COMMISSURAL DEHISCENCE OF CARPENTIER-EDWARDS MITRAL BIOPROSTHESES

Explant analysis and pathogenesis

Manufacturing factors have seldom been implicated as a direct cause of structural deterioration of valvular bioprostheses; this phenomenon has generally been considered to be of a host-dependent origin. We analyzed the clinical and pathologic data from 12 Carpentier-Edwards mitral bioprostheses removed from 12 patients because of severe dysfunction and showing detachment of the porcine aortic wall from the stent in one commissure or more. These 12 prostheses were part of a group of 92 such valves that were explanted and displayed structural deterioration. They belong to a population of 405 Carpentier-Edwards bioprostheses implanted in the mitral position in our institution between May 1978 and November 1988. The patients included three men and nine women with a mean age of 54 ± 13 years. One patient had a history of chronic renal failure, and two had systemic hypertension. Prosthesis sizes were 29, 31, and 33 mm ($n = 4$ for each size). The models of the valves were 6625 ($n = 8$) and 6650 ($n = 4$). Mean duration of implantation of the prostheses was 99 ± 27 months (52 to 136 months) and did not differ depending on the model. There was no significant clustering of commissural detachments depending on valve size, year of implantation, or gender of the patient. No similar phenomenon was observed among 76 explanted aortic Carpentier-Edwards bioprostheses with structural deterioration from a population of 441 valves implanted during the same time frame. Native porcine aortic roots ($n = 5$) and aortic Carpentier-Edwards bioprostheses explanted because of structural deterioration ($n = 4$) were used as controls for comparison. Macroscopic examination showed single commissural dehiscence in 10 patients and double in two. Radiology disclosed no or mild mineralization in eight valves and no calcium in the area of aortic wall dehiscence, except for heavily calcified valves. Light microscopy evidenced a significant thinning of the aortic wall at the paracommissural level of mitral bioprostheses ($351 \pm 68 \mu\text{m}$) compared with either aortic bioprostheses ($526 \pm 59 \mu\text{m}$; $p < 0.01$) or control native porcine aortic roots ($419 \pm 50 \mu\text{m}$; $p < 0.01$). No difference was found in terms of aortic wall thickness between detached ($322 \pm 42 \mu\text{m}$) and intact ($366 \pm 74 \mu\text{m}$) commissures in mitral bioprostheses. It is concluded that the dehiscence of the aortic wall from the Dacron cover of the stent in the commissural area of Carpentier-Edwards bioprostheses in the mitral position is most likely produced by its weakening, as a consequence of excessive trimming with elimination of the outer layers of the aorta, during the manufacturing process. The reason this phenomenon appears in mitral bioprostheses and in a particular commissure seems to be linked to the areas of concentration of mechanical stress. (J THORAC CARDIOVASC SURG 1995;110:688-96)

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PPrimary tissue degeneration stands as the main drawback precluding a wider use of porcine heart valve bioprostheses, and it is the cause of the larger part of structural deterioration instances in this kind of device. This process has been defined as any tear, perforation, thickening, or mineralization of the biologic tissue of the prosthesis, intrinsic in origin, and occurring in the absence of a previous infective disease.^{1,2} Manufacturing factors have seldom been identified as directly involved in deterioration phenomena of porcine bioprostheses, which seems remarkable because these implants are largely hand-crafted.

Detachment of the aortic wall from the stent in the commissural area has been reported as causing clinical dysfunction of mitral Carpentier-Edwards porcine bioprostheses (Baxter Healthcare Corp., Edwards CVS Division, Santa Ana, Calif.).³⁻⁸ Some explanation has been given on the pathogenesis of this phenomenon,⁴⁻⁷ but no attempt has been made to evaluate the clinical and explant analysis data from patients and valves with this complication.

The aim of the present study was to analyze our experience with this complication at the Marqués de Valdecilla University Hospital (University of Cantabria, Santander, Spain) and to search for its causative factors and clinicopathologic correlations.

