
AORTIC AND MITRAL VALVE REPLACEMENT WITH RECONSTRUCTION OF THE INTERVALVULAR FIBROUS BODY

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Objective: The intervalvular fibrous body between the aortic and mitral valves can be damaged by infective endocarditis, degenerative calcification, or multiple previous heart valve operations, making double valve replacement difficult. We have managed this problem by approaching the aortic and mitral valves through the aortic root and the dome of the left atrium. After excising the aortic valve, the diseased fibrous body, and the mitral valve, we suture a properly tailored patch of Dacron fabric or bovine pericardium to the lateral and medial fibrous trigones and to the aortic root, reestablishing the aortic and mitral anuli. A prosthetic mitral valve is implanted and a separate patch is used to close the left atriotomy before implantation of a prosthetic aortic valve. This study was undertaken to determine the efficacy of this operation. **Methods:** Forty-three patients underwent reconstruction of the intervalvular fibrous body during aortic and mitral valve replacement because of infective endocarditis with abscess in 14 patients, extensive calcification in 9, lack of fibrous tissue because of multiple previous operations in 10, and to enlarge the aortic and mitral anuli in 10. The group comprised 18 men and 25 women with a mean age of 58 ± 12 years. Thirty-two patients had had one or more previous heart valve replacements. All patients were in New York Heart Association functional classes III and IV, and 9 patients were in shock before the operation. **Results:** Seven operative deaths occurred (16%). Early prosthetic valve endocarditis developed in two patients and necessitated reoperation. Follow-up extended from 4 to 108 months, with a mean of 38 months. No patient was lost to follow-up. Six late deaths occurred. The actuarial survival at 6 years was $56\% \pm 6\%$. A Doppler echocardiographic study revealed normal prosthetic valve function and anatomically intact anuli in all 30 long-term survivors. **Conclusions:** Reconstruction of the intervalvular fibrous body during aortic and mitral valve replacement is a satisfactory operative approach in patients with complex valve annular pathology. (J Thorac Cardiovasc Surg 1997;114:766-72)

The aortic and mitral valves are connected by a fibrous body that extends from the lateral to the medial fibrous trigones. This fibrous structure is

seldom more than 1 cm in height, and from the ventricular side it is indistinguishable from the anterior leaflet of the mitral valve until the point where the noncoronary cusp of the aortic valve begins. From the atrial side, the dome of the left atrium is attached to it, separating the anterior leaflet of the mitral valve from the aortic root. This fibrous body may be damaged by infective endocarditis, degenerative calcification, or previous mitral valve replacement, making combined aortic and mitral valve replacement difficult.¹⁻⁸

Patients with rheumatic heart valve disease may have small aortic and mitral anuli partially as a consequence of inadequate growth or fibrotic contraction of the fibrous body. Double valve replace-

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Read at the Seventy-seventh Annual Meeting of The American Association for Thoracic Surgery, Washington, D.C., May 4-7, 1997.

Received for publication May 6, 1997; accepted for publication June 5, 1997.

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0022-5223/97 \$5.00 + 0 12/6/84084

ment in these patients is often complicated by patient-valve size mismatch.^{9,10}

This article describes our experience with aortic and mitral valve replacement in patients who needed reconstruction of the intervalvular fibrous body.

Patients and methods

From 1985 to 1996, 43 patients who needed aortic and mitral valve surgery were found to have a diseased intervalvular fibrous body. Table I shows the clinical profile of these patients. The principal indication for operation was infective endocarditis in 14 patients (native valve in 3 and prosthetic valve in 11) and symptoms of congestive heart failure in 29. The offending microorganisms in patients with active infective endocarditis were staphylococci in 6 patients, streptococci in 5, and other bacteria in 3. The indication for reconstruction of the intervalvular fibrous body was abscess in 14 patients, extensive calcification of the mitral anulus and fibrous body in 9, lack of tissue to secure the prosthetic valves because of previous valve replacements in 10, and small aortic and mitral anuli in 10. Eleven patients had more than one of the aforementioned indications.

