SURGERY FOR ACQUIRED HEART DISEASE

EARLY RESULTS WITH PARTIAL LEFT VENTRICULECTOMY

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Objective: We sought to determine the role of partial left ventriculectomy in patients with dilated cardiomyopathy. **Methods:** Since May 1996 we have performed partial left ventriculectomy in 53 patients, primarily (94%) in heart transplant candidates. The mean age of the patients was 53 years (range 17 to 72 years); 60% were in class IV and 40% in class III. Preoperatively, 51 patients were thought to have idiopathic dilated cardiomyopathy, one familial cardiomyopathy, and one valvular cardiomyopathy. As our experience accrued we increased the extent of left ventriculectomy and more complex mitral valve repairs. For two patients mitral valve replacement was performed. For 51 patients the anterior and posterior mitral valve leaflets were approximated (Alfieri repair); 47 patients also had ring posterior annuloplasty. In 27 patients (51%) one or both papillary muscles were divided, additional left ventricular wall was resected, and the papillary muscle heads were reimplanted. **Results:** Echocardiography showed a significant decrease in left ventricular dimensions after resection (8.3 cm to 5.8 cm), reduction in mitral regurgitation (2.8+ to 0), and increase in forward ejection fraction (15.7% to 32.7%). Cardiac index did not increase significantly (2.2 to 2.4 L/min per square meter). Eight patients (15%) required a perioperative left ventricular assist device; one died and was the only perioperative mortality (1.9%). At 11 months, actuarial survival was 87% and freedom from relisting for transplantation was 72%. **Conclusions:** Improved selection criteria are necessary to avoid early failures, and much more follow-up and analyses of data are mandatory. However, the operation may become a biologic bridge, or even alternative, to transplantation. (J Thorac Cardiovasc Surg 1997;114:755-65)

Terminal heart disease is prevalent and expected to steadily increase.¹ Human heart transplantation is severely limited by donor availability, which is

never expected to meet the overwhelming demand.¹⁻³ The search for surgical alternatives to cardiac allografts includes mechanical devices,⁴⁻⁶ xenografts,^{7,8} cardiomyoplasty,^{9,10} and high-risk "conventional" surgery.^{11,12} The increased operative mortality of high-risk conventional surgery in patients with heart failure should be weighed against the mortality of transplantation: approximately 20% die while waiting for a donor heart, 20% die during the first year after transplantation, and mortality is 5% per year thereafter.^{13,14} In addition, the cost of transplantation and the significant morbidity of immunosuppression and other medications have to be considered when deciding between high-risk conventional surgery and cardiac transplantation.

An operation was developed by Batista and co-workers^{15,16} for patients with end-stage dilated car-

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Fig. 1. Partial left ventriculectomy (*left*) with the lateral wall and lateral branch of the circumflex coronary artery excised. After closure (*right*) the LAD artery at the apex (point A) wraps around the reconstructed apex. The wall between the papillary muscles was resected, and during closure the papillary muscles were sutured together.

diomyopathy of various etiologies. The concept of the operation is simple: returning the enlarged heart to a normal diameter will reduce left ventricular (LV) wall tension (the mechanism is related to the law of Laplace). To reduce the heart diameter, large segments of LV wall are resected (hence, Batista's terminology *partial left ventriculectomy*). The heart is then reconstructed to decrease the LV diameter. Clinical results from Brazil are difficult to interpret, but the perioperative mortality is approximately 22% and the 2-year mortality approximately 45%. Most survivors are in improved clinical condition.¹⁶

We sought to study the operation prospectively; to determine whether it could be made safer with more sophisticated perioperative care and support, to assess the operation as a "biologic bridge" or even an alternative to transplantation, and to better clarify patient selection. This report details our early results and operative techniques and how they differ from those of Batista.

Patients and methods

Between May 1996 and April 1, 1997, 53 patients (mean age 53 years, range 17 to 72; 72% male) underwent partial left ventriculectomy, with mitral valve repair in 51 patients and mitral valve replacement in two patients. Our initial experience included primarily patients who were thought

to have idiopathic dilated cardiomyopathy and who were candidates for heart transplantation. After further experience we included three patients not eligible for transplantation and patients with other cardiomyopathy etiologies (familial and valvular).

