PULMONARY HOMOGRAFT: SHOULD IT BE USED IN THE AORTIC POSITION?

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Objective: Retrospective analysis was performed to determine the suitability of pulmonary homograft as an aortic valve substitute.

Methods: From January 1994 through June 1999, 147 patients (mean age, 32.2 ± 17.3 years) underwent aortic valve replacement with either an aortic homograft (group 1: $n = 103$, 25 fresh antibiotic preserved and 78 cryopreserved) or a pulmonary homograft (group 2: $n = 44$, 11 antibiotic preserved and 33 cryopreserved). In group 1 a scalloped subcoronary technique was used in 64 patients, and a root replacement technique was used in 39 patients. In group 2 the scalloped subcoronary technique was used in 34 patients, and the root replacement technique was used in 10 patients.

Results: There were 131 operative survivors (group 1 = 91; group 2 = 40). Follow-up ranged from 2 to 62 months. In group 1 none of the patients had significant aortic regurgitation during the hospital stay. Three patients (all having undergone the scalloped subcoronary technique) had moderate aortic regurgitation after 6 to 32 months. In group 2, 10 patients (9 having undergone the scalloped subcoronary technique and 1 having undergone the root replacement technique) developed significant regurgitation: 2 intraoperatively, 5 in the early postoperative period before discharge from the hospital, and 3 during late follow-up 6 to 12 months postoperatively. Among the various risk factors analyzed for overall homograft failure, use of a pulmonary homograft was the single independent predictor of valve failure (odds ratio, 8.6; 95% confidence interval, 1.9-39; $P = .006$).

Conclusion: Pulmonary homograft, when inserted by means of a scalloped subcoronary technique, is not a suitable aortic valve substitute. (J Thorac Cardiovasc Surg 2000;120:148-55)

With reports of excellent results, the suitability of aortic valve replacement with an aortic valve homograft is well established.¹⁻³ However, limited availability of the aortic homografts is an important factor for their restricted use. Pulmonary homograft was considered an alternative substitute to the diseased aortic valve,⁴ but its use was abandoned after initial trials.^{5,6} Considering the excellent long-term results of pulmonary autografts in the aortic position,^{7,8} which are further supported by the

experienced work showing minimal differences between pulmonary and aortic homografts,⁹ and considering that the pulmonary valve can withstand the higher mechanical stress in systemic circulation,¹⁰ investigators had a renewed interest in pulmonary homografts.¹¹⁻²⁰ However, the use of pulmonary homografts remained in dispute because various investigators reported contrasting results. We started using pulmonary homografts for aortic valve replacement in 1994, and in the present retrospective study we analyze our results to ascertain their suitability.

Patients and methods

Patients. A total of 147 patients (101 male subjects) with an age range from 5 to 68 years (mean, 32.2 ± 17.3 years) underwent aortic valve replacement with either an aortic (group 1, $n = 103$) or pulmonary homograft (group 2, $n = 44$). The causes of aortic valve disease and associated lesions are shown in Tables I and II. All the patients were assigned to New York Heart Association (NYHA) class III or IV, and 26 patients had frank congestive heart failure.

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Table I. Cause of aortic valve disease in patients undergoing aortic valve replacement with an homograft
(*n* = 147)

Cause	Group 1 (<i>n</i> = 103)		Group 2 (<i>n</i> = 44)		Total (<i>n</i> = 147)	
	No.	%	No.	%	No.	%
Rheumatic heart disease	69	67	36	80	105	71
Bicuspid aortic valve	12	12	2	7	14	10
Degenerative aortic valve disease	14	14	3	7	17	12
Prosthetic valve endocarditis	4	4	2	5	6	4
Marfan syndrome	2	2	0	0	2	1
Annuloaortic ectasia	1	1	1	2	2	1
Ascending aortic aneurysm with aortic regurgitation	1	1	0	0	1	1
Total	103	100	44	100	147	100

Table II. Additional significant preoperative morbid conditions (*n* = 147)

Associated morbid condition	Group 1 (<i>n</i> = 103)		Group 2 (<i>n</i> = 44)		Total (<i>n</i> = 147)	
	No.	%	No.	%	No.	%
Significant mitral valve disease	43	42	22	50	65	44
Significant tricuspid valve disease	6	6	1	2	7	5
Left ventricular dysfunction (ejection fraction <40%)	41	40	16	36	57	39
Ischemic heart disease	6	6	0	0	6	4
Native aortic valve endocarditis	9	9	4	9	13	9
Previous cardiac procedure (CMV, BMV, AVBD)	16	16	4	9	20	14
Previous aortic valve operation	9	9	3	7	12	8
Diabetes mellitus	8	8	4	9	12	8
Atrial septal defect	1	1	0	0	0	1
Supravalvular aortic stenosis	1	1	0	0	1	1
Pseudoaneurysm of ascending aorta	1	1	0	0	1	1

CMV, Closed mitral valvotomy; BMV, balloon mitral valvotomy; AVBD, aortic valve balloon dilatation.