Operative procedures. An oblique aortotomy was performed and the aortic valve was excised. The aortotomy was then extended into the noncoronary aortic sinus and aortic anulus until it reached the dome of the left atrium, which was incised cephalad from the aortic root to the level of the right pulmonary artery, as illustrated in Fig. 1. The left atrium was exposed, and the mitral valve and the diseased intervalvular fibrous body were excised. The extensiveness of this resection varied with the pathologic process in the fibrous body. It ranged from an en bloc resection including the dome of the left atrium, the fibrous body, and the mitral valve in patients with a large abscess in this area, to a simple incision in the central portion of the fibrous body in patients who had elective double valve replacement and were found to have small aortic and mitral anuli. The fibrous body was reconstructed with a triangular patch of glutaraldehyde-fixed bovine pericardium in patients who had endocarditis and Dacron fabric in the others, as shown in Fig. 2. The sides of the patch were sutured to the lateral and medial fibrous trigones and aortic root with a continuous 3-0 polypropylene suture. A prosthetic mitral valve was secured to the mitral anulus posteriorly and to the pericardial patch superiorly. Before the prosthetic mitral valve was implanted, a second triangular patch was placed on the outside of the first patch and used to close the left atriotomy, as illustrated in Fig. 3. A prosthetic aortic valve was secured to the aortic anulus and to the pericardial patch, as demonstrated in Fig. 4. The patch was used to close the right side of the aortotomy. This operation was performed in 33 patients.

The posterior mitral anulus was either heavily calcified or destroyed by endocarditis in 10 patients. In addition to excision of the fibrous body, the posterior fibrous mitral anulus was also debrided in these patients. The entire base of the left ventricle was reconstructed with a single large patch of glutaraldehyde-fixed bovine pericardium, as illus-

Table I. Clinical profile

No. of patients	43
Age (yr)	
Mean \pm SD (range)	58 \pm 12 (31-80)
Sex	
Male	18 (42%)
Female	25 (58%)
ECG	
Sinus	31 (72%)
Atrial fibrillation	10 (23%)
Heart block	2 (5%)
NYHA functional class	
III	10 (23%)
IV	33 (77%)
Preoperative shock	9 (21%)
Previous MVR \pm AVR	32 (74%)
Active infective endocarditis	14 (33%)
LV ejection fraction	
>55%	11 (25%)
41%-54%	27 (63%)
21%-40%	5 (12%)
Tricuspid insufficiency	15 (35%)
Coronary artery disease	3 (7%)
Timing of operation	
Elective	16 (37%)
Urgent	18 (42%)
Emergency	9 (21%)

SD, Standard deviation; ECG, electrocardiogram; NYHA, New York Heart Association; MVR, mitral valve replacement; AVR, aortic valve replacement; LV, left ventricular.

trated in Fig. 5. The patch was sutured to the endocardium of the left ventricle posteriorly, to the lateral and medial fibrous trigones, and to the aortic root.

Table II summarizes the operative data.

Results

Seven operative deaths occurred (16%), 2 caused by technical problems, 2 by myocardial infarction, 2 by multiorgan failure, and 1 by hemorrhagic stroke. Three of 14 patients with infective endocarditis died, and 5 of 9 patients in preoperative shock died. Postoperative complications included reexploration of the mediastinum for bleeding in 3 patients, insertion of a permanent transvenous pacemaker for heart block in 6, and early prosthetic valve endocarditis in 2 patients who did not have endocarditis before the operation. In both patients the patches and valves were successfully changed.

Operative survivors have been followed up from 4 to 108 months (mean 38 \pm 29 months). No patient was lost to follow-up. Six late deaths have occurred, caused by cerebral bleeding in 1, late prosthetic valve endocarditis in 1 (patient refused reoperation), congestive heart failure in 2, and noncardiac causes in 2. The actuarial survival is shown in Fig. 6; it was 56% \pm 6% at 6 years.