Candidate selection was based on an LV end-diastolic diameter of 7 cm or more on at least one recent echocardiographic study. Patients were not turned down for surgery on the basis of any reduced level of LV or right ventricular ejection fraction, pulmonary hypertension, mitral or tricuspid regurgitation, or incidental coronary artery disease. Among patients with the appropriate etiology and LV end-diastolic diameter, the most common reason not to offer the operation was that the patient was "too well" and did not meet standard heart transplant listing criteria.¹⁷ All patients were reviewed and approved by the heart failure/transplant team, consisting of cardiologists, surgeons, nurses, and social workers. All patients gave informed consent. Those eligible for transplantation were offered the option of waiting for that operation; those not eligible for transplantation were offered medical therapy.

The 50 (94%) transplant candidates included 22 (44%) who had status I disease according to the criteria of the United Network for Organ Sharing (UNOS). Twenty-one (42%) were dependent on inotropic agents, two were also supported by an intraaortic balloon pump (IABP), and one patient had been supported by the HeartMate implantable left ventricular assist device (LVAD) (Thermo Cardiosystems, Inc., Woburn, Mass.) for 88 days but had a device infection. The other 28 transplant candidates had

Fig. 2. To excise more LV wall, we transected one or both (*left*) papillary muscles and removed additional LV wall (*dotted line*). The resected heads of the papillary muscles were then resuspended with multiple, pledget-supported, full-thickness sutures (*right*).

UNOS status II disease. Excluding the patient with the LVAD, 31 patients (60%) were in New York Heart Association (NYHA) functional class IV; the others were in class III. The patients in class III had had a mean of two admissions for congestive heart failure before the operation. The condition of five of the 22 patients with status I disease was steadily deteriorating (two requiring IABP support). These patients would alternatively have received implantable LVADs as a bridge to transplantation if partial left ventriculectomy had not been available. As further evidence of the end-stage condition of these patients, the mean ($\pm$ standard deviation) peak volume of oxygen use was 10 ± 3.9 ml/kg per minute.

Surgical and echocardiographic techniques. The operation evolved from our earliest procedures using the methods of Batista (heart beating, single mitral valve repair stitch¹⁶) to more complex valve repairs and cardioplegic arrest during ventricular resection. Critical echocardiographic information obtained (both preoperative and intraoperative) included LV end-diastolic diameter and internal interpapillary muscle distance to guide the extent of resection. Intraoperative transesophageal echocardiography was performed in all patients. Standard esophageal and transgastric views were obtained with the use of a HP Sonos 5.0/3.7 MHz probe with an HP Sonos 1500/2500 echocardiography machine (Hewlett-Packard Co., Palo Alto, Calif.). LV end-systolic and diastolic dimensions and volumes were calculated by the method of discs from the apical four-chamber view. The interpapillary distance, used to determine the expected size of the LV after the operation, was obtained from the short-axis view by measuring the distance between the insertion of the two papillary muscle heads into the endocardial surface. The severity of valvular regurgitation was determined by standard color flow Doppler ultrasonography. Forward stroke volume, derived with the use of the velocity time integral from pulsed wave Doppler ultrasonography of the LV outflow tract, is divided by the LV end-diastolic volume to obtain the forward ejection fraction. After the operation, these variables were remea-

sured, and the mitral valve gradient was calculated from the mitral inflow pressure half-time and confirmed by planimetry in the transgastric short axis. Wall motion information was collected but not used to guide the operation.

The operation was performed with the use of cardiopulmonary bypass with antegrade and retrograde cold blood cardioplegia for myocardial protection. The left atrium was opened in the interatrial groove and the Cosgrove-Edwards annuloplasty system (Baxter Healthcare Corp., Edwards Div., Santa Ana, Calif.) was placed.¹⁸ A small ring *undersizes* the dilated anulus, and now we routinely use a No. 26 Cosgrove-Edwards ring (Baxter) to eliminate the central jet of mitral regurgitation.

One patient unexpectedly had dense scar in the left anterior descending (LAD) and right coronary artery distributions. A limited anterior ventriculectomy was performed and she received an LVAD when she could not be weaned from cardiopulmonary bypass. Although she did not undergo typical partial left ventriculectomy, she is included in the series in the category "intention-to-treat failure."

For the typical partial left ventriculectomy resection the incision began approximately 2 cm lateral to the LAD artery and 3 cm proximal to the apex. The incision extended laterally along the base of the anterior papillary muscle of the mitral valve (Fig. 1). The apex of the incision was midway between the papillary muscles and 2 cm from the mitral valve anulus. Typically this incision divided a large branch of the circumflex coronary artery, which was oversewn. At the apex, the incision then was extended 3 cm parallel to the LAD artery and excised the LV apex (Fig. 1). The incision was then extended along the base of the posterior mitral valve papillary muscles and connected to the previous incision, thereby removing a large wedge of LV between the papillary muscles.