Twelve patients had previously undergone various operations for the aortic valve (prosthetic aortic valve replacement in 6, bioprosthesis-homograft aortic valve replacement in 3, and aortic valve repair in 3).

Homograft preparation and preservation. The majority of the homografts (*n* = 141) were obtained from cadaveric donors. Six homografts were procured either from cardiac transplant recipients or from multiorgan donors not suitable for cardiac transplantation. The donor age ranged from 15 to 48 years. From cadaveric donors, hearts were procured within 24 hours of death, and valves were dissected under sterile conditions. Homografts were treated with antibiotic solution²¹ for 48 hours at 4°C. In initial experience, antibiotic-preserved homografts were used directly within 40 days. In the latter part of our experience, the homografts were cryopreserved in a preservative solution consisting of RPMI-1640 tissue culture medium (Roswell Park Memorial Institute tissue culture medium 1640) with 10% fetal calf serum and 10% dimethylsulfoxide. After sterile packaging, the homografts were frozen gradually (−1°C/min) up to −80°C and then were stored in the vapor phase of liquid nitrogen at a temperature between −150°C and −190°C.

Surgical technique. Intraoperative transesophageal echocardiography was performed in all the patients and, depending on aortic root diameter, an appropriately sized homograft was selected. Patients with an aortic root diameter greater than 30 mm received a prosthetic valve. A standard moderately hypothermic or normothermic cardiopulmonary bypass with antegrade, cold, hyperkalemic blood cardioplegia was used in all the cases.

Among patients in group 1, in 64 patients a scalloped subcoronary implantation technique, as described by Ross,²² was used. In 35 patients the root replacement technique of Elkins²³ was used. In 4 patients both the diseased aortic valve and the ascending aorta were replaced by a valved homograft conduit. Among patients in group 2, the scalloped subcoronary technique was used in 34 patients, the root replacement technique was used in 9 patients, and a valved homograft conduit was used in 1 patient. For the purpose of analysis, patients in whom a valved homograft conduit was used were included in the root replacement group. In group 1, 25 patients received an antibiotic-preserved valve, and 78 patients received a cryopreserved valve. In group 2, 11 patients received an antibiotic-preserved homograft, and

Table III. Additional procedures performed in patients undergoing aortic valve replacement with a homograft (n = 147)

Additional procedure	Group 1 (n = 103)		Group 2 (n = 44)		Total (n = 147)	
	No.	%	No.	%	No.	%
Mitral valve repair	24	23	15	34	39	27
Open mitral commissurotomy	20	19	6	14	26	18
Tricuspid valve repair	6	6	1	2	7	5
Coronary artery bypass grafting	6	6	0	0	6	4
Redo mitral valve replacement with a bioprosthesis	1	1	0	0	1	1
Repair of pseudoaneurysm of ascending aorta	1	1	0	0	1	1
Closure of atrial septal defect	1	1	0	0	1	1

33 patients received a cryopreserved homograft. In 73 patients 81 additional procedures were performed (Table III).

At the completion of the procedure, cardiopulmonary bypass was discontinued, and transesophageal echocardiography was performed to assess the valve function.