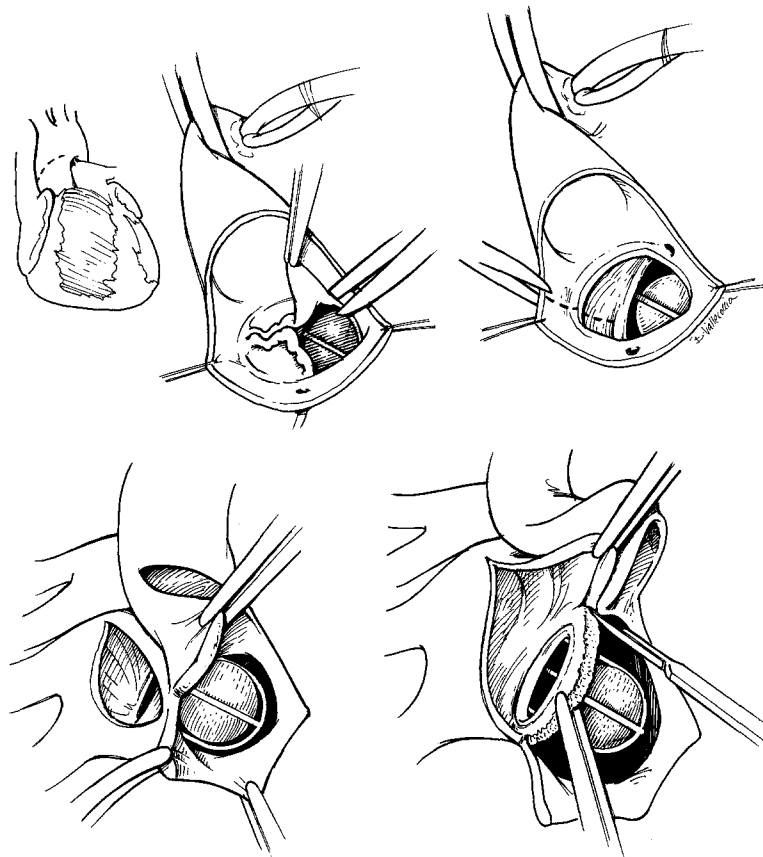


Fig. 1. An oblique aortotomy is extended into the mitral annulus and dome of the left atrium. The aortic and mitral valves are excised.

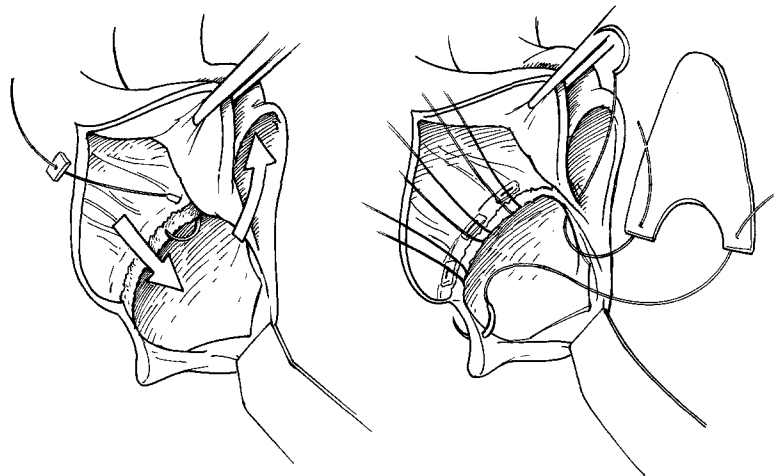


Fig. 2. A properly tailored patch is used to reconstruct the intervalvular fibrous body.

Thirty patients were alive at the last follow-up contact, and only 2 had symptoms attributable to their heart disease. The remaining 28 (93%) had no symptoms referable to cardiac disease. Every

patient had a Doppler echocardiographic study and no patient had prosthetic valve dysfunction or prosthetic valve dehiscence. No echocardiographic or radiographic evidence of calcification

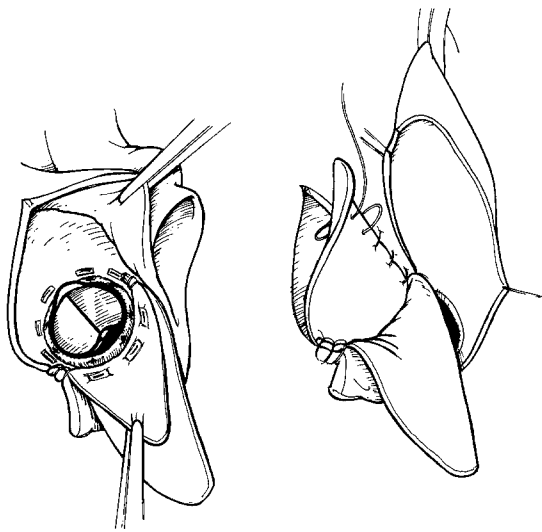


Fig. 3. A mitral valve prosthesis is secured to the mitral annulus posteriorly and to the patch superiorly. A separate patch is used to close the left atriotomy.