The goal of ventriculectomy was to return the LV end-diastolic diameter to near-normal. The LV end-diastolic diameter after resection was predicted by a simple formula: for every 3.14 cm (i.e., π) of LV muscle

Fig. 3. To decrease mitral regurgitation and avoid prolapse from redundant mitral valve chordae, we sewed the free edge of the anterior and posterior mitral valve leaflets (*top left*) together with a 4-0 suture (Alfieri repair). After this suture is tied, the mitral valve has a double orifice (*bottom left*). Echocardiography revealed low gradients across the mitral valve, a “figure-of-eight” appearance, and good mitral valve area (*right*).

circumference resected, the LV end-diastolic diameter was decreased by 1 cm. Therefore, for a typical patient with an LV end-diastolic diameter of 8 cm and 6 cm of LV muscle between the papillary muscles, after partial left ventriculectomy between the papillary muscles, the new LV end-diastolic diameter would be 6 cm. If the LV end-diastolic diameter was very large and the distance between the papillary muscles small, then we judged that we needed to remove more LV muscle and papillary muscle(s) to return the LV end-diastolic diameter to near-normal. In 27 patients (51%), on the basis of echocardiographic measurements and observation of the remaining ventricle, we resected one or both papillary muscles. This allowed further resection of anterolateral or posterior LV wall (Fig. 2). The remaining head of the resected papillary muscles (where the chordae arise from the muscle) were then resuspended with multiple full-thickness pledget-supported 3-0 polypropylene sutures (Fig. 2).

Reconstruction of the ventriculotomy approximated the papillary muscles or resuspended heads of the papillary muscles. Theoretically, this change in orientation of the papillary muscles may create redundant length to the chordae and, therefore, mitral regurgitation as a result of mitral valve prolapse. Batista avoids this problem by approximating the midportion of the free edge of the anterior and posterior leaflets (Alfieri repair) with a single suture.^{16,19} The reconstructed mitral valve has a double orifice and looks like a figure-of-eight on postoperative echocardiograms (Fig. 3). Using a small annuloplasty ring also relieves tension on this suture.

We closed the ventriculotomy in three layers, with strips of soft felt or bovine pericardium to help disperse tension over the long suture line. With the lateral wall removed and large portions of the remaining LV removed in 51% of patients with papillary muscle resuspension, there was considerable tension on the ventriculotomy closure. After closure, the distal LAD wrapped around the newly reconstructed LV apex (Fig. 1), the papillary muscle bodies were approximated to each other, and the LV dimensions and volumes were decreased (Fig. 4). After both papillary muscles have been resected, the remaining LV consists of septum and a portion of the anterior and posterior walls (Fig. 4).

Coronary arteries were bypassed in four patients with coronary artery lesions of more than 50%. The tricuspid valve was repaired if 2+ tricuspid regurgitation or greater was present. A De Vega tricuspid valve repair was used in 30 patients (57%) and a Cosgrove-Edwards ring was placed in one patient with 4+ tricuspid regurgitation. The right ventricle was never plicated as Batista has reported.¹⁶ One patient with moderate aortic insufficiency (2+) had aortic valve repair. One patient with combined moderate aortic stenosis and insufficiency (bicuspid valve) underwent aortic valve replacement. Mitral valve replacement was performed in two patients in whom mitral valve repair was not possible. One patient had rheumatic mitral valve disease and 2+ mitral regurgitation after initial attempted repair. The other had an unusual eccentric jet and had residual 2+ mitral regurgitation after attempted repair. Both underwent mitral valve replacement via left atriotomy. For patients who needed the implantable

Fig. 4. After resection and reconstruction, the LV diameter was reduced. In the most extreme cases, after resection of both papillary muscles, the remaining LV was composed primarily of septum and a portion of the anterior and posterior walls (*left*). Echocardiography demonstrates that the right ventricle (RV) wraps around most of the LV in these extreme cases (*right*).

LVAD after partial left ventriculectomy, the technique for LVAD insertion was similar to that described in our previous report.²⁰

Statistical methods. Data have been presented as mean and standard deviation. Comparisons from one time point to another were done with the use of a paired *t* test. Survival and freedom from relisting for transplantation were calculated by means of the Kaplan-Meier method.

Results

The average weight of the resected specimen was 96 gm (range 30 to 290 gm). Four of our earliest patients had only the Alfieri mitral valve repair (no mitral valve ring). These four patients had a reduction in mitral regurgitation initially, but at follow-up regurgitation had increased. Mean pressure gradients across the mitral valve after the Alfieri repair were uniformly low (mean 2.85 ± 1.43 mm Hg), and the mean mitral valve area (adding both orifices of the mitral valve) was 3.9 ± 1.06 cm. Papillary muscles were resected and resuspended in 27 patients (51%); both muscles were resected in nine patients (17%), the posterior papillary muscle only in 15 patients (28%), and the anterior papillary muscle only in three patients (6%).

