Initial Results of Posterior Leaflet Extension for Severe Type IIIb Ischemic Mitral Regurgitation

Benoit de Varennes, MD; Rakesh Chaturvedi, MD; Surita Sidhu, MD; Annie V. Côté, MD; William Li Pi Shan, MD; Caroline Goyer, MD; Roupen Hatzakorzian, MD; Jean Buithieu, MD; Allan Sniderman, MD

Background—Management of severe ischemic mitral regurgitation remains difficult with disappointing early and intermediate-term surgical results of valve repair.

Methods and Results—Forty-four patients with severe (4+/11001) Carpentier type IIIb ischemic mitral regurgitation underwent mitral valve repair, with or without surgical revascularization, by posterior leaflet extension with a patch of bovine pericardium and a remodeling annuloplasty. Serial echocardiography was performed preoperatively, intraoperatively, and postoperatively to assess mitral valve competence. The postoperative functional status of patients was assessed. The average Parsonnet score was 38±13. Thirty-day mortality was 11%, and late mortality was 14%. Mean follow-up was 38 months. The actuarial freedom from moderate or severe recurrent mitral regurgitation was 90% at 2 years, whereas 90% of patients were in New York Heart Association class I at 2 years.

Conclusion—Posterior leaflet extension with annuloplasty of the mitral valve for severe type IIIb ischemic regurgitation is a safe, effective method that provides good early and intermediate-term competence of the mitral valve and therefore good functional status. (Circulation. 2009;119:2837-2843.)

Key Words: echocardiography ▪ ischemia ▪ mitral valve ▪ regurgitation ▪ surgery

Ischemic mitral regurgitation carries a very poor prognosis, especially after myocardial infarction.1–5 The conventional surgical treatment of severe mitral regurgitation in the absence of morphological abnormalities of the valve has been remodeling annuloplasty because coronary artery bypass grafting alone has led to a very high incidence of postoperative residual mitral regurgitation.6 Unfortunately, although some investigators have reported good results, particularly with aggressive undersized annuloplasty,7,8 other groups have experienced very high rates of recurrence of moderate or severe mitral regurgitation after such repairs.9–12

Animal models have made clear the multifaceted pathophysiological origin of ischemic mitral regurgitation.13,14 In brief, myocardial remodeling leads to papillary muscle displacement in relation to the plane of the annulus; this produces tethering of the chordae tendinae and impaired coaptation of the leaflets during systole. These problems are compounded by significant annular dilation. The resulting jet of mitral regurgitation is usually complex and is composed of a central and posteriorly directed jets of regurgitation (Carpentier types I and IIIb). Isolated remodeling annuloplasty principally addresses the central jet of regurgitation (type I). It is therefore not surprising that many patients continue to suffer from significant mitral regurgitation after such repairs. Consistent with this analysis are the recent observations that MV repair for ischemic mitral regurgitation fails to provide any survival advantage.15–19 This lack of benefit may be explained by the fact that up to 30% of patients develop recurrent mitral regurgitation after annuloplasty and coronary artery bypass graft surgery. The limited benefit of the conventional approach has stimulated the development and testing of new methods to more directly address the complex cause of the mitral regurgitation in these patients.

Enlarging the mitral leaflet by surgically implanting a pericardial patch represents a novel approach to increasing the coaptation of the mitral leaflets. Langer et al20 demonstrated the efficacy of the approach in correcting experimental mitral regurgitation. Unfortunately, to date, only limited data are available on its clinical application. Dobre et al21 and Rendón et al22 have each reported on 2 patients. This article reports the initial clinical results for 44 patients with severe type IIIb ischemic mitral regurgitation

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undergoing posterior leaflet extension with a patch of bovine pericardium.

**Methods**

**Patients**

Between August 2003 and August 2007, 44 patients underwent posterior mitral leaflet extension for severe type IIIb mitral regurgitation performed by a single surgeon (B.d.V.). The director of professional services at McGill University Health Center provided authorization to enter the patients’ data into a database so that the results could be collated and reviewed. Severe mitral regurgitation had been documented by preoperative transthoracic echocardiography and/or left ventriculography and confirmed by intraoperative transesophageal echocardiography under general anesthesia. Mitral regurgitation was classified as type IIIb if mitral regurgitation was present with both a central and posteriorly directed jets.

All patients who did not require concomitant coronary artery bypass graft surgery had previously undergone surgical revascularization, and the grafts were confirmed to be patent at the time of selective angiography.

