Volume 118 Number 2 August 1999

The Journal of
THORACIC
AND
CARDIOVASCULAR
SURGERY

## SURGERY FOR CONGENITAL HEART DISEASE

# AORTIC VALVE REPAIR BY CUSP EXTENSION WITH THE USE OF FRESH AUTOLOGOUS PERICARDIUM IN CHILDREN WITH RHEUMATIC AORTIC INSUFFICIENCY

Afksendiyos Kalangos, MD, PhD[a]
Maurice Beghetti, MD[b]
Ary Baldovinos, MD[a]
Dominique Vala, MD[a]
Thierry Bichel, MD[a]
Bernadette Mermillod, BSc[a]
Nicolas Murith, MD[a]
Ingrid Oberhansli, MD[b]
Beat Friedli, MD[b]
Bernard Faidutti, MD[a]

***Objectives:*** **Our goal was to evaluate the midterm results of aortic valve repair by a more sophisticated tailoring of cusp extension—taking into account the dimensions of the native aortic cusps—with the use of fresh autologous pericardium.** ***Patients and methods:*** **Forty-one children who had severe rheumatic aortic insufficiency (mean age 11.5 ± 2.7 years) underwent aortic valve repair by means of this cusp extension technique over a 5-year period. Twenty-four of them underwent concomitant mitral valve repair for associated rheumatic mitral valve disease. All children were then followed up by transthoracic echocardiography before discharge, at 3 and 6 months after the operation, and at yearly intervals thereafter.** ***Results:*** **Follow-up was complete in all patients and ranged from 3 months to 5 years (median 3 years). No operative and no early postoperative deaths occurred. Only 1 patient died, 9 months after the operation, of septicemia and multiple organ failure. Actuarial survival was 97% at 1 year and has remained unchanged at 3 years. On discharge, the degree of aortic insufficiency was grade 0 for 27 children and grade I for 14. Exacerbation of aortic insufficiency from grade I to grade II was observed in only 1 patient, and none of the children required reoperation for aortic insufficiency during the follow-up period. Mean peak systolic aortic valve gradients at discharge were lower than preoperative values ($P$ = .04), and no significant increase in the peak systolic**

From the Clinic for Cardiovascular Surgery[a] and the Clinic for Pediatric Cardiology,[b] University Cantonal Hospital of Geneva, Geneva, Switzerland.

Received for publication Aug 7, 1998; revisions requested Sept 17, 1998; revisions received Oct 12, 1998; accepted for publication Oct 27, 1998.

Address for reprints: Afksendiyos Kalangos, MD, PhD, Clinic for Cardiovascular Surgery, University Cantonal Hospital of Geneva, 24, Rue Micheli-du-Crest, 1211 Geneva 14, Switzerland.

J Thorac Cardiovasc Surg 1999;118:225-36



0022-5223/99 $8.00 + 0 **12/1/99037**

**transvalvular gradient was detected thereafter during the follow-up period. Mean left ventricular dimensions were significantly reduced at discharge when compared with preoperative values (*P* < .0001).** ***Conclusions:*** **Functional results of aortic valve repair with cusp extension using fresh pericardium have been satisfactory at medium term, particularly in children with a small aortic anulus at the time of initial repair, because the expansion potential of fresh autologous pericardium is equivalent to that of the growing sinotubular junction and aortic anulus diameters. (J Thorac Cardiovasc Surg 1999;118:225-36)**

Increasing awareness of the limitations of prosthetic valve replacement, especially in the young population, forces surgeons to explore and apply more conservative procedures on valves. Standardization, reproducibility, and stable long-term results are the three key points that have made mitral valve (MV) repair an established form of surgical treatment.[1] However, aortic valve (AV) repair is far from fulfilling these key points, although it has evolved in technical refinement and in the identification of a definite subset of patients who might benefit from it. Aortic cusp extension, the oldest of the AV repair techniques, has improved over the years in terms of the shape of the patch and the material used, hence permitting encouraging early and midterm results. This article is a description of the operative procedures we have used to repair the aortic valve by a more sophisticated tailoring of cusp extension using fresh autologous pericardium in children with rheumatic aortic insufficiency (AI) and of the results we have obtained during the past 5 years.

## Patients and methods

This study, from March 1, 1993, through March 1, 1998, reviewed the case histories of 41 children with AI of rheumatic origin who underwent valve repair by the same surgeon (A.K.). The series included 25 male (61%) and 16 female (39%) patients with a mean age of 11.5 ± 2.7 years (range 5-17 years). Preoperatively, 33 patients (80%) were in New York Heart Association functional class III and 8 (20%) were in class IV. No patient had had a previous cardiac operation.

The diagnosis of AV disease was established by preoperative transthoracic 2-dimensional echocardiography with Doppler studies in the long- and short-axis, 5-chamber, and suprasternal notch sagittal views; MV disease was diagnosed by these studies in the long-axis and 4-chamber views. In all patients, the diameters of the aortic anulus and of the sinotubular junction were measured in the long-axis view by transthoracic echocardiography. Indications for operation included the presence of grade III or IV AI resulting in progressive increase in left ventricular dimensions. Three patients had peak systolic AV gradients higher than 20 mm Hg. In 21 patients the associated MV lesion was predominantly regurgitant (grade III), and in 3 patients it was predominantly stenotic with an MV area of less than 1 $cm^2$. Seventeen patients (41%) had isolated AV repair. Twenty-four patients (59%) underwent AV repair in combination with MV repair. Patients with 4 valvular calcifications were excluded from valve repair procedures. None of the patients had active rheumatic carditis at the time of the operation.

Intraoperatively, in all patients, anatomic and functional study of the AV was done before and after cardiopulmonary bypass by transesophageal echocardiography.

**Operative procedures.** Cardiopulmonary bypass with systemic hypothermia to 28°C was used in all patients. A left ventricular vent was placed through the right superior pulmonary vein. An aortotomy was made transversely, 2 cm above the right coronary artery, and extended downward toward the noncoronary sinus. In all cases, myocardial protection consisted of topical hypothermia and hyperkalemic crystalloid solution selectively infused into both coronary ostia, combined with retrograde cardioplegia in 7 patients. Repeated doses were given at intervals of 30 minutes throughout the operation.