Thirty patients were weaned from cardiopulmonary bypass with the aid of inotropic drugs. Two patients had preoperative IABP support. IABPs were placed in four patients (8%) in an attempt to

wean them from cardiopulmonary bypass. In all four patients, the IABP was inadequate. Two immediately received an LVAD and two required a heparin-coated extracorporeal membrane oxygenator; both required LVADs later. One patient received an IABP after chest closure because of rapid hemodynamic deterioration, which in retrospect was precipitated by temporary right main stem intubation. This IABP was successfully removed the next morning.

Intraoperative transesophageal echocardiography showed a significant decrease in LV end-diastolic dimension, mitral regurgitation, and an increase in LV ejection fraction (Table I). The stroke volume did not significantly change. After the operation, typically the contractility of the anterior wall and septum improved.

Before the operation, 21 patients required inotropic drugs, and inotropic agents were added in another nine patients because of hypotension and low cardiac output before the induction of anesthesia; therefore 30 patients (57%) were receiving inotropic agents before bypass. The cardiac index did not significantly increase after the operation, but the left atrial pressure, systemic blood pressure, and pulmonary artery pressures significantly decreased (Table II). Postoperative elevation of cardiac enzymes occurred in all patients, with a mean creatine kinase level of 1793 ± 908 units the day of the operation

Table I. Echocardiographic changes in patients undergoing partial left ventriculectomy

Variable	Preop: Mean (SD)	Intraop pre: mean (SD)	Intraop post: mean (SD)	After 1 week: mean (SD)
LVEDD (cm)	8.1 (1.0)	8.3 (1.1)	5.8 (0.7)	6.4 (0.6)†
LVEDV (cm)	319 (114)	251 (86)	117 (41)*	142.9 (40)†
Forward LVEF (%)	13.7 (6.2)	15.7 (7.3)	32.7 (11)*	23.4 (7.9)†
Mitral regurgitation (0-4+)	2.9 (1.0)	2.8 (1.1)	0.02 (0.1)*	0.2 (0.5)†
Tricuspid regurgitation (0-4+)	1.6 (1.0)	1.3 (0.9)	0.14 (0.4)*	1.1 (1.0)†
Stroke volume (ml)	41.2 (15)‡	35.8 (17)‡	36.1 (9.7)‡	37.1 (10)‡

SD, Standard deviation; LVEDD, left ventricular end-diastolic dimension; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction.

* $p < 0.001$ intraop pre to intraop post.

† $p < 0.001$ preop to 1 week.

‡Changes in stroke volume, p not significant.

and a total creatine kinase MB level of 220 ± 72 units (range 21 to 300 units).

Eight patients (15%) required an LVAD in the perioperative period. Of these eight patients, one showed steady improvement in cardiac function and underwent successful LVAD removal after 86 days. Four successfully underwent transplantation and are well. Two are alive and well as outpatients awaiting transplantation, and one died. The patient who died had a complicated course with LVAD insertion that also required insertion of a right ventricular assist device. Eventually, she died of *Candida* sepsis and multiple organ failure. This is the only perioperative mortality (1.9%). Two other patients could not be weaned from inotropic drugs and were relisted for cardiac transplantation. One of these patients subsequently underwent transplantation, and one was converted to an outpatient dobutamine regimen.

Postoperative morbidity was common. Two patients had transient renal failure necessitating dialysis; an additional two patients required dialysis after LVAD insertion. Two reoperations for bleeding were necessary (3.8%). Two patients required reintubation for respiratory failure and subsequently required tracheostomies. One patient had a major stroke in the distribution of a previous preoperative stroke, and one patient had a new transient ischemic attack. One patient had a saddle embolus to the aortic bifurcation on postoperative day 12, arising from new left atrial thrombus. In addition, of two patients who later underwent transplantation, one had mitral valve replacement with a mechanical valve and one patient had a left atrial thrombus 5 months after the operation, despite routine postoperative warfarin administration.