**Echocardiographic Evaluation**

A comprehensive multiplane transesophageal echocardiography examination was performed after induction of anesthesia in all patients with a Vivid 7 system with a 6T-OR 3- to 7-MHz transesophageal echocardiography multiplane probe (General Electric Healthcare/Vingmed, Wauwatosa, Wis) in the operating room. Two-dimensional echocardiography and color-flow imaging were used to determine the severity, origin, and mechanism of the mitral regurgitant jet. All 44 patients had severe (4+) type IIIb mitral regurgitation as evidenced by the presence of a complex jet of regurgitation that consisted of a central (usually dominant) and posteriorly directed jets (Figures 1 and 2) with systolic flow reversal into at least 1 pulmonary vein. The posteriorly directed jets were isolated using the midesophageal midcommissural (60°) and long-axis (120°) views. The jets were found to emerge between the P3 and A2 scallops using the midcommissural view and were confirmed with the long-axis view.

Transthoracic echocardiography was performed with either a Sonos 5500 system with an S4 2.0- to 4.0-MHz Ultraband adult cardiac phased-array transducer (Philips Medical System North America, Bothell, Wash) or a Vivid 7 Dimension system with a 3S-RS phased-array transducer (General Electric Healthcare/Vingmed). The severity of mitral regurgitation was graded as described by the American Society of Echocardiography. This approach is based on the integration of the 2-dimensional and color Doppler evaluations. In brief, mild mitral regurgitation is characterized by a small central jet (<20% of left atrial area, narrow vena contracta width, and no or minimal flow convergence. Systolic flow is dominant in the pulmonary veins with A-wave–dominant mitral

![Figure 1. Preoperative transesophageal echocardiographic appearance of the central jet of mitral regurgitation.](image1)

![Figure 2. Preoperative transesophageal echocardiographic appearance of the posteriorly directed jet of mitral regurgitation.](image2)
inflow. Severe mitral regurgitation is characterized by a central jet >40% of the left atrial area, broad flow convergence, and broad vena contracta width. Systolic flow reversal is present in pulmonary veins with dominant E-wave mitral inflow. Moderate mitral regurgitation has evidence of more than mild mitral regurgitation but no criteria for severe regurgitation.

**Surgical Technique**

All patients underwent complete revascularization on the basis of the preoperative angiogram. Left internal thoracic arteries (when available) were used to bypass the left anterior descending artery; greater saphenous vein grafts were used on other coronary arteries. All operations were performed with cardiopulmonary bypass under mild hypothermia (lowest nasopharyngeal temperature, 32°C) with bivacaval cannulation. Myocardial protection was achieved with cold-blood cardioplegia diluted 4:1. Mitral valve repair was performed before any aortic and/or tricuspid procedures.

For the first 10 patients, the mitral valve was exposed via a left atriotomy incision through the interatrial groove above the right pulmonary veins. Subsequently, the superior transseptal approach to the mitral valve was used. After snaring of both cavae, the lateral wall of the atrium was opened 3 to 4 cm above the interatrial groove. This incision was then extended superiorly between the superior vena cava–right atrial junction and then from within the right atrium toward and through the fossa ovalis. The septal incision was then extended superiorly onto the dome of the left atrium for a few centimeters underneath the ascending aorta. Pledged sutures were used to retract the interatrial septum and to expose the entire mitral apparatus.

Direct operative inspection confirmed that the mitral valve was morphologically normal with no evidence of any rupture of chordae tendinae or papillary muscles. Both leaflets were to be pliable with no leaflet or annular calcifications. The posterior leaflet was then detached at its base from the middle of P2 all the way up to the posterior commissure. Annuloplasty sutures were then applied around the circumference of the mitral annulus. A bovine pericardial patch (Dura-Guard, Synovis Surgical Innovations, St. Paul, Minn) was rinsed thoroughly and cut in an oblong fashion with a height of 1 cm and a length of 3.5 to 4.5 cm. As demonstrated in Figure 3, the patch was sutured to the edge of the posterior leaflet defect and the posterior mitral annulus with a running 4-0 polytetrafluoroethylene suture (W.L. Gore and Associates, Flagstaff, Ariz).

The annuloplasty rings were true-sized. The intercommissural distance and the exact surface area of the anterior leaflet were then used to select the appropriate Physio (Edwards LifeSciences, Irvine, Calif) annuloplasty ring. The most common ring size was 28 (22 patients); sizes 24, 26, and 30 were used in 1, 14, and 7 patients, respectively. After the ring was put in place, the atriotomy incision was closed, and the patient was weaned off cardiopulmonary bypass.

The adequacy of the valve repair was assessed by intraoperative transesophageal echocardiography. Postoperative transthoracic echocardiograms were obtained at 3 months and then yearly thereafter.