First, the MV was explored through a left atriotomy. In 24 patients, concomitant MV repair consisted of 24 annuloplasties with a Carpentier-Edwards prosthetic ring (Baxter Healthcare Corp, Edwards Division, Santa Ana, Calif), 20 resections of secondary posterior and/or anterior chordae, 14 transpositions of anterior secondary chordae to the free edge of the anterior cusp, 6 chordal shortenings, and 3 commissurotomies with splitting of both papillary muscles.

At the aortic level, two stay sutures were first placed through the aortic wall close to the margin of the lower flap of the aortotomy at sites corresponding to the right-left coronary and the right-noncoronary commissures. Another stay suture was then placed at the midpoint of the upper flap of the aortotomy and fixed to the right superior border of the pericardium to adequately expose the AV. All aortic cusps were first inspected and measured with particular regard to the length of the free edge, the height of the cusps and commissures, and the appearance of the sinuses of Valsalva, the aortic anulus, and the sinotubular junction. In all patients, tricuspid morphology of the AV was confirmed. No patients had aortic cusp calcification. In 27 patients, a variable degree of cusp thickening and retraction was found with no evidence of cusp prolapse, 15 of whom needed cusp shaving. Six patients

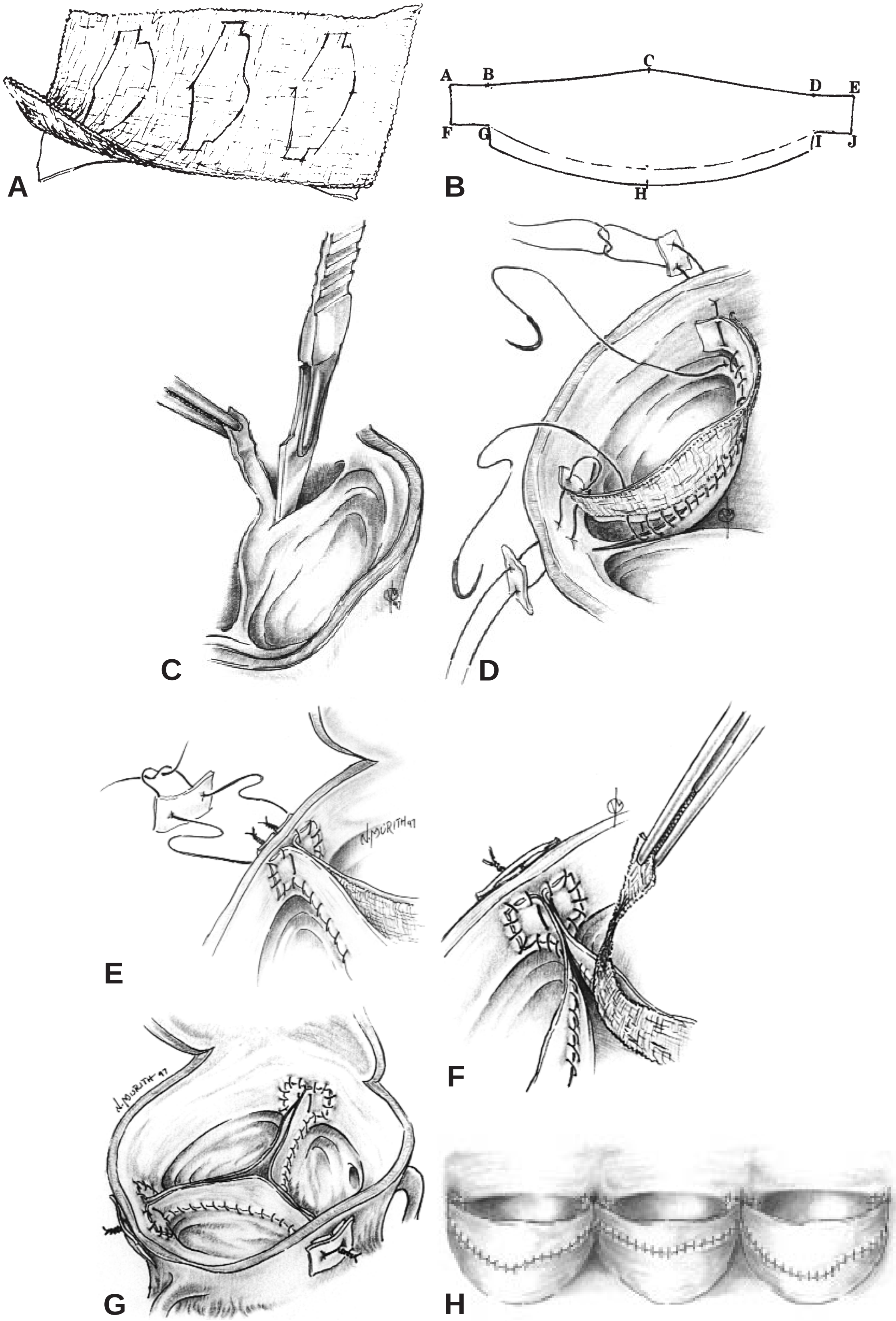


**Fig 1. A-H,** Surgical technique of aortic cusp extension with fresh autologous pericardium.

**Table I.** *Postoperative change in the degree of AI in 41 children after aortic cusp extension*

| | *Preop (TTE)* | *After CPB (TEE)* | *Discharge (TTE)* | *6 mo (TTE)* | *1 y (TTE)* | *2 y (TTE)* | *3 y (TTE)* | *4 y (TTE)* | *5 y (TTE)* |
|---|---|---|---|---|---|---|---|---|---|
| Grade 0 | | 29 | 27 | 24 | 22 | 19 | 14 | 7 | 1 |
| Grade 1 | | 12 | 14 | 15 | 8 | 10 | 9 | 6 | |
| Grade 2 | | | | 1 | 1 | | | | |
| Grade 3 | 24 | | | | | | | | |
| Grade 4 | 17 | | | | | | | | |
| Total patients | 41 | 41 | 41 | 40 | 31 | 29 | 23 | 13 | 1 |

*TTE*, Transthoracic echocardiography; *TEE*, transesophageal echocardiography, *CPB*, cardiopulmonary bypass.

underwent concomitant aortic commissurotomy before proceeding to aortic cusp extension. Among the 14 remaining patients, 6 had one aortic cusp prolapse with elongation of the free edge and retraction in the height of the cuspal tissue: in 4 of them, the noncoronary cusp was prolapsed and in 2, the right coronary cusp was prolapsed. Eight patients had associated right and noncoronary cusp prolapse. In these cases, thickening of the prolapsing cusp was slight or moderate and concentrated especially on the free edge.