For the patients not having an LVAD or transplantation, the mean hospital length of stay was 13 days (range 6 to 31 days). The average hospital

charge for these patients was $\$54,165 \pm \$28,624$ (range $\$30,395$ to $\$187,317$). PredischARGE echocardiography showed a significant persistent decrease in LV end-diastolic diameter, decrease in mitral regurgitation, and increase in LV ejection fraction (Table I). We believe the changes from the intraoperative studies after partial left ventriculectomy are different from the 1 week studies because of different loading conditions. After surgery, medical therapy for heart failure was resumed for all patients and included angiotensin-converting enzyme inhibitors, diuretics, and digoxin. Amiodarone was initiated because of sudden death in the Brazilian experience. Warfarin was instituted after the operation because of the potential for LV thrombus along the suture line.

Follow-up is complete in all patients and ranges from 1 to 11 months (mean 5 months). After discharge 16 patients were rehospitalized, and three were relisted for transplantation. One of these patients eventually received an LVAD and died of multiple organ failure 87 days after partial left ventriculectomy and 20 days after LVAD insertion. Two patients died of presumed cardiac causes 100 days and 209 days after partial left ventriculectomy. One patient had a peak volume of oxygen use of 24 ml/kg per minute at 3-month follow-up and had returned to functional class I. However, he died suddenly and autopsy did not reveal the cause of death. The second patient had undergone LVAD removal for infection and then partial left ventriculectomy. Seven months later he was admitted to another hospital and died rapidly of heart failure. One patient with valvular cardiomyopathy underwent transplantation 5 months after partial left ventriculectomy. This patient died 8 days after transplantation of right heart failure caused by pulmonary hypertension resulting from chronic mitral

Table II. Intraoperative hemodynamic findings in patients undergoing partial left ventriculectomy

Hemodynamic variable	Preoperative: mean (SD)	Postoperative: mean (SD)	p Value
Cardiac index (L/min/m ²)	2.2 (0.7)	2.4 (0.5)	0.25
Heart rate (beats/min)	92 (17)	102 (16)	0.001
Blood pressure systolic (mm Hg)	100 (15)	87 (12)	<0.001
Blood pressure mean (mm Hg)	73 (11)	63 (9)	<0.001
Blood pressure diastolic (mm Hg)	59 (14)	52 (8)	0.004
Left atrial pressure (mm Hg)	24 (9)	14 (5)	<0.001
Pulmonary artery systolic (mm Hg)	49 (14)	38 (8)	<0.001
Pulmonary artery mean (mm Hg)	36 (11)	30 (6)	0.001
Pulmonary artery diastolic (mm Hg)	26 (9)	24 (5)	0.16

valve disease. Of the discharged patients not undergoing transplantation, 35% (13 patients) are in functional class I, 32% (12 patients) are in functional class II, and 27% (10 patients) are in functional class III.

In summary, among the 53 patients there was one (1.9%) operative mortality in a patient who required LVAD support. There were four other deaths: one late after LVAD insertion, one after transplantation, and two of cardiac causes after partial left ventriculectomy. Actuarial survival at 11 months was 87%, and actuarial freedom from relisting for transplantation was 72%. Of the discharged patients who did not undergo transplantation, 67% were in functional class I or II. Of all 53 patients, 25 (47%) were in class I or II after partial left ventriculectomy. Most failures occurred early after the operation (Fig. 5).

Discussion

The concept of reducing LV volume to improve function of the remaining LV muscle is not new. LV aneurysmectomy reduces LV cavity size to treat heart failure.²¹⁻²³ The mechanism of action is thought to relate to improvement in wall tension and myocardial efficiency resulting from effects engendered by the law of Laplace. Partial left ventriculectomy differs from aneurysmectomy in that the LV scar is not removed; instead, viable but over-distended LV muscle is resected.^{15, 16} Theoretically, this procedure rapidly reverses the detrimental remodeling associated with dilated cardiomyopathy and heart failure. Early experience, however, leaves many unanswered questions: Who are the best candidates? How much muscle can or should be removed? What is the role of mitral valve repair in the clinical outcome? What are the early hemodynamic and physiologic effects? Are the early changes temporary or sustained? How does the underlying

myocardial disease and myocyte function correlate with early and late clinical outcomes? What are the molecular biodynamic effects of the operation on remaining muscle? What is clear from the experience in Brazil is that for *some* patients in NYHA class IV the operation returns the patient to class I, and this improvement may be sustained for years. With this knowledge and the overwhelming need for alternatives to cardiac transplantation, we prospectively studied this operation. Our clinical experience, length of follow-up, and analyses do not yet allow us to answer these questions.