The presence and severity of residual mitral regurgitation were assessed as previously described.

Patients received Coumadin for 3 months after surgery unless contraindicated by preoperative comorbidities such as bleeding disorders or gastrointestinal bleeding. Acetylsalicylic acid 80 mg QD was added for all patients. At the 3-month follow-up visit, Coumadin was discontinued if the patients were in normal sinus rhythm. Statistical analysis was done by ANOVA and paired and unpaired t tests as appropriate.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

**Results**

Forty-four patients underwent mitral valve repair for type IIIb ischemic mitral regurgitation between August 2003 and August 2007. The clinical characteristics of the patients are given in Tables 1 and 2. Twelve patients required additional valvular surgery. Of these, 9 had tricuspid repair and 3 required aortic valve replacement for aortic stenosis and tricuspid repair. This was the first cardiac surgical procedure for 38 patients; 5 patients had been operated on once before; and 1 patient had undergone surgery twice previously. Ten patients did not require aortocoronary bypass grafting. In the others, the mean number of bypass grafts was 1.6 ± 1. The mean cross-clamping time was 106 ± 36 minutes (minimum, 57 minutes; maximum, 212 minutes). Eight patients required intra-aortic balloon pump counterpulsation to be weaned off cardiopulmonary bypass.

The mean Parsonnet score of all patients was 38 ± 13 (minimum, 14; maximum, 69) for a mean predicted mortality risk of 24 ± 17%. The observed in-hospital mortality was 5 of 44 (11%). Data are available after discharge for all survivors. The average length of follow up was 38 ± 17 months. The late

<table>
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<th>Table 1. Patient Characteristics</th>
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<td>Male/female</td>
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<td>Age, y</td>
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<td>Parsonnet score</td>
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<td>Preoperative NYHA class, n (%)</td>
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<td>II</td>
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<td>III</td>
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<tr>
<td>Bypass grafts, n</td>
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<td>First surgery, n (%)</td>
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<td>Redo surgery, n (%)</td>
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<td>Second redo surgery, n (%)</td>
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<td>Elective/urgent/emergent, n (%)</td>
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Values are expressed as mean ± SD when appropriate. *Minimum to maximum.

LV indicates left ventricular ejection fraction as determined by transthoracic echocardiography.
mortality after hospital discharge was 16% (7 of 44), producing a total mortality of 27%. The causes of late death were as follows: 2 cerebrovascular accidents (14 and 18 months postoperatively), 1 pneumonia (33 months), 1 pulmonary embolus (9 months), 1 multiorgan failure (4 months), 1 aortic stenosis and heart failure (36 months), and 1 colon perforation (23 months).

Figure 4 demonstrates the functional status in the postoperative period. Although 68% were in New York Heart Association (NYHA) class III or IV preoperatively, 92% of patients were in class I at the 2-year follow-up. All patients who were in NYHA class III or IV in the preoperative period improved by at least 1 class. Overall, at the last follow-up, 2 patients did not improve (1 was in class I and 1 was in class II preoperatively), 5 patients improved by 1 NYHA class, 18 patients improved by 2 NYHA classes, and 9 patients improved by 3 classes.

Echocardiographic assessment of valve competence is illustrated in Figure 5. At 1 year, only 3 of 32 patients had developed recurrent moderate mitral regurgitation; all the others had none (n=5), trace (n=8), or mild (n=16) regurgitation. The actuarial freedom from moderate or severe recurrent mitral regurgitation was 90%, 93%, and 100% at 2, 3, and 4 years, respectively.

One of the patients who developed moderate mitral regurgitation underwent mitral valve replacement with a mechanical prosthesis for class III congestive heart failure symptoms. Despite no obvious evidence of an early postoperative ischemic event, this patient continued to demonstrate severe, progressive left ventricular dilation (left ventricular diastolic diameter, 64 mm) and developed moderate regurgitation as soon as 3 months after surgery. He was reoperated on 16 months later, and the pericardial patch was intact and very pliable with no evidence of any annuloplasty ring dehiscence despite the presence of a large central jet of regurgitation at the time of reoperation. After an initial significant improvement, the patient developed progressive heart failure despite a competent mitral prosthesis and died 41 months after the first surgery.

A second patient operated on in 2004 developed moderate and then severe regurgitation in the fourth postoperative year. Serial echocardiograms confirmed progressive remodeling of the left ventricle despite no documented new ischemic event. The preoperative left ventricular diastolic diameter was 71 mm and increased to 98 mm at the last follow-up. Thus, he has been placed on a list for heart transplantation because he now suffers from class III heart failure symptoms.

