valve reconstruction with those of mitral valve replacement and have concluded that the preservation of the annular-chordal-papillary muscle continuity results in maintenance of LV function and geometry, leading to better patient outcome.^{17-20, 30}

On the basis of the poor natural history of this population with end-stage cardiomyopathy, mitral regurgitation, and heart failure resistant to medical therapy, for whom heart transplantation has previously been the only therapy available, mitral reconstruction was believed to be a new and viable option. Technically, mitral reconstruction via annuloplasty effectively corrected mitral regurgitation in these patients, confirmed both on intraoperative transesophageal echocardiography and on follow-up transthoracic studies. Furthermore, despite high operative risks, there were no operative or in-hospital deaths and the actuarial survival was 75% at 1 year.

Not only was survival improved but functional status has changed remarkably in these patients. Importantly, no patient has had a worsening clinical status during follow-up. Additionally, echocardiographic analyses revealed a strong trend toward decreased LV volume after the operation, with significant increases in ejection fraction and forward cardiac output. Improvement in echocardiographic parameters of ventricular function were not necessarily anticipated, because LV function has been shown to decrease after mitral valve replacement, presumably as a result of loss of chordal attachments and normal systolic ventricular shortening. Second, loss of the low-impedance left atrial chamber associated with correction of mitral regurgitation might be expected to lead to further systolic impairment in the setting of preexisting ventricular dysfunction. However, LV function improved after the operation in these patients. This improvement implies that a component of ventricular decompensation observed before the operation was due to volume overload associated with severe mitral regurgitation superimposed on dilated cardiomyopathy. Postulated mechanisms for the improvement in ventricular function include stabilization of the mitral anulus and LV unloading that has induced a more favorable ventricular geometry. A recent study denotes that remodeling of LV geometry may be rapid and complete in patients with resultant regurgitant fractions of less than 30% after correction of mitral regurgitation.³¹ Our patients had a mean residual regurgitant fraction of only 16% after the operation, which

could favor rapid LV remodeling. Furthermore, there is evidence from recent trials with a left ventricular assist device that unloading the dilated cardiomyopathic LV encourages ventricular recovery.³² A second mechanism may involve correction of mitral regurgitation, decreasing the volume overload of the LV and establishing a better position on the Frank-Starling curve.

Mitral valve reconstruction for mitral regurgitation complicating dilated cardiomyopathy has been previously reported in only a single patient,³³ who had significant ischemic disease. Each patient included in our present study had dilated cardiomyopathy with advanced symptoms of congestive heart failure despite aggressive medical therapy, a poor prognostic sign for patients with end-stage disease.³⁴ Mitral reconstruction performed in this cohort resulted in significant improvement in survival and symptomatic status on early follow-up and was associated with favorable changes in ventricular size and function. Although longer term follow-up with greater numbers of patients and perhaps case-matched controls are necessary, this pilot study demonstrates the feasibility of operative repair for correction of mitral regurgitation, with an excellent technical outcome and a low operative mortality. Other aspects of follow-up for these patients, including assessment of maximum oxygen consumption during exercise and LV remodeling, are being undertaken at present. Finally, the effect on need for transplantation, the ability to perform transplantation in these patients, and the cost-effectiveness versus transplantation remain to be answered. However, we are encouraged by these early results and believe that mitral reconstruction offers a new strategy for patients with end-stage cardiomyopathy.

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Discussion

Dr. Vaughn A. Starnes (*Los Angeles, Calif.*). It would not have been intuitively obvious to me that these patients would have benefited from this operation. To summarize some of the demographics, the group comprised an older population, mean age 65 years, with severe heart failure (class IV) who had been symptomatic for more than 6 years. During the prior 6 months these patients had been hospitalized six to nine times. The patients had dilated cardiomyopathy with an average ejection fraction of 16% and 4+ mitral regurgitation. The authors repaired the mitral valve to get rid of the mitral regurgitation and "improve" the ejection fraction. Their results are surprisingly good.

The actuarial survival is 75%. To put this in perspective, reports from the literature indicate a 2-year survival of only 20%. Clearly this operation has extended the survival time of this group of patients. Transplantation offers a better 1-year survival, around 80% to 90%, but donor shortage is a real problem.

This operation decreased the systolic volume by 15%, increased the ejection fraction from 16% to 25%, and increased the cardiac output on the average by 35%. These are truly surprising results.

Dr. Bolling, how do you account for the improvement in LV function after the repair of a regurgitant lesion in a patient with severe cardiomyopathy? Is it due to remodeling or is it due to some other factor yet undetermined? Do you have any echocardiographic data to support the idea of remodeling?

Dr. Bolling. We do have preliminary echocardiographic data on remodeling, and I think that is occurring. We thought that the ejection fractions would not change and that if we could get the majority of the patients through the operation perhaps their systolic function would not improve, but their forward flow would. Despite that hypothesis, the ejection fractions improved.

Regarding long-term remodeling, Dr. Frasier reported on a patient who was supported for 550 days by the HeartMate device (Thermo Cardiosystems, Inc., Woburn, Mass.) and died of a stroke when the HeartMate left ventricular assist device was turned off. The ejection fraction had increased from about 12% up to about 45%, and the patient was sustained by his own heart. Whether this is long-term change attributable to LV remodeling and whether that will be sustained under a heavier load, we do not know.