We decided to perform partial left ventriculectomy only in those without extensive myocardial scar or fibrosis. We reasoned that constructing a smaller heart that remained extensively scarred would produce minimal benefit. Attesting to the relative lack of specificity of coronary angiography, we found unexpected scar in two patients: both required LVAD insertion. One other patient failed to improve and continued to wait status I until transplantation: pathologic examination of the heart demonstrated extensive myocardial fibrosis. Another patient had unexpected myocarditis and is awaiting transplantation. In future analyses we will compare histologic characteristics with clinical outcomes, which may permit correlation of preoperative studies with myocardial histology and allow us to more reliably predict outcome.

We sought to return the LV end-diastolic dimension to *near* normal. Preoperative echocardiographic measurements allowed us to predict what the diameter of the ventricle would be after resection. If papillary muscles had to be resected, the posterior wall was usually thinner than the anterior wall. We therefore preferentially resected the posterior papillary muscle and posterior wall. Preoperative echocardiographic studies typically suggested that the lateral LV wall contraction was the most

Fig. 5. At 11 months, actuarial survival was 87%, and actuarial freedom from relisting for transplantation was 72%. Most failures necessitating relisting for transplantation occurred early.

vigorous. After resection, the septum and anterior wall showed the best contractility, and the area immediately adjacent to the ventriculotomy was relatively akinetic.

By performing the Alfieri mitral valve repair, Batista significantly decreased mitral regurgitation.¹⁶ Bolling²⁴ and Bach²⁵ and their colleagues have shown that patients with severe ventricular dysfunction and severe mitral regurgitation improve clinically after mitral valve repair alone. Because the mitral regurgitation potentially contributed to the symptoms in our patients, and because we wanted to avoid progressive LV dilatation caused by volume overload from residual mitral regurgitation, we began to add posterior annuloplasty for all our patients, even if the preoperative mitral regurgitation was only 1 to 2+. In addition, our early studies confirmed that with normal pliable mitral valve leaflets, the Alfieri repair produced very low pressure gradients and an acceptable mitral valve orifice. The contribution of mitral valve repair to overall clinical improvement remains to be determined. Many of our patients whose condition clinically improved after partial left ventriculectomy and mitral valve replacement had only 2+ mitral regurgitation before the operation; therefore it is unlikely that mitral valve replacement played the primary role in the clinical improvement for these patients. However, for patients with 4+ mitral regurgitation, mitral valve repair may have had a synergistic, or even predominant, effect in clinical improvement.

Our early follow-up indicates that clinical improvement will be sustained for months in most patients. In general, the failures have been early after the procedure, during the initial hospitalization. After discharge, clinical deterioration so that patients were relisted for transplantation was uncommon (three patients). At this time, however, we do not have enough follow-up to predict how long these changes will persist. Clinical follow-up from Batista indicates that at least some patients will have persistent improvement for up to 4 years after the operations.¹⁶

From our early experience we draw the following initial conclusions. Many patients with idiopathic dilated cardiomyopathy and mitral regurgitation will show significant clinical benefit from mitral valve repair with partial left ventriculectomy. Failures that occur early after the operation may be from myocardial fibrosis, other myocyte inflammatory processes (myocarditis), and ischemic cardiomyopathy. It is our impression that a favorable clinical response to inotropic agents and a dobutamine echocardiographic study showing improvement in myocardial contractility may help identify patients who will respond well to surgery. However, this ventriculectomy *improves* heart function but does not return it to normal. Patients with extremely poor LV function (e.g., 5% forward LV ejection fraction and stroke volume less than 20 ml) may not realize enough improvement to live through the perioperative period and then return to functional class I or

II. Until we improve our predictors of outcome, we will continue with our strategy to offer ventriculectomy primarily to transplant candidates. These early conclusions are tentative and can only be verified with further clinical experience, longer follow-up, and further analyses of prospectively gathered data. However, our overall clinical impression is that the operation may serve as a relatively inexpensive "biologic" bridge to transplantation for some UNOS status I patients, and, at best, it may benefit other patients for many years and be an alternative to transplantation or medical therapy for patients who are not candidates for transplantation.

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Discussion

Dr. D. Craig Miller (Stanford, Calif.). All of us have had the pleasure of hearing the second rigorous scientific presentation of the clinical results of the Batista procedure, with the São Paulo group presenting the first proper report last fall at the meeting of the American Heart Association. This is a much better forum than *20/20* or *Nova* or the *New York Times* or even the *National Enquirer* to assess these results.

I congratulate Dr. McCarthy and his coworkers for their efforts to elucidate whether partial left ventriculectomy works, and if so, why it works. I also compliment them for their cautious initial approach, which retained fall-back safety nets, namely resorting to the use of LVADs and transplantation. This work is a fine example of a careful, prospective investigation, albeit lacking a suitable control group, which brings me to my first and most important question.