The overall impact of operation on left ventricular function and size is summarized in Table 3. The data are the average results for all survivors. Note that all subjects did not undergo examination at all time points. No significant difference in

![Post-Operative Echocardiographic Data Degree of Mitral Regurgitation](image)

![Evolution of NYHA Classification](image)

![Changes in NYHA functional status for each patient.](image)

![Echocardiographic assessment of mitral competence.](image)
left ventricular ejection fraction was found during follow-up. Similarly, no significant differences were found in left ventricular end-diastolic and end-systolic diameters during follow-up. Thus, for the survivors, left ventricular function appears to remain stable with neither improvement nor deterioration.

Discussion

Ischemic mitral regurgitation is produced by a complex combination of pathophysiological processes. On the one hand, dynamic and fixed valvular and subvalvular dysfunctions lead to displacement of the posteromedial papillary muscle in relation to the plane of the annulus. This displacement causes tethering of the chordae and therefore incomplete posterior leaflet coaptation during systole and consequently a posterior-directed jet of mitral regurgitation (type IIIb). The mitral regurgitation leads to annular and left ventricular dilatation, which begets further mitral regurgitation, which characteristically is central in relation to the valve. Remodeling from the ischemic insult also produces annular and left ventricular dilatation and further contributes to the generation of central mitral regurgitation. The net result is a complex interplay of regurgitant jets with the central (type I) jet principally resulting from the annular dilatation and the posterior-directed ones (Type IIIb) principally resulting from the posterior displacement of the posteromedial papillary muscle.

Remodeling annuloplasty, which deals with only one of these problems, annular dilatation, was the initial approach to surgical repair of mitral regurgitation. Unfortunately, the results have been, on the whole, disappointing because many patients develop recurrent moderate or severe mitral regurgitation after surgery. The wide variety of surgical approaches may explain, in part, the variable results of annuloplasty alone, but ongoing changes in left ventricular shape and volume almost certainly contribute importantly to the pathophysiology of recurrent mitral regurgitation. For example, Hung et al demonstrated that, despite achieving intraoperative competence of the mitral regurgitation in the majority of their patients, 72% developed moderate or severe recurrent mitral regurgitation in the late postoperative period. Importantly, the appearance of mitral regurgitation was associated with increases in left ventricular volume and sphericity, with the more spherical shape leading to tethering of the posterior mitral leaflet as a result of displacement of the posteromedial papillary muscle. These observations are supported and extended by those of Kuwahara et al, who demonstrated, by careful echocardiographic analysis of the anterior and posterior leaflet tethering angles, that in patients with ischemic mitral regurgitation treated by annuloplasty alone who developed recurrent regurgitation, there was worsening of the posterior leaflet tethering. The anterior leaflet tethering significantly improved and remained stable in these patients, confirming that it is the augmented and, more important, progressive posterior leaflet tethering that is related to recurrent mitral regurgitation after annuloplasty. On the other hand, coronary bypass grafting and stringent downsizing of the mitral annulus by Physio rings 2 sizes smaller than measured have had promising results with excellent competence of the mitral valve at the 2-year follow-up. This aggressive approach also provided for reverse remodeling of the left ventricle, which, presumably, is key to the long-term success of this approach.

Because myocardial dysfunction is the fundamental cause of ischemic mitral regurgitation, not surprisingly, many approaches have attempted to tackle the problem at that level. In general, however, none has yet been shown to be highly successful. Infarct plication and restraint of the infarcted wall with a mesh directly address the myocardial issues but have not yet been widely applied. The restraint technique would probably be best used in the acute period of the infarct. Repositioning of the posteromedial papillary muscle has been attempted externally and internally in animal models, and the Coapsys system is currently part of a clinical trial. Hvass et al described their clinical experience with moderately undersized annuloplasty and using a Gore-Tex tube to reapproximate the posterior and anterior papillary muscles in 10 patients with severe ischemic mitral regurgitation. The “tensing effect” was reduced and valve competence was achieved in most patients. Of interest was the reduction in the distance between the 2 papillary muscles achieved by the surgical remodeling.