A large rectangular piece of anterior autologous pericardium was cut and applied on a wet woven Dacron patch with its mesothelial surface upward after the loose areolar tissue attached to its surface had been removed by blunt dissection. Subsequently, the shape of each pericardial patch was traced onto the Dacron patch, which served as a template for sizing, trimming, and handling of the attached pericardium according to the established dimensions (Fig l, *A*). In Fig l, *B*, the dimension *B-D* equals the diameter of the sinotubular junction plus 15% of this diameter to account for eventual loss resulting from pericardial shrinkage. The dimension *G-I* equals the length of the cusp's free edge. The height of the pericardial extensions (*A-F* and *E-J*) is equal to 3 mm. The dimension *C-H* is designed according to the height of each cusp in the middle of each one's axis. The height of the patches should be 5 mm or longer, depending on the height of the native cusps, so as to render equal the height of each extended aortic cusp at the sinotubular junction after repair. In our series, the mean height of pericardial extensions was 6 ± 0.4 mm, varying from 5 to 8 mm. The height of each patch was oversized by approximately 2 mm to allow for the suture line on the free edge of each corresponding aortic cusp. The cusps were first thinned if their mobility was restricted (Fig l, *C*). However, we tried to maintain a leathery consistency at cuspal free edges to better hold the sutures. A 5-0 double-ended polypropylene suture (Cardionyl, Péters Pharm Lab, Bobigny, France) was passed through the midpoint of the pericardial patch and through the midpoint of the corresponding cusp in such a manner that, when tied, the knot stayed on the aortic aspect. Two vertical 5-0 polypropylene "U" stitches were then passed through both commissural extensions of the pericardial patch (*ABFG* and *DEIJ*) and aortic wall above both commissures (Fig l, *D*). These two commissural stitches were passed through pericardial pledgets and then tied outside the aorta. This was done to facilitate the next step of cusp extension, which consisted of running one of the arms of the tied suture over and over from the midpoint of the cusp, up the commissures, to the end point of pericardial extension on the aortic wall. A similar procedure was followed for the other half of the cusp's free edge with the use of the second arm of the suture (Fig l, *D*). The suture was then brought out through the aortic wall and kept tense. The running sutures of the neighboring cusps brought out through the aortic wall at the same commissural extension level were then tied together over a pericardial pledget outside the aorta (Fig l, *E*). The Dacron patch, applied on the pericardium to aid in its handling, was then removed (Fig 1, *F*). The same procedure was repeated for each cusp with each specifically corresponding scalloped pericardial patch. Fig 1, *G*, shows the final view of the reconstructed AV; the mesothelial surface of the pericardium is placed so as to correspond to the aortic aspects of the cusps. Fig 1, *H*, shows the profile view of the reconstructed aortic cusps through a longitudinally opened aorta. Note that the height of each of the three pericardial extensions is different, owing to the variable amount of retraction of each of the native aortic cusps. With this technical approach, the extended aortic cusps remain separated from the aortic wall during systole because the total length of the free edge is smaller than the circumference of the bulging sinuses. This prevents eventual occlusion of the coronary ostia by direct apposition of the patches to the wall of the sinuses of Valsalva. We added two commissural extensions on each pericardial patch to ensure better balanced tension, especially concentrated on the commissural regions during systole when flexion of the free edges of the extended cusps is minimal. In this geometric configuration, the length of the free edge of each cusp, which is equivalent to 2 times the radius of the sinotubular junction plus 15%, is equal to the diameter of the aortic orifice and remains constant whether the valve is open or closed. Hence there is neither a lack of nor an excess of cusp tissue. The combined surface of the three cusps is approximately 15% larger than that of the sinotubular junction, assuring the coaptation of the cusps.

**Echocardiographic follow-up.** In all patients, transthoracic echocardiography was performed before discharge (6-12 days after the operation), 3 and 6 months after the operation, and at yearly intervals thereafter. Doppler echocardiographic grade of AI was measured with color Doppler flow and graded from the width and length of the regurgitant jet in the left

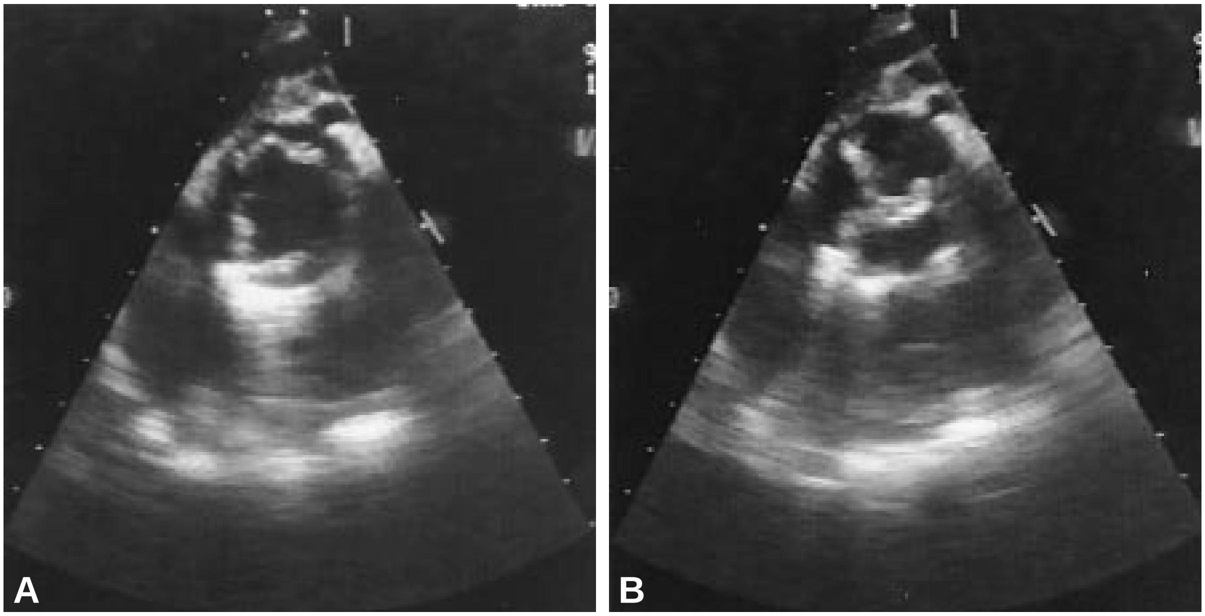


**Fig 2.** Postoperative echocardiography during the fourth year after aortic cusp extension: **A,** The AV in systole showing the wide open valve orifice of the reconstructed valve. **B,** The AV in diastole showing the tricuspid coaptation lines of the reconstructed valve.