Follow-up and assessment of valve function. A transthoracic echocardiographic evaluation was carried out 5 to 7 days postoperatively before discharge from the hospital. Subsequently, patients were followed up in the outpatient department, and transthoracic-transesophageal echocardiography was performed 1, 3, and 6 months postoperatively and then at every 6 months. On echocardiography, aortic regurgitation was graded according to published criteria.²⁴ Significant aortic regurgitation was considered with aortic regurgitation of grade III or IV severity. Valve-related events were reported as per prescribed guidelines.²⁵

Statistical analysis. Continuous or interval-related variables were expressed as mean values $\pm$ SD. Categorical variables were expressed as percentages. Groups were compared by the χ^2 and Student *t* tests. Actuarial estimates have been calculated by using the Kaplan-Meier technique.²⁶

The incidence of homograft failure was examined in relation to a variety of variables (Appendix I). The native anulus sizes and homograft sizes were also categorized (ie, <25 mm and $\geq$ 25 mm). Crude relative risks (RRs) with 95% confidence intervals (CIs) were computed. Logistic regression models based on the generalized estimating equations described by Liang and Zeger²⁷ were used to generate adjusted odds ratios as estimates of adjusted RRs with appropriate 95% CIs. Variables were included in the final model for adjustment if their presence in the logistic models altered the crude RR by at least 10%, except for age, year of surgery, cardiopulmonary bypass time, and aortic crossclamp times, which were included in all models. Statistical analysis was performed with the use of the SAS system (SAS, Inc, Cary, NC).

Results

Early results

Early mortality. In group 1, there were 12 early deaths (12%). Cause of death included low-output syndrome (n = 4), intractable ventricular arrhythmia (n = 2), pro-

gressive left ventricular dysfunction (n = 3), infective endocarditis (n = 2), and catastrophic hemorrhage (n = 1). In group 2, 4 (9%) patients died during the hospital stay, and the causes included low-output syndrome (n = 1), ventricular arrhythmia (n = 1), infective endocarditis (n = 1), and progressive left ventricular dysfunction (n = 1).

Survivors. Among survivors, 91 (69%) patients received an aortic homograft (23 antibiotic-preserved and 68 cryopreserved homografts), and 40 (31%) patients received a pulmonary homograft (10 antibiotic-preserved and 30 cryopreserved homografts). Profiles of these patients are shown in Table IV.

Valve function. None of the patients who received an aortic homograft had significant regurgitation during the hospital stay.

Two patients who underwent aortic valve replacement with a cryopreserved pulmonary homograft placed by means of a scalloped subcoronary technique had significant aortic regurgitation intraoperatively, as detected by transesophageal echocardiography. In both patients the homograft was replaced with a mechanical prosthesis. In addition, 2 more patients (patient 5 and 7, Table V) showed signs of mild aortic regurgitation on intraoperative transesophageal echocardiography. In the remaining patients there was no aortic regurgitation.

At the time of discharge, 5 patients had moderate to severe aortic regurgitation. All of these patients received a pulmonary homograft by means of a scalloped subcoronary technique. The profile of patients with early valve failure is shown in Table V. Two of these patients (patients 5 and 7, Table V) had a large aortic anulus, with diameters of 28 and 30 mm, respectively. Two other patients had mild aortic regurgitation at the time of discharge.

Late results. Follow-up ranged from 2 to 62 months (mean follow-up: group 1, 24.5 ± 13.2 months; group 2, 23.4 ± 12.9 months) and was 97% complete.

Table IV. Profile of operative survivors (*n* = 131)

Variable	Homograft		Aortic vs pulmonary
	Aortic (<i>n</i> = 91)	Pulmonary (<i>n</i> = 40)	
Mean age ± SD (y)	34.4 ± 17.3	29.0 ± 17.4	<i>t</i> = 1.6, <i>P</i> = .7
Mean follow-up ± SD (mo)	24.5 ± 14.2	23.4 ± 12.9	<i>t</i> = 0.4, <i>P</i> = .7
CPB time (min)	128.4 ± 34.7	130.1 ± 26.6	<i>t</i> = 0.3, <i>P</i> = .6
Aortic crossclamp time (min)	101.3 ± 26.9	105.4 ± 21.1	<i>t</i> = 0.8, <i>P</i> = .5
Cause			
Rheumatic	68	34	χ^2 = 1.6, <i>P</i> = .3
Nonrheumatic	23	6	
Physiologic lesion			
Aortic stenosis	26	6	χ^2 = 2.1, <i>P</i> = .2
Aortic regurgitation	65	34	
Aortic annulus size			
<25 mm	77	31	χ^2 = 0.5, <i>P</i> = .5
≥25 mm	14	9	
Left ventricular dysfunction (ejection fraction < 40%)	32	12	χ^2 = 0.1, <i>P</i> = .7
Mitral valve disease	40	19	χ^2 = 0.0, <i>P</i> = 1.0
Preoperative endocarditis	9	6	χ^2 = 0.3, <i>P</i> = .6
Reoperation	6	3	χ^2 = 0.0, <i>P</i> = 1.0
Preservation			
Antibiotic preserved	23	10	χ^2 = 0.0, <i>P</i> = 1.0
Cryopreserved	68	30	
Technique			
Scalloped subcoronary	56	31	χ^2 = 2.5, <i>P</i> = .1
Root replacement	35	9	

CPB, Cardiopulmonary bypass.