Methods

All prosthetic valve specimens were obtained at reoperation of the patients, and clinical data were collected in a retrospective manner. Bioprostheses were carefully explanted and rinsed with either Hanks or normal saline solution, care being taken not to alter the pathologic features. They were then immersed in a cold 10% buffered formaldehyde solution (pH 7.2) or a 2.5% glutaraldehyde solution with a 0.1 mol/L concentration of cacodylate buffer (pH 7.3) at 4° C. Valves were macroscopically inspected and the findings described. Color and black and white macrophotographs were obtained. The sewing ring and stent of each prosthesis were then carefully removed. Radiographs of the valves were obtained with the use of a General Electric-CGR apparatus (model Senographe 500T, 0.1 mm focus; General Electric Company, Fairfield, Conn.) for high-resolution mammography. After decalcification with sodium ethylenediamine tetraacetic acid, the valves were dehydrated in a graded series of alcohol solutions, and the whole specimen was embedded in methyl methacrylate. Circumferential sections, 5 μ m thick, were obtained at different levels to fully display the features of the commissural areas. The sections were stained with hematoxylin-eosin, Masson trichrome, orcein, and van Gieson techniques. Measurements of aortic wall thickness were taken in both normal and detached commissures with a calibrated microgrid (Olympus BH microscope, Olympus WHK 10 $\times$ /20L microgrid, magnifi-

cation 10 $\times$; Olympus Corp., Lake Success, N.Y.). Four aortic Carpentier-Edwards bioprostheses implanted during the same time frame, sizes 29 or 31 mm and explanted because of structural deterioration, were processed in a similar fashion, and the same protocol of measurements was applied.

Five porcine aortic roots, obtained at a slaughter factory, were used as controls. They were carefully dissected from the heart, rinsed with normal saline solution, and immersed for 15 minutes in a 1% solution of glutaraldehyde in phosphate buffer (pH 7.4) at room temperature. They were then transferred to a 0.625% solution of glutaraldehyde in phosphate buffer (pH 7.4) and kept at room temperature for 24 hours and at 4° C afterward until being processed for histologic studies. The porcine aortic roots were then dehydrated and embedded in paraffin. The sectioning and staining techniques used were similar to those applied to the clinical explants.

Clinical, operative, echocardiographic, and hemodynamic data were obtained from the medical records of the patients. Operative data included the manufacturer's code number of the valves. The methods and definitions used throughout the present study follow the recommendations issued by the liaison committee of The American Association for Thoracic Surgery and The Society of Thoracic Surgeons in 1988 for reporting results of cardiac valvular operations.²

Continuous variables were compared by a two-tailed Student's *t* test (for paired data when pertinent) or a Newman-Keuls test, and categorical data by a χ^2 test (performed on incidence rates within the total patient population undergoing mitral valve replacement), corrected if appropriate by Yates' formula or Fisher's exact test. Significance levels smaller than 0.05 were considered meaningful.

Results

Twelve xenografts featuring a dehiscence between the prosthetic part of the valve and the aortic wall in the commissural region were found. The bioprostheses were all in the mitral position and were implanted between May 1978 and November 1988, belonging to a series of 405 mitral Carpentier-Edwards bioprostheses implanted in our unit during this time frame. These 12 valves were part (13%) of the total of 92 mitral prostheses from this population that were explanted for any type of structural deterioration. The model of the valves was 6625 (standard Carpentier-Edwards bioprostheses) in eight patients and 6650 (supraannular Carpentier-Edwards bioprostheses) in four patients. No similar commissural detachments were found, at reoperation or at autopsy, in any of the 76 valves displaying structural deterioration from a cohort of 441 aortic Carpentier-Edwards bioprostheses implanted during the same time period.

There were nine female and three male patients,

Table I. Demographics and clinical data

Characteristic	n = 12
Age $\pm$ SD (years)	54 $\pm$ 13 (range 30-71)
Gender	
Female	9
Male	3
Risk factors for PTF	
Systemic hypertension	2
Chronic renal failure	1
Pregnancy	0
Origin	
Rheumatic	11 (1 rereplacement)
Unknown	1 (rereplacement)
Associated procedures	
Aortic valve replacement	7
Tricuspid annuloplasty	3
None	4

SD, Standard deviation; PTF, primary tissue failure.