Our medical colleagues treating patients who have heart failure have achieved astoundingly good results in possibly similar patients using angiotensin-converting enzyme inhibitors and beta blockers, if the patient can initially be tuned. This "tuning" step is a big caveat.

Indeed the use of carvedilol in patients with dilated cardiomyopathy now goes by the moniker "medical Batista procedure." Dr. McCarthy, were your candidates for partial left ventriculectomy judged too sick by Drs. Young and Starling for such treatment or had this approach already failed? If not, could they have possibly been randomized for control purposes? If so, why did you not elect to randomize them? On the basis of what you have learned so far, do you have any future plans for a randomized trial?

Second, the 87% survival and 72% free-from-transplant relisting figures at 11 months are major improvements over the São Paulo results. In what respect do you attribute this to the prophylactic use of amiodarone with or without an automatic implantable cardioverter-defibrillator?

Third, focusing on technique, I believe you currently strive at all costs to avoid reimplanting the anterolateral papillary muscle. Could you amplify what problems this caused you early on? Also, given the fact that at least four patients, 8% of your series, sustained left atrial thrombosis, what are your thoughts now concerning anticoagulation?

Fourth, and perhaps most telling, are you seeing any signs of recurrent LV dilatation in patients with 9 months' follow-up or more? Partial left ventriculectomy may well potentially be a biologic bridge to transplantation, but it could turn out to be a very short bridge.

I will not explain in detail why ejection fraction must increase after partial left ventriculectomy as a result of simple changes in geometry and ventricular mechanics. Simply put, if the laws of physics are the same in the Northern Hemisphere as they are in the Southern Hemisphere, the increase in LV ejection fraction is not mysterious or mythical; it is *predictable*.

Dr. Fernando A. Lucchese (*Porto Alegre, Brazil*). Please allow me, as a Brazilian and old friend of Dr. Batista, to comment about the creator of this intriguing idea. This partial left ventriculectomy is just one of the many incredible ideas he has shared with us, as were stentless aortic valves, surgical treatment of atrial fibrillation, and reduction of pulmonary hypertension in Eisenmenger's syndrome.

I would like to mention briefly our experience with 42 cases of Batista's procedure. The main difference with Dr. McCarthy's cases is that most of our patients were not candidates for transplantation, for a variety of reasons. They were already in the hospital, receiving maximum medical therapy. Hospital mortality was 16% and 2-year actuarial survival was around 40%. Before the operation, four survivors had high pulmonary resistance as a contraindication for transplantation, varying from 6 to 8 Wood units. From 6 to 12 months after the operation, pulmonary resistance dropped to 2 to 3 Wood units. One of these patients underwent successful transplantation 18 months after Batista's procedure. As the first center to do this operation after Batista, we think there is a place for it. Further studies are obviously needed.

Dr. Akira T. Kawaguchi (*Kanagawa, Japan*). It was in the AATS meeting 3 years ago in Boston that there was a discussion on cardiac volume reduction of the left atrium. When we reported an increased incidence of atrial fibril-

lation after the modified maze procedure in patients with a larger left atrium, with Dr. Cox as the moderator, Dr. Batista showed that simple reduction in left atrial dimension ablated atrial fibrillation as well. He went on to apply the same mass/radius concept on the LV 3 months later. We started collaborating and studied 70 patients by means of pressure-volume loop analysis immediately before and after the partial left ventriculectomy.

In brief, reduction in LV volume improves all the indices of systolic function, but it is at the cost of diastolic dysfunction, which was not apparent in the previous experience with the left atrium because the atrium works as a conduit in diastole rather than as a contractile chamber. On the average, total energy consumption of the LV decreased more than the stroke work, improving myocardial energy efficiency.

This observation, however, does not seem to fully account for the improvements we see after ventriculectomy. We think that our pressure-volume observation was made too early after the operation because 20 other patients undergoing angiographic study 9 days after the same procedure had an increased stroke volume at unchanged end-diastolic pressure, indicating improved LV function. These observations made us speculate that reduction in mitral regurgitation may be mainly responsible for the hemodynamic improvements immediately after partial left ventriculectomy, allowing patients to be weaned from bypass and to recuperate. Mechanoenergetic changes as observed by pressure-volume study would improve hemodynamics or course of the underlying disease sometime later.