Table V. Profile of operative survivors who had early valve failure

Patient			Cause	Lesions	Type of	Preservation of	Technique	Outcome
No.	Age (y)	Sex			homograft	homograft		
1	62	M	Rheumatic	AR	Pulmonary	Antibiotic	SS	Moderate AR at discharge, severe AR after 2 mo, death
2	15	F	Rheumatic	AR	Pulmonary	Antibiotic	SS	Moderate AR at discharge, severe AR at 6 mo
3	20	F	Rheumatic	AR	Pulmonary	Antibiotic	SS	Severe AR at operation, replacement with mechanical prosthesis
4	15	F	Rheumatic	AR	Pulmonary	Cryopreservation	SS	Severe AR at operation, replacement with mechanical prosthesis
5	15	M	Rheumatic	AR	Pulmonary	Cryopreservation	SS	Moderately severe AR at discharge, reoperation after 18 mo
6	12	M	Rheumatic	AR	Pulmonary	Cryopreservation	SS	Moderate AR at discharge, moderate AR at 18 mo
7	19	F	Rheumatic	AR	Pulmonary	Cryopreservation	SS	Moderate AR at discharge, severe AR after 6 mo

AR, Aortic regurgitation; SS, scalloped subcoronary.

Late mortality. In group 1 there were 3 (3%) late deaths. Two patients died 3 and 5 months postoperatively as a result of *Aspergillus*-induced endocarditis. Another patient died as a result of progressive left ventricular dysfunction after 24 months.

There were 4 (9%) late deaths in group 2. One patient (patient 1, Table V) died after 2 months as a result of congestive heart failure. Mediastinitis with secondary hemorrhage was the cause of death in 1 patient. Endocarditis (*Aspergillus*-induced endocarditis in 1, and *Staphylococcus aureus*-induced endocarditis in the other) was the cause of late mortality in 2 patients.

Valve-related events and clinical status. There were 3 reoperations. Two patients (1 in group 1 and 1 in group 2) were reoperated on for fungal endocarditis (*Aspergillus*) 6 and 5 months postoperatively, respectively. Both the patients died as a result of overwhelming fungal infection.

One patient (patient 5, Table V) developed severe aortic regurgitation after 6 months and was reoperated on after 18 months. The pulmonary homograft was replaced with a mechanical prosthesis. At reoperation, valve cusps were found to be thin and transparent. There was no coaptation with each other. All the cusps

Follow-up in Months	0	12	24	36	48	60
Aortic Homograft						
Patients at Risk	91	73	35	16	5	2
Freedom from AR (% ± SE)	100	99±1.1	97.5±1.7	93.8±4	93.8±4	93.8±4
Pulmonary Homograft						
Patients at Risk	40	23	9	5	2	0
Freedom from AR (% ± SE)	100	73.1±7.4	73.1±7.4	73.1±7.4	73.1±7.4	73.1±7.4

Abbreviations: AR: Aortic Regurgitation
SE: Standard Error

Fig 1. Probability of freedom from significant aortic regurgitation (Kaplan-Meier) in operative survivors who either received an aortic or a pulmonary homograft.

Table VI. Profile of patients who had late valve failure

Patient No.	Age (y)	Sex	Cause	Lesion	Type of homograft	Preservation of homograft	Technique	Comments
1	25	M	Bicuspid aortic valve	AR	Pulmonary	Antibiotic	Scalloped subcoronary	Moderate AR after 12 mo
2	25	M	Rheumatic	AR	Aortic	Cryopreservation	Scalloped subcoronary	Moderate AR after 32 mo
3	17	M	Rheumatic	AR	Pulmonary	Antibiotic	Scalloped subcoronary	Mild AR at discharge, moderate AR at 6 mo
4	19	M	Bicuspid aortic valve	Aortic stenosis	Aortic	Cryopreservation	Scalloped subcoronary	Moderate AR after 12 mo
5	12	M	Rheumatic	AR	Pulmonary	Cryopreservation	Root replacement	Mild AR at discharge, moderate AR at 12 mo
6	24	F	Rheumatic	AR	Aortic	Cryopreservation	Scalloped subcoronary	Moderate AR after 14 mo

AR, Aortic regurgitation.

were intact. Microscopically, the endothelial and mesenchymal cells were characteristically absent, and only the fibrous skeleton of the valve was left in situ.