ventricle (grade 0-IV) according to previously described criteria.[2] Peak gradient across the AV was estimated by measuring peak systolic velocity from Doppler studies in the transthoracic 5-chamber or suprasternal notch sagittal view. Left ventricular end-diastolic (LVEDD) and end-systolic diameters (LVESD) were measured at the midpapillary level in the standardized parasternal transthoracic long-axis and short-axis positions.

**Statistical analysis.** Continuous data such as peak systolic AV gradient and left ventricular dimensions were expressed as mean ± standard deviation, and changes between measurement time points were analyzed by means of the paired Student *t* test. All tests were 2-tailed. Change across time in left ventricular dimensions and mean peak systolic AV gradients was estimated by means of a slope for each patient on and after discharge. AI grade was presented as frequency distributions. Kaplan-Meier methods were used to derive the probability for survival.

## Results

The mean aortic crossclamping time was 66 ± 5 minutes (range 45-80 minutes) for 17 children undergoing isolated AV repair and 114 ± 17 minutes (range 92-135 minutes) for 24 children undergoing concomitant MV and AV repair.

**Clinical follow-up.** Clinical follow-up was complete in all cases and ranged from 3 months to 5 years (median 3 years). No operative or early postoperative deaths have occurred and few perioperative complications. Two patients (5%) required mediastinal re-exploration for the control of postoperative hemorrhage, but none had wound complications or neurologic events. Postoperative atrial fibrillation, later converted into sinus rhythm, occurred in 8 patients who underwent concomitant MV repair. All the other patients remained in sinus rhythm after the operation. The mean postoperative hospital stay was 9 ± 2 days. One patient who had had concomitant AV and MV repair died 9 months after the operation of septicemia and multiple organ failure in another hospital with an apparently competent AV and MV. Actuarial survival was 97% at 1 year (95% confidence limits: 85%-99%) and has remained unchanged at 3 years (95% confidence limits: 81%-99%). Late follow-up revealed functional improvement in all patients. Ninety-three percent of the patients are now in New York Heart Association class I and 7% in class II according to the latest follow-up examinations. None of the children required reoperation for AI during the follow-up period. However, 2 patients who had had concomitant MV repair required reoperation because of failure of the MV repair during the second and fourth postoperative years, respectively. In both of them the MV was replaced by a mechanical prosthesis. Although none of them had AI, the AV was explored at the time of reoperation to have a precise idea regarding

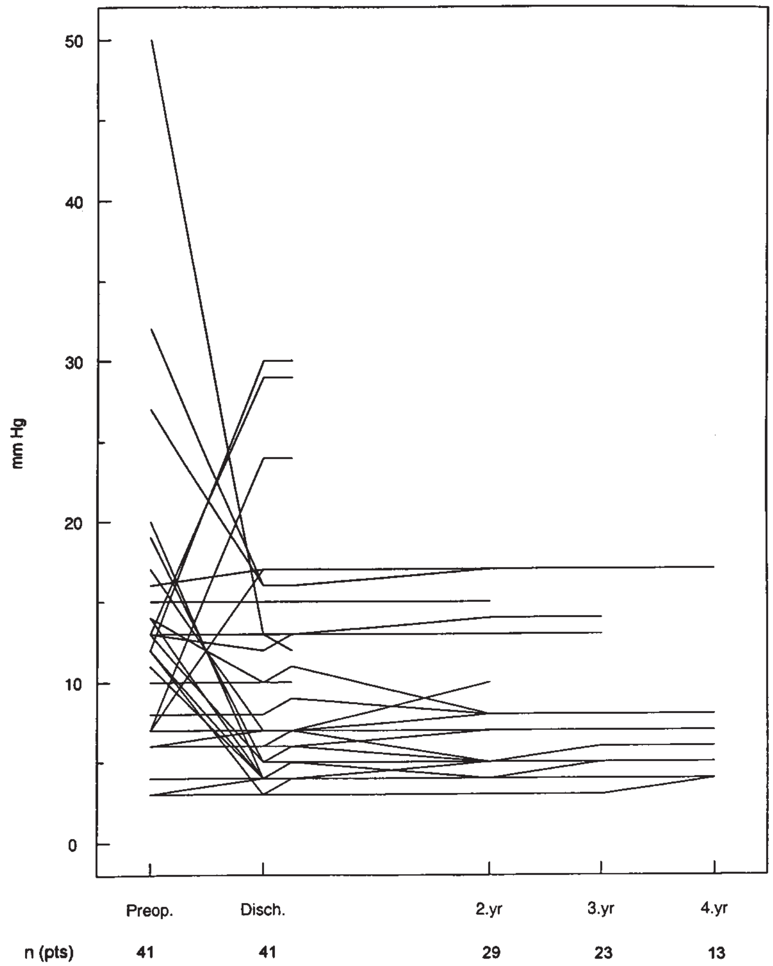


**Fig 3A.** Evolution of postoperative peak systolic AV gradients for each case with respect to the preoperative value at discharge *(disch.)* and at the second, third, and fourth postoperative years as measured by Doppler transthoracic echocardiography. In certain cases, part of the decrease in peak systolic AV gradients may be due to reduction in the degree of regurgitation with a resulting decrease in stroke volume or to cusp shaving, which renders cusps more flexible.

the outcome of the fresh autologous pericardium. Inspection showed slight homogeneous thickening of pericardial patches at their edges, but preserved pliability in their central portion. Cusp mobility was not altered, and pericardial commissural extensions were well incorporated into the aortic wall with complete neointimal covering. In the remaining patients who underwent concomitant MV repair, transthoracic echocardiographic examinations of the MV were stable during the entire follow-up period. No residual mitral insufficiency was detected after repair in 12 patients, and mild insufficiency was detected in 8 patients during the most recent follow-up examination. In all patients, anticoagulation with warfarin began on the second postoperative day and was stopped during the third postoperative month. No thromboembolic or hemorrhagic events were observed up to the most recent follow-up examinations.