Dr. Gregory A. Misbach (*San Bernardino, Calif.*). The authors are to be congratulated for their careful patient selection and careful analysis of results in helping us to understand how to apply the Batista procedure. One continuing puzzle will be whether in idiopathic dilated cardiomyopathy the initiating cause of dilation will recur and dilate the hearts once again after the Batista procedure. It seems that the larger series in this country will come out of transplant centers where there are tertiary referrals and thousands of patients from whom to select, such as in The Cleveland Clinic. I think there is, however, one reason for more of us to have some familiarity with the Batista technique. Dr. Dor has presented persuasive information that argued for the broadening of application of endoventricular patch repair to postinfarction LV aneurysms, not just to areas that are dyskinetic but also to areas that are akinetic. Dr. McCarthy, although patients with diffuse fibrosis on magnetic resonance imaging scan are poor candidates for partial left ventriculectomy, do you believe those with localized fibrosis in one region of the heart might be candidates for a modified Batista technique where the worst portion of the heart that is virtually akinetic is excised, the diameter is reduced, and the resultant wall tension reduction combined with coronary grafting can benefit the residual contractile areas?

Dr. Hisayoshi Suma (*Kamakura, Japan*). My question concerns the preoperative evaluation of the LV characteristic itself, not the ventricular function. What kind of evaluation before the operation did you do to define the ventricular wall characteristic and what did you learn?

Dr. McCarthy. I would like to thank all the discussants for their points.

First for Dr. Miller, our control patients were patients otherwise going on to transplantation. These 53 patients were chosen from among literally thousands of referrals that we received after the media blitz. We chose patients who were very sick, who my cardiology colleagues thought would not respond to beta blockers. Carvedilol is currently indicated for patients with class II or III heart failure. Experience in patients with class IV heart failure is very limited. If you look at the carvedilol trial published in the *New England Journal of Medicine*, the survival in the placebo group was quite good, so the population studied was not as sick as ours. It is important that 44% of our patients were in the hospital receiving inotropic drugs, and therefore status I, and had had unsuccessful previous medical therapy.

Will this become a randomized trial? First, who is going to pay for it? It won't be a drug study or a device study, so no company will stand to benefit by paying for the study. The National Institutes of Health is not ready to fund a study at this point. Second, historically enrollment in other surgical randomized trials, such as the REMATCH LVAD trial, or cardiomyoplasty, has been slow because of very difficult inclusion/exclusion criteria. Third, we think that with more experience we may be able to compare our results with the real control population, transplant patients. We already know those results: 20% die waiting for a heart, 15% to 20% die the first year, and 4% to 5% die every year after transplantation. Finally, we are still on the learning curve regarding selection, technique, and post-operative care. If we start a trial too early it may not show the potential of this operation.

We used amiodarone in all of the patients after the operation. Amiodarone probably contributes to the low risk of sudden death that we have seen so far. Only one patient required a new defibrillator, and there has been only one sudden cardiac death, which is very different from the Brazilian experience. We started amiodarone because of what we had learned from the sudden death rate in Brazil.

We try not to resect the anterior papillary muscle

because the echocardiogram typically shows that the anterior wall and the septum do most of the work after the resection. Therefore, if possible, we simply resect the posterior papillary muscle and leave the anterior papillary muscle.

We have seen a high rate of left atrial thrombus formation, and all patients now are placed on a regimen of warfarin sodium (Coumadin). Even so, we have seen left atrial thrombus form despite therapeutic levels of warfarin. I think it reflects the low flow state in some of these patients with end-stage heart failure.

We do not have enough long-term follow-up yet to make definitive statements about late recurrence of LV dilatation, but we have not seen this yet in the patients that we have observed. At 3 months their echocardiograms look the same as at 1 week. I am certain that redilatation will occur in some patients, but as yet we have not seen that.

Dr. Misbach, if LV scar can be identified and removed, that should improve LV function. Preoperatively, we have not been able to consistently identify regional differences in scar location in dilated cardiomyopathy.

We purposely excluded patients with ischemic cardiomyopathy from our study. We already know that aneurysmectomy, or patch repair as noted by Dr. Dor, improves function in the remaining LV. The contribution of Dr. Batista was not regarding that principle for ischemic cardiomyopathy; rather it was the new idea to extend that concept to dilated cardiomyopathy.

Dr. Suma, your question addresses what is now our most important area of research: correlating the preoperative studies with the pathologic characteristics of the myocardium and then with clinical outcome. Preoperative studies included echocardiograms, stress echocardiograms, positron emission tomographic scans with fluorodeoxyglucose, and magnetic resonance imaging studies. This analysis will be difficult and will take time. No easy answer has presented itself. However, in our early experience we think that a favorable response to dobutamine echocardiogram and a positron emission tomogram/fluorodeoxyglucose scan showing little fibrosis will predict a good outcome.