Clinically, 102 patients are currently in NYHA class I, and 12 are in class II (left ventricular dysfunction in 8, significant mitral regurgitation in 2, and significant aortic regurgitation in 2). Six patients are in NYHA class III (left ventricular dysfunction in 5 and severe mitral regurgitation in 1).

Valve function. In patients with moderate aortic regurgitation at discharge, the severity increased, and 4 of them had severe aortic regurgitation over a period of 2 to 6 months (1 underwent a second operation and 1 died). In 1 patient (patient 6, Table V) the degree of aortic regurgitation remained the same after a follow-up of 18 months.

An additional 6 patients (3 in group 1 and 3 in group 2) had significant aortic regurgitation over a follow-up

Table VII. Univariate analysis of various risk factors for homograft failure

Variable	n	Homograft failure	Crude RR	95% CI	P value
Sex					
Male	86	9	1.2	0.3-5	.8
Female	45	4			
Cause					
Rheumatic	102	11	1.6	0.3-60	.8
Nonrheumatic	29	2			
Lesion					
Aortic stenosis	32	1	0.2	0.02-1.9	.3
Others	99	12			
Left ventricular dysfunction	44	6	1.8	0.5-6.5	.5
Mitral valve disease	64	5	0.6	0.2-2.3	.6
Previous procedure	17	3	2.2	0.2-10.5	.5
Aortic anulus					
≥25 mm	23	3	1.4	0.02-6.6	.8
<25 mm	108	10			
Preservation of homograft					
Antibiotic preserved	33	5	2.0	0.7-7.5	.4
Cryopreserved	98	8			
Technique					
Scalloped subcoronary	87	12	6.9	0.8-60	.07
Root replacement	44	1			
Type of homograft					
Pulmonary	40	10	9.7	2.3-35	.0005
Aortic	91	3			

of 6 to 36 months. Profiles of these patients are shown in Table VI. Two of these patients (patients 3 and 5, Table VI) had mild aortic regurgitation at the time of discharge.

Freedom from significant aortic regurgitation in operative survivors who received either an aortic or a pulmonary homograft is shown in Fig 1.

Various risk factors were analyzed for overall homograft failure (Appendix I). In univariate analysis (Table VII) the use of a pulmonary homograft was a significant risk factor for valve failure (crude RR, 9.7; 95% CI, 2.3-35; $P = .0005$). The mean age of patients with valve failure (21.5 ± 13 years) was significantly less than that of those without valve failure (34 ± 17.5 years; $t = 2.5$; $P = .005$). However, in multiple regression analysis, use of a pulmonary homograft was the single independent predictor of valve failure (odds ratio, 7.4; 95% CI, 1.7-34; $P = .007$). Although the majority of patients with valve failure had undergone aortic valve replacement with a scalloped subcoronary technique, a statistical correlation could not be established because of the small number of patients.

Discussion

In our experience the use of a pulmonary homograft to replace the diseased aortic valve was associated with

a significantly higher risk of valve failure. Although other factors, such as age, cause, physiologic lesion, use of amphotericin in homograft treatment, and surgical technique,^{3-5,20} have also been considered important factors for homograft failure, we did not find any correlation between these factors and the risk of homograft failure.

Investigators have found that the ultrastructure of the pulmonary homograft cusps and that of the aortic homograft cusps is the same, with no substantial ultrastructural difference.^{9,15} Although the pulmonary leaflets are thinner than the aortic leaflets, with a lesser content of elastic tissue in the ventricular layer, the ultimate tensile strength was similar for both the aortic and pulmonary valves.¹⁵ Gorczynski and colleagues¹⁰ have also demonstrated that breaking strain for the aortic and pulmonary valve leaflets is similar because even though the pulmonary leaflets are thinner, they do have greater tensile strength than the aortic leaflets. Pragliola and colleagues¹⁷ have further demonstrated a larger coapting surface with a pulmonary valve than with an aortic valve. Despite these favorable morphologic and mechanical characteristics, investigators^{18,28} were reluctant to use pulmonary valves in the systemic circulation because of concern about the durability of the pulmonary structures subjected to systemic pres-

sure. Although both clinical and experimental studies^{7,8,10} have shown the suitability of the pulmonary autografts in systemic circulation, the results could not be reproduced with the pulmonary homografts. Lack of antigenicity and absolute vitality of the autograft may be the chief contributors to the excellent results obtained by use of an autograft. However, another significant difference appears to be the use of different techniques for insertion of a pulmonary autograft and a pulmonary homograft. In recent decades, the root replacement technique is the preferred technique for autograft insertion, whereas most of the investigators have used either a scalloped subcoronary technique or intra-aortic cylinder technique for pulmonary homograft insertion.