**Echocardiographic follow-up.** All patients had echocardiographic assessment during the third postoperative month, 40 during the sixth postoperative month, 31 during the first postoperative year, 29 during the second year, 23 during the third year, 13 during the fourth year, and 1 during the fifth postoperative year.

*AI.* Transesophageal echocardiography on termination of cardiopulmonary bypass showed AI grade 0 in 29 patients and grade I in 12 patients. At discharge, the number of patients with grades 0 and I were 27 and 14

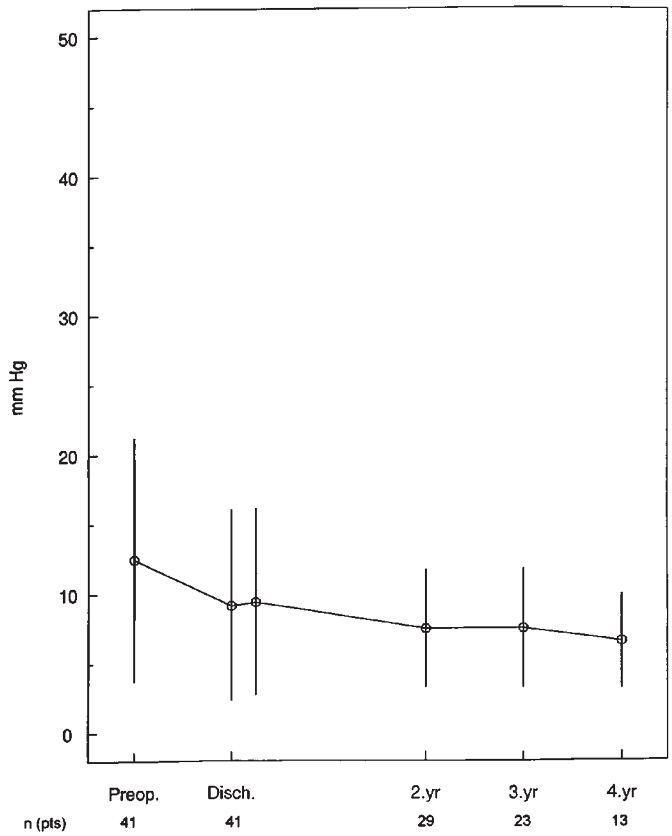


**Fig 3B.** Comparison of preoperative and postoperative mean peak systolic AV gradients after aortic cusp extension (means and standard deviations are shown). Postoperative values at discharge were significantly lower than preoperative values ($P = .04$).

by transthoracic echocardiography, respectively. Exacerbation of AI from grade I to grade II was observed in 1 patient during the sixth postoperative month, but no further significant change in the degree of AI was found thereafter, throughout 16 postoperative months. This patient is now in New York Heart Association class I. Table I shows the frequency distributions of AI before the operation, during the operation after repair (by transesophageal echocardiography), at discharge, at 6 months after the operation, and during the follow-up period at yearly intervals. Diameters of the aortic anulus and the sinotubular junction were measured before the operation and during each transthoracic echocardiographic examination in all patients who were under 12 years of age at the time of the initial repair. Eight patients under the age of 12, operated on in 1993, had a mean aortic anulus diameter of 16.2 ± 1.2 mm (range 14-17.4 mm) just before the operation and a mean sinotubular junction diameter of 15.8 ± 0.9 mm (range 13.8-17 mm). Seven of them had grade 0 and 1 had grade I AI at discharge. Only 2 of them passed on to grade I AI during the fourth postoperative year, despite the growth of the aortic anulus to 19.7 mm and 19.4 mm, respectively, and that of the sinotubular junction to 18.8 mm and 18.3 mm, respectively. The late follow-up transthoracic echocardiogram of 1 of these children during the fourth postoperative year showed mobile cusps with pericardial extension maintaining pliability, resulting in a wide valve orifice opening in systole (Fig 2, *A*) and good coaptation of extended cusps in diastolic closure (Fig 2, *B*).

*Peak systolic AV gradient.* Peak systolic AV gradients were 12.5 ± 8.8 mm Hg (range 3-50 mm Hg) before the operation, 9.2 ± 6.8 mm Hg (range 3-30 mm Hg) at discharge, 9.5 ± 6.7 mm Hg at 3 months, and 7.5 ± 4.2 mm Hg in the second postoperative year. Fig 3, *A*, shows the evolution of peak systolic AV gradient for each patient during the follow-up period. Six patients who had a decrease in peak systolic AV gradients at discharge underwent aortic commissurotomy for various

