Older Patients Fare Better With the Ross Operation

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Background. **The Ross operation has an established position in young patients. We address the question of whether any age group profits most from the Ross operation, and we compare the results in various ages.**

Methods. **From February 1995 to August 2001 we performed 250 Ross operations. Group 1 consisted of 46 patients, ages 2 to 25 years (median age, 15 years). Group 2 consisted of 123 patients, ages 26 to 49 years (median age, 39 years). Group 3 consisted of 81 patients, ages 50 to 67 years (median age, 55 years). Echocardiography was performed perioperatively, at 2 to 6 months, and then yearly.**

Results. **Mean follow-up for the three groups was 32, 31, and 28 months, respectively ($p = 0.36$). One patient from group 2 died after 25 months caused by suppurative pneumonia and 3 patients from group 3 died (1 from suspected acute thoracic aorta dissection at 40 months, 1 from ventricular fibrillation after 25 months, and 1 from an undiagnosed sudden death at 5 months). Autograft replacement was necessary for 3 patients from group 2 and 1 from group 3. Autograft repair was necessary for 1 patient from group 2, and pulmonary homograft reoperation was necessary for 1 patient from group 1. All other autografts currently have physiologic gradients and clinically insignificant regurgitation. Median peak gradient across the right ventricular outflow tract was 23.6 ± 18 mm Hg for group 1, 14.6 ± 8 mm Hg for group 2, and 11.5 ± 7 mm Hg, which was significantly lower for group 3 patients ($p < 0.001$). Eleven patients are under close follow-up for right ventricular outflow tract gradients ≥ 40 mm Hg; eight of these patients are from group 1, 3 are from group 2, and there are none from group 3.**

Conclusions. **Although the Ross operation provides excellent results in all age groups, the problem of right ventricular outflow tract stenosis has not been seen in patients older than 50 years, which implies that it offers superior results for aortic valve disease in middle aged and older patients.**

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Aortic valve replacement with the pulmonary autograft, or the so-called Ross Operation, has achieved an established position in children, adolescents, and young adults [1–3]. The underlying reasons for the increasing acceptance of the Ross procedure are the growth potential of the pulmonary autograft demonstrated when used as an aortic valve substitute in children [4, 5], the excellent physiologic hemodynamic properties [6], and the absence of any anticoagulation. These factors allow a near normal quality of life after the Ross operation and make it an interesting alternative for older patients with active and sportive lifestyles or with contraindications to lifelong anticoagulation.

Since the introduction of the Ross operation at our institution in February 1995, this procedure has been performed in increasingly older patients. Subsequently, in light of the limited supply of homografts the question arose of whether this complex and demanding operation should be reserved for young patients and whether it was justified for older patients. Therefore we compared the results of different age groups.

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Patients and Methods

Patients

From February 1995 to August 2001 we performed the Ross operation in 250 patients, aged 2 to 67 years. All patients were classified into age groups. Group 1 consisted of younger patients less than or equal to 25 years of age, whereas group 2 consisted of middle-aged patients older than 25 years and younger than 50 years of age. Group 3 consisted of older patients 50 years of age and older. The detailed group characteristics and underlying aortic valve pathology are shown in Table 1.

Surgical Technique

Mild hypothermic bypass and near-continuous cold antegrade and retrograde blood cardioplegia were used in all patients. The pulmonary autograft and cryopreserved pulmonary homograft to reconstruct the right ventricular outflow tract were both implanted as freestanding roots, using previously published detailed standardized techniques [7]. All operative details and concomitant procedures are listed in Table 2.

Echocardiography

Follow-up consisted of in-house clinical examination and transthoracic echocardiography (two-dimensional, M-mode, color flow and Doppler flow echocardiography) at