Similar to the experience of others,^{14,15,17,20} we also had a high failure rate with pulmonary homografts inserted by means of a scalloped subcoronary technique. There were fewer failures when a root replacement technique was used, which was also evidenced by others.¹⁷ We believe that because of the delicate and frail structure of the pulmonary homograft, it is very difficult to maintain the normal geometry of the pulmonary homograft during intra-aortic implantation, by using either the scalloped subcoronary or cylinder technique. The same belief is shared by others.^{17,20} This is evident from early failures of implanted pulmonary homografts in our experience and the experience of other investigators.^{4,15-20} In most of these reports^{4,15-20,29} either prolapse of one of the cusps or central regurgitation as a result of altered geometry was responsible for the failure of the pulmonary homograft.

Besides early failure, 3 of our patients who had received a pulmonary homograft had late valve failure. Two of these (patients 3 and 5, Table VI) had mild aortic regurgitation at the time of discharge. Thus these failures may also be considered results of the altered geometry of the homograft, leading to progressive aortic regurgitation. Other investigators have found intrinsic valve failure in the form of cusp rupture or cusp perforation as a cause of late homograft failure.^{5,20,30} Although pulmonary valves are identical to aortic valves in respect to ultrastructural and mechanical qualities, intrinsic structural failure is reported more commonly and after a shorter interval with a pulmonary homograft. Mair and colleagues²⁰ postulate that because of the delicate nature of the pulmonary homograft, minor alterations in the geometry of the valve do occur at the time of intra-aortic implantation. These changes are so subtle that they are overlooked easily and may not necessarily produce graft incompetence in the early phase but lead to

minimal unphysiologic stress to the cusps. This minimal unphysiologic stress, in a subsequent time course, can cause material fatigue and, consequently, structural failure of the valve.

The size of the used homograft is another important consideration. Because the pulmonary homografts are inherently larger than the aortic homografts, they might be selected for implantation in the large, often dilated anulus, the pathology of which would favor continued dilatation and early homograft failure. Two of our patients (patients 5 and 7, Table V) had a large anulus (28 and 30 mm, respectively), and homografts with one size smaller than the aortic root were inserted, as is our practice during insertion of an aortic homograft. Both these homografts failed as a result of central incompetence. Others^{11,15} have suggested a pulmonary homograft slightly larger than the aortic root to counteract this. They believe that minor changes in homograft geometry at the time of insertion are neutralized by the choice of a large homograft due to availability of a larger coaptation surface. Another option is reducing and remodeling the aortic anulus³¹⁻³³ at the time of implantation of the homograft. However, we believe that in patients with a large aortic anulus, pulmonary homograft valve should not be used, and if required, a root replacement technique with a homograft slightly larger than the native root is a better option in such conditions.

To conclude, we believe that a pulmonary homograft, when inserted by means of a scalloped subcoronary technique, is not a suitable aortic valve substitute. Although the root replacement technique appears safe, a larger and longer study is required to judge its suitability.

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Appendix 1. List of variables analyzed as risk factors for homograft failure

Preoperative variables

- Age
- Sex
- Cause: rheumatic versus nonrheumatic
- Physiologic lesion: aortic stenosis versus others
- Associated mitral valve disease
- Left ventricular dysfunction
- Endocarditis
- Previous cardiac procedure
- Previous aortic valve surgery
- Aortic anulus (<25 mm vs ≥25 mm)

Preoperative variables

- Year of operation (1993-1999)
- Type of homograft: aortic versus pulmonary
- Preservation of homograft: cryopreserved versus antibiotic preserved
- Technique of graft insertion: scalloped subcoronary versus root replacement
- Cardiopulmonary bypass time
- Aortic crossclamp time
- Homograft size (<25 mm vs ≥25 mm)