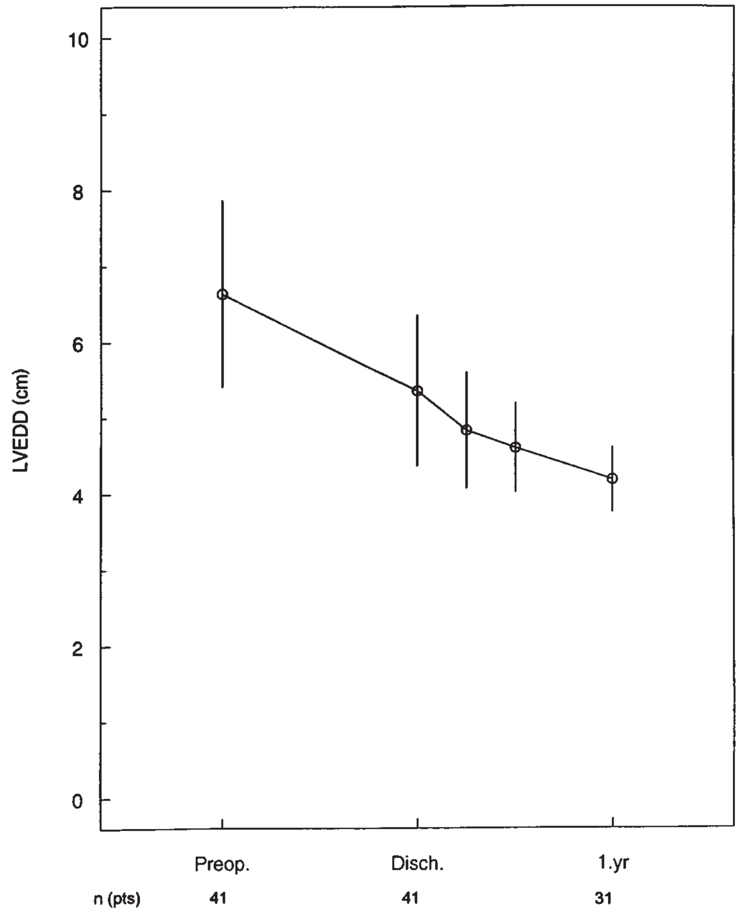


**Fig 4.** Comparison of preoperative and postoperative mean LVEDDs as measured by Doppler transthoracic echocardiography (means and standard deviations are shown). Postoperative values at discharge were significantly lower than preoperative values ($P < .0001$).

degrees of commissural fusion before proceeding to cusp extension. On the other hand, in 4 patients in whom an increase in AV gradients was disclosed at discharge, the sinotubular junction diameter was smaller than 15 mm before the operation. Postoperative AV gradients at discharge were significantly lower than preoperative values ($P = .04$). During the 4 postoperative years, mean peak systolic AV gradients tended to increase by 0.041 ± 0.29 mm Hg per 3 months, which was not statistically significant across time ($P = .37$) (Fig 3, *B*).

*Left ventricular dimensions.* Because of the increased preload caused by AI in 17 patients and associated mitral insufficiency in 24 patients, LVEDD and LVESD were enlarged before the operation with values of 6.6 ± 1.2 cm and 4.4 ± 0.9 cm, respectively. Mean LVEDD and LVESD were significantly reduced at discharge, with values of 5.4 ± 1.0 cm ($P < .0001$) and 3.7 ± 0.8 cm ($P < .0001$), respectively. During the first postoperative year, the decrease in LVEDD was 0.29 ± 0.17 cm in 3 months (Fig 4) and that in LVESD was 0.24 ± 0.17 cm in 3 months (Fig 5), the trimestrial decreases being statistically significant across time for both diameters ($P < .0001$). Two patients required reoperation for failure of the MV repair, but the remaining patients did not have an increase in left ventricular dimensions during the follow-up period. All patients had normal preoperative fractional shortening (>30%), and no deterioration was detected during the follow-up period.

## Discussion

AI caused by acute rheumatic fever remains a surgical challenge, especially in the pediatric population, in which the use of bioprostheses is threatened by limited durability, that of mechanical prostheses by problems resulting from permanent anticoagulation, and some-

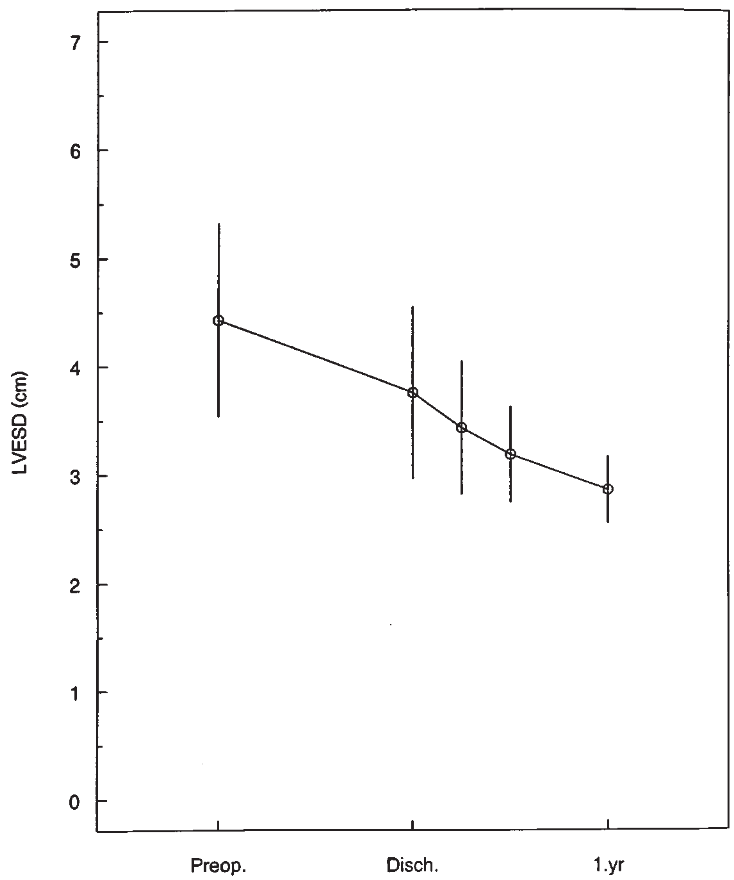


**Fig 5.** Comparison of preoperative and postoperative mean LVESDs as measured by Doppler transthoracic echocardiography (means and standard deviations are shown). Postoperative values at discharge were significantly lower than preoperative values ($P < .0001$).

times that of any prosthesis by the presence of a small valve anulus. The superiority of MV repair rather than replacement in the young rheumatic population recently renewed interest for AV repair in a selected subset of patients with rheumatic AI. As was observed in our series, this subset of pediatric patients usually had thickened, retracted aortic cusps with somewhat preserved mobility, the cusp involvement being attenuated with a thinned appearance in some cases. The aortic anulus in these patients remained normal or minimally dilated. In 1 of 3 or 4 cases, cusp prolapse caused by the elongation of the free edge could be seen. Fusion of the commissures was inconsistent and, when present, remained mild or moderate. In our series, only 6 patients required concomitant aortic commissurotomy, which explains why postoperative mean peak systolic AV gradients were significantly lower than preoperative values.