Addressing leaflet tethering and its resultant reduction of coaptation surface by surgical extension of the leaflet appears to be a simple, physiologically coherent approach to correct type IIIb ischemic regurgitation. In a bovine model of ischemic mitral regurgitation, Langer et al demonstrated that posterior leaflet extension without annuloplasty corrected the regurgitation despite apical displacement of the posterior mitral leaflet edge, demonstrating that patch extension can, at least in this circumstance, compensate for the tethering. The first case report of 2 patients with echocardiographic evidence of type IIIb ischemic mitral regurgitation and tethering of the posterior leaflet successfully managed by extending the posterior leaflet height with a patch of pericardium was by Dobre et al in 2000. Shortly thereafter, a Spanish group

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Table 3. Left Ventricular Ejection Fraction and Left Ventricular Diameters

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<tr>
<th></th>
<th>Preoperative</th>
<th>At 3 mo</th>
<th>At 1 y</th>
<th>At 2 y</th>
<th>At 3 y</th>
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<tr>
<td>n</td>
<td>Mean±SD</td>
<td>n</td>
<td>Mean±SD</td>
<td>n</td>
<td>Mean±SD</td>
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<tr>
<td>LVEF</td>
<td>37 0.38±0.15</td>
<td>23 0.42±0.15</td>
<td>27 0.44±0.15</td>
<td>19 0.44±0.14</td>
<td>14 0.46±0.13</td>
</tr>
<tr>
<td>LVS, mm</td>
<td>19 41±8</td>
<td>23 41±11</td>
<td>27 38±10</td>
<td>19 41±12</td>
<td>14 39±13</td>
</tr>
<tr>
<td>LVD, mm</td>
<td>21 56±7</td>
<td>23 54±10</td>
<td>27 52±8</td>
<td>19 55±9</td>
<td>14 55±11</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; LVS, left ventricular systolic diameter; and LVD, left ventricular diastolic diameter. Values are expressed only for postoperative surviving patients.
reported 2 similar cases. In all 4 patients, the preoperative echocardiogram clearly demonstrated that the patients had complex jets of mitral regurgitation composed of centrally and posteriorly directed jets. Anterior leaflet extension with a pericardial patch has been reported in 25 patients demonstrating restricted leaflet motion. The results were acceptable, but longer-term follow-up is necessary to assess whether such a technique may compensate for the progressive posterior leaflet tethering described above.

The present approach significantly increases the coaptation surface in the region of the leaflet most affected by tethering from the posteromedial papillary muscle by extending the posterior leaflet height by \(\sim 1\) cm in the area of P3 and the medial half of P2. This also provides a safety margin of tissue in case left ventricular remodeling continues after surgery. Although the 2 patients who developed recurrent severe mitral regurgitation demonstrated progressive ventricular remodeling, their preoperative conditions and ventricular diameters did not differ from most of the patients in this series. It remains very difficult to objectively predict which patients will experience ongoing remodeling leading to recurrent regurgitation.

From a technical standpoint, we recommend the superior transapical approach, which made it much easier to perform the suture line between the patch and valve tissues. Only long-term follow-up will establish whether the long-term pliability and structural integrity of the bovine pericardial patch are comparable to other pericardial valve prostheses. Glutaraldehyde-treated autologous pericardium has been used successfully in the reconstruction of other mitral valve pathologies such as rheumatic disease and endocarditis with quite acceptable long-term results, but no data are available on the long-term results of the use of bovine pericardium in such reconstructions.

The technique described here should be easily reproducible and offers consistent positive results in the early and midterm postoperative period. Longer follow-up is essential in all these patients. Alternatives such as mitral valve replacement with total preservation of the subvalvular apparatus should be explored as part of future trials to determine whether preservation of mitral competence prevents the progressive remodeling that occurs in ischemic patients despite appropriate revascularization. Moreover, it must be appreciated that mitral regurgitation is only 1 element determining the final clinical outcome of these patients. Progressive ventricular remodeling and decapsulation continue in an important subset of these patients who will, unfortunately, not benefit from any reconstructive surgical procedure of the mitral valve.

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Disclosures
Dr de Varennes is a consultant for Edwards LifeSciences Inc, Canada. The other authors report no conflicts.

References
extension: an adjunctive repair option for ischemic mitral regurgitation? 


CLINICAL PERSPECTIVE

Severe ischemic mitral regurgitation continues to carry a very high mortality and morbidity when treated medically and remains a significant surgical challenge. The valvular problem is caused by a primary myocardial dysfunction for which revascularization is not an adequate treatment. Myocardial approaches to ischemic mitral regurgitation are not readily applicable and remain mostly experimental. Annuloplasty alone has not been shown to produce sustained benefits in many patients presumably because of continuous ventricular remodeling in the postoperative period. Posterior mitral leaflet extension with a patch in combination with remodeling annuloplasty addresses the type I and IIIb mechanisms of mitral regurgitation; in addition, the increased leaflet coaptation surface may compensate for eventual ventricular remodeling and its associated tethering of the posterior leaflet. The surgical procedure is relatively simple and reproducible and could offer longer-term mitral competence in patients suffering from severe ischemic mitral regurgitation.

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