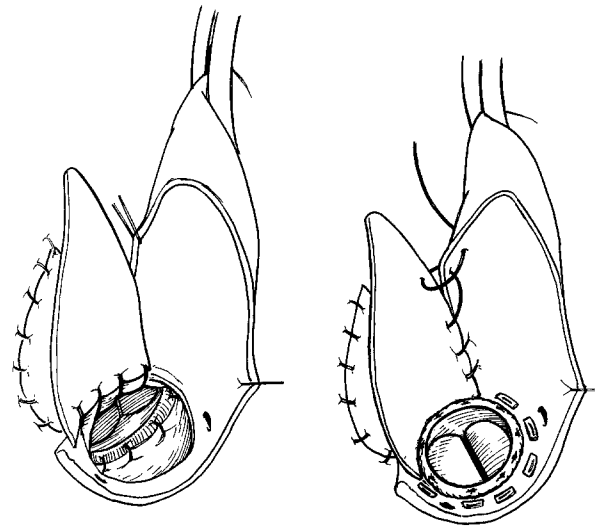


Fig. 4. An aortic valve prosthesis is secured to the aortic annulus and patch. The patch is used to close the right side of the aortotomy.

was detected in any of the bovine pericardial patches.

Discussion

The surgical disease described in this study is uncommon. At the Toronto Hospital, where approximately 650 patients with heart valve disease are operated on annually, only 3 to 5 patients with aortic and mitral valve disease are found to have a damaged intervalvular fibrous body. The most common cause of damaged intervalvular fibrous body is infective endocarditis of the aortic and mitral valve with paravalvular abscess.^{1,2} Only a few reports on the surgical treatment of abscess of the intervalvular fibrous body have been published.³⁻⁵ In the present study, 14 patients had endocarditis of the aortic and mitral valve with an abscess in the intervalvular fibrous body. Eleven of 14 patients had prosthetic valve endocarditis. The radical resection of the abscess and reconstruction of the base of the heart with glutaraldehyde-fixed bovine pericardium and valve replacement cured the infection, and 11 operative survivors had normally functioning prosthetic valves during the years of follow-up.

Another uncommon pathologic process that makes mitral valve surgery difficult and hazardous is the so-called "horseshoe" calcification of the mitral annulus, which occasionally extends into the fibrous trigones and intervalvular fibrous body.^{6,7} Although mitral valve repair is feasible in some of these patients,^{7,8} when the calcification extends into the

Table II. Operative data

Reconstruction of fibrous body	33 (77%)
Reconstruction of fibrous body + MA	10 (23%)
Patch material	
Bovine pericardium	28 (65%)
Dacron fabric	15 (35%)
AVR + MVR	
Mechanical valves	30 (70%)
Bioprosthetic valves	13 (30%)
Associated procedures	
TV annuloplasty	15 (35%)
PV replacement	1 (2%)
Coronary artery bypass	3 (7%)
Aortic root replacement	1 (2%)
Crossclamp time (min)	121 ± 22
Mean ± SD	
Cardiopulmonary bypass time (min)	145 ± 28
Mean ± SD	

MA, Mitral annulus; AVR, aortic valve replacement; MVR, mitral valve replacement; TV, tricuspid valve; PV, pulmonary valve; SD, standard deviation.

fibrous trigones and aortic valve, combined aortic and mitral valve replacement is usually necessary. Of 9 patients with this disease, 6 of them had already had at least one previous mitral valve replacement elsewhere and were referred because of prosthetic mitral valve dehiscence. The calcium had not been removed in any of these 6 patients at the previous operations. We managed these patients by excising all calcified tissues and reconstructing the entire base of the left ventricle with bovine pericardium. One of these 9 patients required reoperation for early prosthetic valve endocarditis. He had had 4

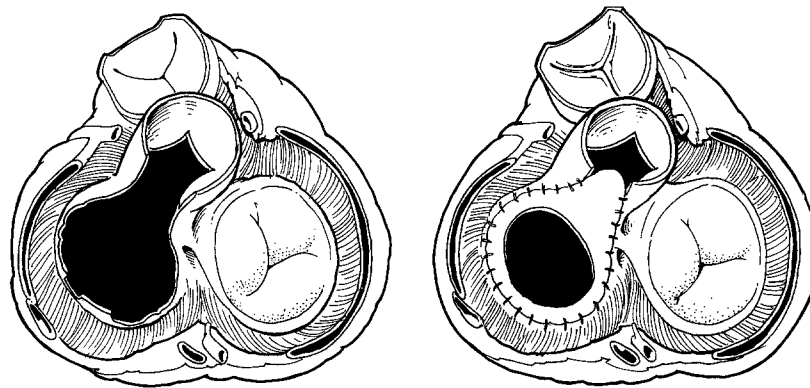


Fig. 5. Technique used to reconstruct the entire mitral annulus and the intervalvular fibrous body.