Aortic cusp extension as an AV repair technique has already been reported by some authors as a valuable surgical tool in rheumatic AI, with satisfactory clinical midterm results regarding freedom from reoperation, thromboembolic events, and endocarditis.[3-7] Although the tendency over time has evolved from the use of heterologous[3-5,8] to autologous pericardium[6] and from a single-patch to a three-patch configuration,[9] in none of these series have the real dimensions of native aortic cusps been taken into account in the tailoring process of the patches used in cusp extension. The reason seems to be either total excision of the cusps or resection of the thickened part of the cusps, leaving an equal height of remaining tissue in each cusp before re-establishing the dimensions of a native AV. We believe that such techniques of AV replacement rather than cusp extension demand more patch tissue and may not have the same midterm or long-term outcome when compared with true cusp extension, where the amount of patch used is less and cuspal integrity of the native AV is preserved. We totally agree that the native AV should have a certain mobility despite fibrosis before proceeding to true cusp extension. In these series in which total or partial excision of cusps was performed, mean patients' ages were older than those in our series.[3-7,9] Aging could favor more marked cuspal thickening and retraction in the context of evolving rheumatic valvular disease and, hence, no longer provide another surgical issue other than to totally or partially excise the cusps. On the other hand, we know that in the majority of cases of rheumatic AI, cusps have various degrees of pathologic involvement, rendering them nonidentical to each other in terms of height, length of the cuspal free edge, and thickness. Our series demonstrated that there was no similar value in terms of cuspal free edge and height between cusps of the same AV in 37 of 41 patients. This is the reason we have adopted a more geometric concept of cusp extension by adding the dimensions necessary to render cusps identical in height and length of cuspal free edge at the sinotubular junction, considered as the new coaptation level between extended cusps. Excessive stereotypic length of a patch's free edge without respect for the diameter of the sinotubular junction may unnecessarily create either festooned cuspal free edges or prolapse during diastole and floating patches during systole. On the other hand, equal heights of patches without taking into account the height of each aortic cusp can create unequal redundant cusps during diastole. Cuspal distortion and redundancy by excessive coaptation surface are major technical factors creating cuspal malalignment and thereby inducing turbulences. We believe that respecting the dimensions of native aortic cusps refines cuspal alignment, leading to better stress distribution, and may therefore prevent precarious shrinkage of the pericardial patches. Bahnson and associates[10] showed that a correctly sized and placed pericardial valve closely resembles an aortic homograft in the pulse duplicator. They concluded that a replaced cusp should be an accurate copy of the natural one.

The other important point in this technique is the choice of material. Although promising midterm clinical results have been reported by authors who have performed aortic cusp extension by glutaraldehyde-treated bovine pericardium after partially excising aortic cusps,[3,4] renewed interest is now focused on the use of autologous tissue and avoiding problems related to calcification and primary tissue failure found with heterograft bioprostheses and homograft valves, particularly in the pediatric population. However, Liao and colleagues[11] demonstrated in a rat model that autologous tissues treated with glutaraldehyde biologically behave like glutaraldehyde-treated xenogeneic ones: there was no difference between them regarding calcification tendency, so that neither was rendered preferable to the other. For this reason, short-time incubation (15 minutes) of the autologous pericardium in 0.62% glutaraldehyde-buffered solution was proposed for slowing down excessive scar formation and avoiding calcification.[12] This method showed encouraging results in both experimental and clinical studies, because such pericardium appeared to neither calcify nor shrink after implantation.[11,13,14] However, even short-time glutaraldehyde fixation may be enough to change the biologic identity of autologous pericardium, especially that of its protein-composed surface antigen by cross-linking protein chains. As a result, the short-time glutaraldehyde-treated autologous pericardium may be recognized as a "foreign body" by the host and induce certain inflammatory reactions.[11] Nevertheless, aortic cusp extension with short-time glutaraldehyde-treated autologous pericardium[9] seems to yield the same satisfactory clinical results as those obtained by mitral leaflet enlargement with similarly treated autologous pericardium.[14]

On the other hand, the literature concerning the use of fresh autologous pericardium in valvular surgery seems to be divided between those who observed progressive contracture and loss of pliability of fresh pericardium[15-18] and those who observed preserved pliability despite some degenerative changes and healing.[19-22] Frater and associates,[16,17] who reported that mitral and tricuspid leaflet extension with fresh pericardium results in thickening, retraction, and hyaline deposition, recognized that technical difficulties and consequent malalignment of the patch might be a contributing factor. Ross and Olsen[18] also seemed to blame the technique more than the fresh pericardium itself. Besides the inherent difficulty of reconstructive AV surgery, most of the previous work of AV replacement or cusp extension with fresh autologous pericardium was done at a time when myocardial protection was practically nonexistent and techniques were not well established. Preservation of cusp mobility and coaptation, the lack of evidence of transvalvular turbulence and cuspal calcification, and the lack of progression in immediately detected mild transaortic gradients at midterm echocardiographic studies demonstrate that the process of pericardial and native valvular shrinkage seems to be delayed in our series. In addition, direct observation of the reconstructed AV in 2 patients requiring reoperation for failure of MV repair showed pliable and mildly thickened extended aortic cusps. This reinforces the hypothesis that the disparity between fresh and glutaraldehyde-treated autologous pericardium, although attributed to glutaraldehyde by some authors, could also be due to the refined aortic cusp extension technique, resulting in better tissue alignment and stress distribution.

Although it may seem conflicting, the other important observation in favor of fresh autologous pericardium is the homogeneous adaptation of the reconstructed AV in time, with the increase in diameters of the aortic anulus and sinotubular junction. Despite concern regarding the viability and growth capacity of fresh autologous pericardium, we believe that the expansible potential of untreated pericardium was preserved in 8 patients who were under 12 years of age at the time of initial repair. The preserved cusp motion and coaptation despite the increase in diameters of the aortic anulus and the sinotubular junction in repeated echocardiographic studies for up to 4 years indirectly proves that fresh pericardium does not limit its expansile potential with time. Holdefer, Edwards, and Dowling[23] reported in 1968 that untreated autologous pericardial valves in calves demonstrated good evidence of viability up to 51 days. Maintenance of viability was suggested by Thomas and coworkers[24] in dogs with untreated autologous pericardium–lined skeletal muscle ventricles in circulation for variable intervals up to 2 years. The pericardium was smooth, without evidence of thrombus formation or calcification. On the other hand, glutaraldehyde treatment for 10 minutes limited the expansile characteristics of pericardium used for replacing an excised aortic arch segment in piglets.[25] The untreated pericardial patch, when compared with the treated one, was significantly longer and wider, with no statistically significant difference in the strength of the patches 6 months after implantation. We believe that glutaraldehyde initially inhibits the change in size of the patch, contrary to the untreated pericardial patch in which it probably takes more time for fibrosis to definitively stop its expansion.