We are interested in calcium handling of the myocyte, and we are investigating that right now.

Dr. Starnes. What are the acute hemodynamic effects of pulmonary artery wedge pressures in these patients in the immediate postoperative period?

Dr. Bolling. Left ventricular end-diastolic pressures and wedge pressures do not change acutely. These patients are far out on the Starling curve. In the immediate postoperative period, if they have a wedge on a left ventricular end-diastolic pressure of 25 to 35 mm Hg, they need to be at that pressure. However, their pulmonary pressures drop and, as in the transplant population, they continue to drop. In some of these patients the pulmonary pressures are dramatically lower at 1 year's follow-up.

Dr. Starnes. Your results certainly lead one to consider this operation as a bridge to transplantation. Seven of your patients were actually being evaluated for transplantation, and two have been accepted onto your transplant list. Are you considering such an option for some of your patients?

Dr. Bolling. These are very early results and we certainly do not want to overstate them. In fact, I would rather understate the results. However, I think for some patients this operation could be a bridge to transplantation. For some it may obviate the need for transplantation.

Dr. Joseph Cleveland (Missoula, Mont.). Could you amplify some of the details of the reconstructions, such as the exact type of ring and whether you resected any of the posterior leaflet? Did you simply put in a ring? What was the average size of the ring?

Dr. Bolling. We have extensive experience with mitral valve reconstruction at our institution; however, these patients were picked because they had no primary mitral disease. They merely had annular dilatation. None of the patients had chordal transfers or leaflet resection. All received a flexible ring, either the Medtronic Duran or the Baxter Physio ring. We undersized the trigone-to-trigone distance, believing that we probably would not induce stenosis because these patients had normal mitral subvalvular apparatuses. Our postoperative data bear out that belief. At 6 months' follow-up for the majority of these patients, they do not have mitral stenosis. We undersized the rings perhaps by one size. The average size ring that we inserted in these patients was a 29 mm ring.

Dr. D. Craig Miller (Stanford, Calif.). Let me go farther than Dr. Starnes in terms of surprise. I am flabbergasted. These results are absolutely phenomenal. Why? Your radionuclide end-diastolic volumes were about the same pre- and post-load. Do you have any M-mode echocardiographic data to tell us what happened over time to end-diastolic dimension, end-systolic dimension, and LV shape?

Dr. Bolling. We do have that information, but it was not presented here. However, with further follow-up, most of the patients now have end-diastolic volumes that clearly are smaller. We are just as surprised as everyone else, but I think our series has to stand by itself. Again, I would like to understate the series, not overstate it.

Dr. Miller. They are indeed smaller ventricles, but are they also more elliptical ventricles after the operation?

Dr. Bolling. Definitely. In fact, some of our echocardiologists, who were assessing the echocardiograms in a blind fashion, had to check the name of the patient for fear that they were reading the wrong echocardiogram. In one of our patients the ejection fraction increased from 15% to 44%.

Dr. Miller. Before we accept the increase in stroke volume and ejection fraction, what was the afterload during both studies, as estimated by systolic, mean, or diastolic blood pressure? I trust it was the same, because a discrepancy could certainly confound your data.

Dr. Bolling. All those are the same afterload reductions. In fact, the patients were studied prospectively, so the echocardiograms were done on an outpatient basis by the same echocardiologist with the same afterload and the same mean arterial blood pressure.

Dr. Edward Verrier (Seattle, Wash.). There has to be something unique about this group of patients. The most striking point is the 6.6-year period of therapy for chronic heart failure with some stability. According to the numbers that Dr. Starnes alluded to, these patients should have all been dead. Is part of the take-home message that this longer period of stability is necessary? We are not going to get the same results if we start applying this technique in anybody with an ejection fraction of 12%.

Dr. Bolling. I totally agree. If we applied this technique to every patient with an ejection fraction of 12%, we would have a horrendous operative mortality. However, these patients had the cardiologists totally frustrated because they were stable and then suddenly became unstable. These were the patients who could not be kept out of the hospital. One patient was admitted and discharged nine times in 6 months. We postulated that this might be a volume overload problem. Perhaps these are patients in whom failure accelerates rapidly, and we are catching them right at the end.

Dr. John Benfield (Sacramento, Calif.). One of the things that we have learned in clinical trials of patients with lung cancer is that the patients we enter into protocols and studies are not necessarily the same as those whom we do not enter into the protocols. Therefore, one must be very careful in interpreting results as representing a step forward unless one knows the full denominator of patients from whom we choose the ones we select for operation. Therefore, my question is this: Improved survival compared with what? Were these patients truly representative? How were they selected?

Dr. Bolling. No patient was turned down who met these criteria, but I think you are right. The patients who never came to us are probably a more interesting group. That brings up the need to do case-matched controls. All we had were the historical controls from the three large series that predicted terrible 1- and 2-year survivals for this group of patients with end-stage dilated cardiomyopathy and severe uncontrollable mitral regurgitation. This pilot study was more for operative feasibility: Could we operate on these patients? Could they survive the operation? I agree that case-matched controls are the next logical step to be taken in this group.