Table II. Echocardiographic data

Variable	n = 11
Wall thickness (mm)	
Septum	11 $\pm$ 3
Posterior wall	11 $\pm$ 2
Ejection fraction (%)	64 $\pm$ 8 (range 57-84)
Mitral regurgitation	
Moderate	2
Moderate-severe	3
Severe	7
Mitral stenosis	1

with a mean age at explantation of 54 ± 13 years (Table I). The crude incidence of this complication was not significantly different for female patients (3.8%, 9/238) than for male patients (1.8%, 3/167). Only three patients had characteristics considered to be risk factors for primary tissue failure (one had chronic renal failure, and two had systemic hypertension). There were no pregnancies among the female patients. The original valve disease was rheumatic in all but one of the patients, and two patients received their Carpentier-Edwards valve as a second mitral bioprosthesis (Table I). Associated surgical procedures performed during the first operation included aortic valve replacement (seven patients) and tricuspid annuloplasty (three patients).

Echocardiographic data obtained before explantation of Carpentier-Edwards bioprostheses were available in 11 patients and are shown in Table II. The left ventricular systolic function was within the normal range in all patients, and 10 of 12 had a higher than moderate degree of mitral insufficiency.

Table III. Valve size, duration, and incidence of failure

	Size			
	27 mm (n = 0)	29 mm (n = 4)	31 mm (n = 4)	33 mm (n = 4)
Duration (mo)		89 $\pm$ 36	102 $\pm$ 23	106 $\pm$ 27
Incidence of failure (%)	0 (0/31)	3.1 (4/130)	2.6 (4/156)	4.7 (4/85)

One patient had a mixed mitral lesion with significant stenosis.

Mean duration of implantation of the valves with commissural aortic wall detachment exceeded 8 years (99 ± 27 months), with a range of 52 to 136 months. There were no statistically significant differences in duration among the valve size cohorts or in the proportions of valves of each size that failed (Table III). The duration of the bioprostheses also did not differ on the basis of model (110 ± 25 months for model 6625, 78 ± 21 months for model 6650; not significant). However, the mean age at implantation was significantly lower for patients with a model 6625 mitral bioprosthesis (48 ± 11 years versus 67 ± 6 years; $p < 0.05$). Distribution of failing valves by year of implantation showed a fairly constant crude incidence of failure between 1978 and 1988, with no significant differences between periods (Table IV).

Gross examination of the specimens (Table V) disclosed dehiscence of two commissures in two valves and of one commissure in 10 valves (Figs. 1 and 2). Further macroscopic lesions were evidenced in nine valves, consisting of mineral deposits that were combined with tears in eight instances. Three valves showed no other macroscopic abnormality apart from the commissural dehiscence. Close examination of the detached commissures showed complete integrity of the junction between the leaflets and the aortic wall and a scalloped edge of the dehiscent aortic wall (stamp effect; Fig. 2, A). The inner Dacron cover of the stent was readily visible in the affected commissures, and the outer aspect of the aortic wall was covered by a fibrinous sheath of variable thickness (Fig. 2, A). All valves displayed in their inflow aspect evident prolapse of one or two leaflets (Fig. 1).

Roentgenographic examination evidenced mineral deposits in the valve specimens that were considered minimal or absent in three patients, mild in five (Fig. 1, C), moderate in three, and severe in one (Fig. 1, F, Table VI). Detached commissures

Table IV. Year of implantation and incidence of failure

	Year					
	1978-79	1980-81	1982-83	1984-85	1986-87	1988
Incidence of failure (%)	2.6 (2/78)	2.9 (4/140)	2.6 (2/78)	4.3 (2/46)	2.6 (1/39)	4.2 (1/24)

showed no calcification at all in some cases and, most commonly, mineral concretions in the area of insertion of the leaflets with the surrounding aortic wall free from calcium (Fig. 1, C).

Light microscopy revealed a generalized reduction in aortic wall thickness in Carpentier-Edwards mitral prostheses (Table VII) when compared with either the reference porcine aortic roots ($p < 0.01$) or the aortic Carpentier-Edwards bioprostheses ($p < 0.01$). No statistically significant difference in mean wall thickness was found between intact or detached commissures of mitral bioprostheses. Porcine aortic roots used as a reference had significantly ($p < 0.01$) smaller wall thicknesses than degenerated aortic bioprostheses.

In the histologic study, the lack of adventitia and surrounding adipose tissue in the bioprostheses was evident. In the commissural areas, mineralization was most often evident along the insertion of the collagen bundles of the leaflets in the aortic wall. Detached commissures displayed different degrees of tissue edema and a well-developed fibroelastic layer covering the outer face of the aortic wall with abundance of erythrocytes, macrophages, and giant multinucleated cells. This fibroelastic sheath was thinner or even absent in nondehiscent commissures, although some degree of foreign body reaction tissue was present in all of them in the areas of interface between the biologic tissue and the Dacron cover or the suture material of the prosthesis.