## Conclusion

Reconstruction of the AV by cusp extension with autologous pericardium, because of its ready availability, excellent midterm results, and outstanding hemodynamic performance, can at present be especially recommended for children in whom anticoagulation and a small aortic anulus could generate problems. The surgical techniques of aortic cusp extension are simple, safe, and reproducible. We believe that the better extended cuspal alignment—taking into account the dimensions of the native aortic cusps—may increase the durability and preserve the pliability of pericardial patches in an area where very precise geometry and lack of redundant tissue is imperative. Expansion potential of fresh autologous pericardium parallel to growing sinotubular junction and aortic anulus diameters makes it very interesting and advantageous in children with a small aortic anulus, since the currently available valve prostheses make aortoventriculoplasty almost inevitable. Moreover, aortic cusp extension techniques in the context of evolving rheumatic AI can allow children as much time as possible for the aortic anulus to grow, which will later allow for the replacement of the AV by a larger valvular prosthesis.

## REFERENCES

1. Deloche A, Jebara VA, Relland JYN, et al. Valve repair with Carpentier techniques: the second decade. J Thorac Cardiovasc Surg 1990;99:990-1002.
2. Perry GJ, Helmcke F, Nanda NC, Byard C, Soto B. Evaluation of aortic insufficiency by Doppler color flow mapping. J Am Coll Cardiol 1987;9:952-9.
3. Batista RJV, Dobrianskij A, Comazzi M, et al. Clinical experience with stentless pericardial monopatch for aortic valve replacement. J Thorac Cardiovasc Surg 1987;93:19-26.
4. Yacoub M, Khaghani A, Dhalla N, et al. Aortic valve replacement using unstented dura or calf pericardium: early and medium term results. In: Bodnar E, Yacoub M, editors. Biological and bioprosthetic valves. New York: Yorke Medical Books; 1986. p. 684-90.
5. Al Fagih MR, Al Kasab SM, Ashmeg A. Aortic valve repair using bovine pericardium for cusp extension. J Thorac Cardiovasc Surg 1988;96:760-4.
6. Duran C, Kumar N, Gometza B, Al Halees Z. Indications and limitations of aortic valve reconstruction. Ann Thorac Surg 1991; 52:447-54.
7. Duran CMG, Alonso J, Gaite L, et al. Long term results of conservative repair of the rheumatic aortic valve insufficiency. Eur J Cardiothorac Surg 1988;2:217-23.
8. Senning A. Fascia lata replacement of aortic valves. J Thorac Cardiovasc Surg 1967;54:465-70.
9. Tapia M, Brizard C, Fremont D, et al. Chirurgie conservatrice pour insuffisance aortique rhumatismale. Arch Mal Coeur 1998; 90:1611-4.
10. Bahnson HT, Hardesty RL, Baker LD, et al. Fabrication and evaluation of tissue leaflets for aortic and mitral valve replacement. Ann Surg 1970;171:939-47.
11. Liao K, Frater RWM, LaPietra A, Ciuffo G, Ilardi CF, Seifter E. Time-dependent effect of glutaraldehyde on the tendency to calcify of both autografts and xenografts. Ann Thorac Surg 1995; 60:S343-7.
12. Carpentier A. Le concept de bioprothèses et son application à la fabrication de matériaux biologiques [PhD thesis]. Paris, Orsay; 1975.
13. Kumar SP, Prabhakar G, Kumar M, et al. Comparison of fresh and glutaraldehyde-treated autologous stented pericardium as pulmonary valve replacement. J Card Surg 1995;10:545-51.
14. Chauvaud S, Jebara V, Chachques JC, et al. Valve extension with glutaraldehyde-preserved autologous pericardium: results in mitral valve repair J Thorac Cardiovasc Surg 1991;102:171-8.
15. Bailey CP, Jamison WL, Bakst AE, et al. The surgical correction

of mitral insufficiency by use of pericardial grafts. J Thorac Surg 1954;28:551-603.
16. Frater RWM, Berghuis J, Brown AL, Ellis FH Jr. Autogenous pericardium for posterior mitral cusp replacement. Surgery 1963; 84:260-8.
17. Frater RWM, Berghuis J, Brown AL, Ellis FH Jr. The experimental and clinical use of autologous pericardium for the replacement and extension of mitral and tricuspid valve cusps and chordae. J Cardiovasc Surg 1965;6:214-28.
18. Ross JK, Olsen GJ. Mitral valve reconstruction by posterior cusp advancement using a pericardial graft: long-term follow up in nine patients. Thorax 1976;31:326-31.
19. Templeton JY III, Gibbon JH Jr. Experimental reconstruction of cardiac valves by venous and pericardial grafts. Ann Surg 1949; 129:161-76.
20. Bakst AA, Loewe RI. Pericardial valve graft in the surgical therapy of mitral insufficiency. Surg Forum 1957:8:383-6.
21. Sauvage LR, Wood SJ, Bill AH, et al. Pericardial autografts on the mitral valve: a preliminary report. J Thorac Cardiovasc Surg 1962:44:67-72.
22. Rittenhouse EA, Davis CC, Wood SJ, et al. Replacement of ruptured chordae tendineae of the mitral valve with autologous pericardial chordae. J Thorac Cardiovasc Surg 1978;75: 870-6.
23. Holdefer WG, Edwards WS, Dowling ER. An experimental approach to mitral valve replacement with autologous pericardium. J Thorac Cardiovasc Surg 1968;55:873-81.
24. Thomas SA, Isoda S, Hammond RL, et al. Pericardium-lined skeletal muscle ventricles: up to two years' in-circulation experience. Ann Thorac Surg 1996;62:1698-707.
25. Haluck RS, Richenbacher WE, Myers JL, Miller CA, Wise RK, Waldhausen JA. Pericardium as a thoracic aortic patch: glutaraldehyde-fixed and fresh autologous pericardium. J Surg Res 1990;48:611-4.