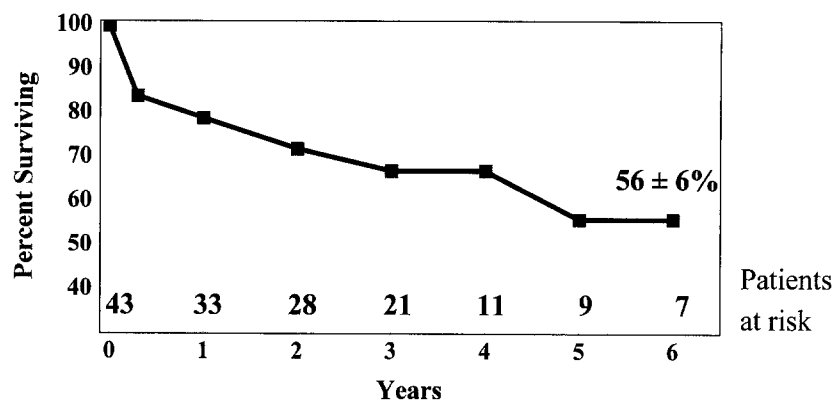


Fig. 6. Actuarial survival.

mitral valve replacements elsewhere and the reconstruction of the base of the heart had been his fifth heart operation. The postoperative course after the sixth operation was stormy, but he survived and eventually did very well. The remaining 8 patients had uneventful postoperative courses and had normally functioning prosthetic valves at the most recent examination.

Reoperations for failed prosthetic heart valves are usually associated with increased operative mortality and morbidity because of advanced symptoms or complex anatomic factors, or both. One of these anatomic factors is a damaged valve annulus. This problem is often seen after multiple previous valve replacements or when the prosthetic valves have been in place for a long time, usually one or two decades. The intervalvular fibrous body becomes rigid and sometimes calcified, and it fractures as the valve sutures are placed or tied down. Although it is possible to secure the aortic valve prosthesis in the

sewing ring of the mitral valve prosthesis in that area, we do not believe this should be done when mechanical valves are used. Inadequate intervalvular fibrous body was the indication for reconstruction in 10 patients in our series. Two of these patients died; both were in cardiogenic shock before the operation because of thrombosed mechanical valves.

Prosthetic valve-patient mismatch has been documented after aortic valve replacement.^{9, 10} It has been suggested by Dusmenil and associates¹¹ that the indexed prosthetic aortic valve area should not be less than 0.9 cm²/m². This type of information is not available for mitral valve replacement, but we estimate that the indexed prosthetic mitral valve area should be 1.3 to 1.5 cm²/m². Smaller prosthetic mitral valves are usually associated with symptoms of pulmonary congestion during exercise.

In 1979 Manouguian, Abu-Aishah, and Neitzel¹² described an experimental procedure whereby both

the aortic and mitral anuli were enlarged by increasing the width of the intervalvular fibrous body. In a report on patch enlargement of the aortic anulus in 8 patients, Manouguian and Seybold-Epting¹³ described the case of 1 patient who had severe mitral regurgitation after aortic valve replacement with enlargement of the aortic anulus and required mitral valve replacement 4 days later. Because the fibrous body had been divided at the initial operation, a Dacron patch was used to close the gap and to enlarge both the mitral and aortic anuli. We have performed this procedure in 10 patients who required aortic and mitral valve replacement and were found to have small anuli. All patients had rheumatic heart disease; 5 had had previous valve surgery and had prosthesis-patient mismatch; no patient had an aortic anulus larger than 19 mm, and 3 patients had a mitral anulus of 23 mm or less. After reconstruction of the intervalvular fibrous body, the prosthetic mitral valve implanted was 27 or 29 mm in all patients and the prosthetic aortic valve was 21, 23, or 25 mm, depending on the patient's body surface area, to prevent prosthesis-patient mismatch. Two patients died after the operation; operative survivors have had normally functioning prosthetic valves and adequate effective valve areas.