Discussion

Primary tissue valve degeneration is a complex and incompletely understood phenomenon that involves the biologic component of heart valve bioprostheses, changes the biomechanical characteristics, and leads to the functional failure of the implant and, eventually, to reoperation or death of the host patient.¹ The process is not exclusive to bioprostheses and ultimately affects any biologic valve substitute, except for those made of autologous valvular tissue. Several factors, including patient characteristics, tissue origin, valve preservation procedure, calcium-mitigating treatments, and posi-

Table V. Macroscopic findings

Lesion	Number
Commissural dehiscence	
Single	10
Double	2
Tear and calcification	8

tion of implantation, influence both the precocity and intensity of the phenomenon, as has been proved clinically and experimentally.¹ However, failure of valvular bioprostheses related to manufacturing maneuvers and not to intrinsic degeneration of the tissue has not been a common finding in the valve industry era.

Failure of mitral Carpentier-Edwards bioprostheses because of detachment of the aortic wall from the stent post in the commissural area has previously been noted³⁻⁸ and identified as a mode of tearing unique to this particular bioprosthesis that constitutes a significant proportion of the attrition that results from structural deterioration.³ The Vancouver group⁴⁻⁷ has reviewed the problem, mainly from the clinical standpoint, and speculated on its relation with the manufacturer's procedure for tailoring the valves. Before 1986, the manufacturer trimmed the aortic wall near the commissures to increase the effective orifice area and improve hemodynamics. After this date (between 1986 and 1988), the trimming performed in these areas was reduced, initially in the large sizes but subsequently in all valves.

In our series, this phenomenon has been an exclusive feature of mitral Carpentier-Edwards bioprostheses and was not found in aortic bioprostheses. The reason for this clear-cut difference is probably multiple. Mitral prostheses are subjected to systolic hydraulic pressures and, hence, to higher mechanical stresses than aortic prostheses, which withstand diastolic pressures.^{9,10} On the other hand, systolic flow imposes relatively physiologic, axially oriented, flexion stresses on aortic porcine bioprostheses but abnormal, obliquely oriented stresses on mitral prostheses of the same origin.^{11,12} Furthermore, we are taking valves designed by nature for

Fig. 1. Macroscopic and radiographic findings in mitral Carpentier-Edwards bioprostheses showing dehiscence of the aortic wall from the stent in the commissural area. **A to C**, Bioprosthesis of size 31 mm implanted for 7.5 years in a woman who was 63 years old at implantation. **A**, Prolapse of one of the leaflets is evident in the inflow aspect. **B**, Dehiscence of the aortic wall from a stent post of the prosthesis is readily visible. The detached portion of the aortic wall displays an irregularly scalloped edge, probably caused by the "stamp effect." **C**, Mild radiologic mineralization is evident and involves the leaflet-aortic wall junction of the detached commissure but not the torn edge of the aortic wall (*arrowheads*). **D to F**, Bioprosthesis of size 33 mm implanted for 10.5 years in a woman who was 34 years old at implantation. **D**, Gross examination reveals a severe degeneration of the xenograft with rigidity, perforations, and severe mineralization that probably prevented a greater prolapse of the leaflets associated with the commissural dehiscence. **E**, The outflow view discloses detachment of two commissures. Part of a calcified perforated leaflet was lost during explantation. **F**, The roentgenogram shows diffuse and severe mineralization of the biologic tissue involving the commissures, central portion of the leaflets, and aortic wall.

Table VI. Radiologic mineralization

Severity	Number
Minimal or nil	3
Mild	5
Moderate	3
Severe	1

Table VII. Aortic wall thickness values

Type of valve	Wall thickness (mean $\pm$ SD in μ m)
Mitral Carpentier-Edwards bioprostheses ($n = 36$)	
Overall	351 $\pm$ 68*
Detached commissures ($n = 12$)	322 $\pm$ 42†
Intact commissures ($n = 24$)	366 $\pm$ 74
Aortic Carpentier-Edwards bioprostheses ($n = 12$)	526 $\pm$ 59‡
Porcine aortic roots ($n = 15$)	419 $\pm$ 50

SD, standard deviation.

* $p < 0.01$ versus aortic Carpentier-Edwards bioprosthesis or porcine aortic roots.

† p not significant versus intact commissures.

‡ $p < 0.01$ versus porcine aortic roots.

the aortic orifice and using them in the mitral position. On the other hand, we did find a significant difference in aortic wall thickness between mitral and aortic Carpentier-Edwards bioprostheses (Table VII), which suggests a less extensive trimming of the large aortic valves that may have prevented the weakening of their aortic wall.

Recently, Glower and coworkers¹³ from Duke University analyzed, with multivariable analysis, the determinants of reoperation for structural deterioration in a population of 447 mitral and 432 aortic Carpentier-Edwards bioprostheses. These authors found a significant correlation between increasing size of the valve and the incidence of this complication, but only for the mitral valve position. The fact that aortic valves are on average smaller than mitral valves may favor the difference observed by these authors in terms of reoperations for tissue failure. However, the commissural detachment phenomenon must not be identified with classic primary tissue failure, even though both probably share some pathogenic factors. We were not able to find a similar trend in our series of patients, when comparing the mean durability of valves of different sizes, and therefore have no evidence that larger valves are at higher risk for aortic wall commissural detachment. On the other hand, the number of events in our experience is too small for differences to reach statistical significance. Glower and col-

Fig. 2. Detailed view of the commissural areas in mitral Carpentier-Edwards bioprostheses showing the aortic wall detachment phenomenon. **A**, Detached commissure of a 33 mm mitral bioprosthesis that was implanted for almost 6 years in a woman who was 41 years old at implantation. The “stamp effect” is evident in the torn edge of the aortic wall. The bare Dacron cloth from the inner aspect of the stent is visible, and there is a fibroelastic sheath covering the outer aortic wall. **B**, Close-up view of two detached commissures from the bioprosthesis shown in Fig. 1, **D** to **F**. The desuspension of the commissures, resembling aortic dissection, is evident.

leagues¹³ also reported that 35 of 44 patients with a failing mitral Carpentier-Edwards bioprostheses larger than 29 mm had pure mitral regurgitation, whereas this condition occurred in only 8 of 21 with valves smaller than 31 mm (χ^2 test 95% confidence level 3.841, $p < 0.01$). In view of our findings, and considering that both Glower’s and our patients received their valves in a similar time frame, it could be suspected that some of their patients with failing large mitral bioprostheses could actually have an aortic wall commissural dehiscence.

Female gender has been identified, in several

recent long-term follow-up studies of large numbers of patients, as a significant predictor of structural deterioration of porcine bioprostheses,¹⁴ particularly in the mitral position.^{4, 15} The crude incidence found in this report of the commissural detachment complication was higher in female patients with a Carpentier-Edwards bioprosthesis, but the difference was not significant because of the small number of instances. Such a comparison should probably be made using actuarial or logistic regression analysis, on a larger population of valves, and with more cases of detachment.

The echocardiographic findings disclosed a normal or increased left ventricular function and a mild increase in ventricular wall thickness (see Table II). These data, together with the relatively high prevalence of aortic valve disease necessitating valve replacement (7 of 12 patients), suggest that the commissural dehiscence may have appeared first in those patients whose mitral prostheses sustained the highest systolic leaflet stresses.

Our data from gross examination, radiology, and light microscopy seem to identify a weakened aortic wall as the main cause of the detachment. We found a significant difference in aortic wall thickness at the paracommissural level between normal porcine aortic roots, conditioned in a manner similar to conventional bioprostheses, and mitral Carpentier-Edwards bioprostheses. Moreover, this difference is probably underestimated because some manufacturing factors (Dacron cover) and postimplantation changes (inflammation, local mineralization) of the bioprosthesis tend to increase the aortic wall thickness. Further, histologic examination disclosed the lack of adventitia and, probably, part of the media.