Combined aortic and mitral valve replacement with reconstruction of the intervalvular fibrous body is a long operation, particularly in patients who have had multiple previous heart valve operations. Long operations are associated with an increased risk of intraoperative contamination and perioperative bacteremia. Early prosthetic valve endocarditis developed in two of our patients. We ordinarily give prophylactic antibiotics for 24 hours after cardiac operations, but because of this study we changed our policy and will give intravenous antibiotics for at least 2 days in patients who undergo complex and long cardiac valve operations.

The approach used to treat this small number of patients with aortic and mitral valve disease and complex annular abnormalities was associated with high mortality and morbidity, but we believe that most of these patients would not survive conventional heart valve replacement.

Although we have concerns about the durability of glutaraldehyde-treated bovine pericardium as heart valve anuli because it may degenerate and the prosthetic valve may dehiscence, it has been an excellent material for use in reconstructing the heart in patients with paravalvular abscess and has given very satisfactory midterm results.^{1,8}

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Discussion

Dr. Alain F. Carpentier (Paris, France). The authors must be congratulated for bringing to our attention a very difficult problem for construction of the aortic-mitral junction when destroyed by infection or calcification. The type of reconstructive operation depended in their series on the extension of the lesions. For lesions limited to the aortic-mitral triangle, a simple triangular patch can be used. For lesions extending to the aortic and the mitral anulus, they used a large single patch of bovine pericardium in which an opening is made through which to place and secure a mitral valve prosthesis.

The first of these techniques is known and widely used by most surgeons with good results. The second, however, is a significant improvement over a similar technique described some 10 years ago, in which a large oval patch was sutured to the left atrial wall in the supraannular

position. The technique proposed by these authors has a lower incidence of patch and valve dehiscence.

The most important difference is the fact that the authors propose to secure the patch to the left ventricle itself. This approach allows the surgeon to use a smaller patch and to secure the patch in a stronger area; therefore the risk of displacement of the mitral prosthesis and dehiscence of the patch is reduced.

It is probably appropriate to mention another alternative that I prefer to use in this difficult situation, which is used often in a homograft aortic valve with the mitral leaflet left attached to the aortic homograft. This anterior leaflet is used to reconstruct the junction, and then the mitral valve annulus is reconstructed with a sliding plastic repair of the left atrium secured to the upper limit of the ventricle.

I have three questions for Dr. Kuo. First, is your patch reinforced with a layer of Dacron fabric? Second, how do you minimize the motion of the mitral valve between systole and diastole to reduce the incidence of dehiscence, patch dehiscence or valve dehiscence? Third, why do you not use the homograft valve more often and use the mitral valve as a living tissue, particularly in patients with bacterial endocarditis?

Dr. Ratna A. Magotra (*Bombay, India*). I would like to share the sentiments regarding some patients with rheumatic heart disease with mitral stenosis, aortic stenosis, and gross left ventricular hypertrophy.

In our experience, even after double valve replacement, it was not infrequent to have less than satisfactory regression of the left ventricle. These patients continued to have symptoms, with normally functioning prosthetic heart valves but very high transvalvular gradients.

The technique described by the authors has been successfully used to enlarge the mitral and aortic annuli so that larger prosthetic valves can be implanted.

My only question for Dr. Kuo concerns heart block. He describes six patients requiring permanent pacemakers, and I presume they include the two patients who had permanent heart block before the operation. Were there any instances of temporary heart block and, if so, how long did it last?

Dr. Kuo. I would like to thank Professor Carpentier for his kind remarks. In answer to his first question, we did not use Dacron fabric for reinforcement.

Regarding his second question, the rocking motion of the mitral valve during systole and diastole does worry us a bit. Dr. David modified his technique to try to reduce this rocking motion by not leaving too much patch between the sewing ring and the newly reconstructed mitral annulus. Obviously this is a worry, but we have not had any periprosthetic leak as a result of that in the 30 patients who survived the operation.

Third, we have not actually used a homograft in this situation.

Regarding Dr. Magotra's question, I suspect that the poor regression of the left ventricular hypertrophy in her group of patients may be the result of patient-valve size mismatch. If too small a valve is used, the left ventricular hypertrophy does not regress as much, as is shown very nicely in patients who previously had stentless valves.

The six patients who had pacemakers all had complete heart block after the operation. That did not include the two patients with complete heart block who had a pacemaker in place before the operation.