Another factor that could play a role in the pathogenesis of this type of complication is the internal tension generated during the assemblage of the bioprosthesis because of the stent/tissue mismatch. These tensions are transmitted to the aortic wall by the leaflets and are more distally applied on the stent posts and more intense as the mismatch increases.¹⁶

A thinned, noncalcified aortic wall would start detaching from the inner Dacron cover of the stent over the commissure with maximal mechanical stress of the valve—both passive stress caused by stent/tissue mismatch and active stress caused by valve position, size of leaflets, and so on. The initial dehiscence would promote an increase in the mechanical activity in this area that would facilitate a “stamp effect” along the line of suture of the

weakened aortic wall. Desuspension of the commissure would induce prolapse of one or two leaflets and significant regurgitation. Also, turbulence in this area would favor fibrin deposition on the outer aortic wall.

Calcification of the porcine aortic wall is a common finding with clinically explanted bioprostheses^{17, 18} and in many cases bears no functional significance. It could be argued, however, that mineralization of the aortic wall remnant in the commissural area of the Carpentier-Edwards bioprosthesis could have favored in our series the appearance of a dehiscence between it and the stent. The roentgenographic study did show some aortic wall calcification, but it involved the dehiscent portions of the aortic wall only in the heavily calcified valves. In most valves, there was either minimal or no radiologically detectable mineralization, or the concretions were mild and, across the commissural junction, in continuity with some calcific streaks of the leaflets. This pattern has previously been described as a form of early mineralization of porcine bioprostheses, and it closely follows the distribution of the maximal mechanical stress concentration areas.¹⁸⁻²⁰

The reason why a particular commissure in a valve, and not any of the others, tears off is not clear. We measured aortic wall thickness in both intact and detached commissures and found no significant difference, which rules out the possibility of a more aggressive trimming of one commissure with asymmetric weakening of the aortic wall. Other factors probably play a role, including comparative leaflet size (the larger leaflets and their commissures holding higher membrane mechanical stresses), the position of implantation of each leaflet (because the systolic stress is asymmetric), and an increased stent/tissue mismatch at this particular point.

Addendum

During the review process of this article, two more Carpentier-Edwards mitral bioprostheses that were explanted in our unit showed commissural dehiscence. The patients (a man and a woman aged 50 and 62 years at implantation) belong to the same series mentioned above. The valves were sizes 29 and 31 mm and models 6625 and 6650. The detachment affected one commissure in one valve and two commissures in the other. The duration of implantation of the valves was 128 and 111 months.

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Commentary

We wish to thank the Editor for allowing us to respond to this excellent article by Nistal and associates. In 1991 Jamieson and colleagues^{1,2} identified the failure mode of stent dehiscence that resulted in release of porcine tissue from a strut, causing prolapse of two leaflets and valvular regurgitation in Carpentier-Edwards porcine bioprostheses. Before these reports the Edwards CardioVascular Surgery (CVS) Division of Baxter Healthcare Corporation reviewed the cause of this complication and found that a manufacturing factor, the extensive degree of trimming of the outer layers of the aorta, was probably responsible for commissural stent dehiscence in both standard and supraannular valve models. The involved valves were almost exclusively large mitral sizes and the dehiscence usually occurred at the commissure located between the two largest cusps. Reduced trimming during valve manufacture was initiated for 29 to 35 mm mitral valves in 1986. Reduced trimming was extended to include all valve sizes, both aortic and mitral, in January of 1988. No reports of commissural stent dehiscence in valves manufactured since the process change have been received. Jamieson and colleagues^{1,2} observed their first incident approximately 4 years after implantation, and eight cases were observed in their patient population. More recently, this group of investigators compared the incidence of stent dehiscence in valves before and after reduced trimming.³⁻⁵ Commissural stent dehiscence was identified as a cause of structural valve dysfunction in 20 of 931 trimmed mitral prostheses and in 0 of 207 reduced trimming prostheses. Christie and Barratt-Boyes⁶ recently presented data suggesting that the forces transmitted to the aortic wall by the leaflets increased in intensity and

migrated up the stent post as the degree of stent/tissue mismatch increased. On the basis of these observations the time to failure by tissue detachment from the stent post would be linearly dependent on wall remnant thickness, the resultant force on the sutures, and the suture pull-out resistance of the tissue. Such observations may answer the concluding question of Nistal and associates, "why a particular commissure in a valve, and not any of the others, tears off. . . ." Christie and Barratt-Boyes⁶ also concluded that the practice of thinning the aortic wall remnant would decrease the time to failure. The published observations of Jamieson and colleagues¹⁻⁵ and our unpublished observations support this conclusion.

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