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Table 1. Detailed Group Characteristics and Underlying Aortic Valve Pathology

	p[a]	Group 1	p (Group 1 Vs 2)	Group 2	p (Group 2 Vs 3)	Group 3	p (Group 1 Vs 3)
No. of patients		46		123		81	
Age[b] (y)		14.8 ± 5		39.2 ± 7		54.6 ± 3	
Gender ratio (male:female)	0.603	34:12		97:26		66:15	
Body weight[c] (kg)	< 0.001	54 (11,5–98)	< 0.001	80 (50–116)	0.441	80 (54–105)	< 0.001
Height[c] (cm)	< 0.001	164 (86–186)	< 0.001	176 (152–198)	0.902	176 (155–189)	< 0.001
Preoperative surgical diagnosis		n (%)		n (%)		n (%)	
Aortic stenosis		8 (17)		32 (26)		42 (52)	
Aortic insufficiency		19 (41)		49 (40)		20 (25)	
Combined disease		18 (39)		40 (32)		18 (22)	
Aortic prosthesis replacement		1 (2)		2 (2)		1 (1)	
Bicuspid aortic valve	0.130	23 (50)		51 (41)		26 (32)	
Ascending aorta aneurysm	0.009	0		10 (8)		12 (15)	
Active endocarditis		1 (2)		5 (4)		4 (5)	
Preop NYHA III and IV	< 0.001	10 (22)	< 0.001	62 (50)	0.708	43 (53)	< 0.001

[a] Global test: Kruksal-Wallis test or χ^2 test; [b] Mean ± standard deviation; [c] Median (range).

Preop NYHA = preoperative New York Heart Association functional class.

2 to 6 months, 12 months, and then annually thereafter. Autograft regurgitation was graded using the method of Perry and colleagues [8]. Pulmonary homograft regurgitation was estimated whenever possible according to the grading of Chan and coworkers [9]. Peak and mean systolic gradients across the autograft and pulmonary

Table 2. Operative Details and Concomitant Procedures

	p[a]	Group 1	p (Group 1 Versus 2)	Group 2	p (Group 2 Versus 3)	Group 3	p (Group 1 Versus 3)
Bypass time[b] (min)	0.804	148 (98–235)		147 (95–237)		152 (104–271)	
Cross-clamp time[b] (min)	0.088	118 (80–189)		121 (51–185)		126 (81–194)	
Aortic annulus[b] (mm)	< 0.001	23 (17–35)	<0.001	27 (20–37)	0.110	27 (21–35)	<0.001
Autograft diameter[b] (mm)	< 0.001	23 (17–31)	<0.001	27 (23–31)	0.255	27 (23–33)	<0.001
Homograft diameter[b] (mm)	< 0.001	23 (20–29)	<0.001	25 (19–33)	0.739	25 (21–33)	<0.001
		n (%)		n (%)		n (%)	
Previous cardiac/aortic operation		21 (46)		13 (11)		3 (4)	
Combined procedure	0.055	7 (15)		24 (20)		26 (32)	
Ascending aorta replacement		0		11[c] (9)		15 (19)	
Coronary artery bypass grafting	0.091	1 (2)		10 (8)		11 (14)	
LVOT enlargement (Manouguin)		2 (4)		0		0	
VSD plus LVOT enlargement (Manouguin)		1 (2)		0		0	
VSD		1 (2)		0		0	
VSD plus subaortic membrane		1 (2)		0		0	
VSD plus ASD		0		1 (1)		0	
PDA		1 (2)		0		0	
Ostioplasty		0		2[c] (2)		0	
Modified maze procedure		0		1 (1)		0	
Pericardial fenestration repair		0		0		1 (1)	
Mitral valve repair		0		1 (1)		0	
PAPVR		0		0		1 (1)	

[a] Global test: Kruksal-Wallis test or χ^2 test; [b] Median (range); [c] Additional coronary artery bypass grafting in 1 patient.

ASD = atrial septal defect; LVOT = left ventricular outflow tract; PAPVR = partial anomalous pulmonary vein return; PDA = patent ductus arteriosus; VSD = ventricular septal defect.

Table 3. Major Complications

	Group 1	Group 2	Group 3
	n	n	n
Early mortality	0	0	0
Perioperative bleeds	2 (4.3)[a]	3 (2.4)[a]	1 (1.2)[a]
Transiently elevated myocardial enzymes	1 (2.2)[a]	1 (0.8)[a]	1 (1.2)[a]
Prolonged ventilation	0	1 (0.8)[a]	1 (1.2)[a]
Pacemaker insertion	1 (2.2)[a]	2 (1.6)[a]	0
Late death	0	1[b] (0.8)[a]	3 (3.7)[a]
Late hemorrhagic event	0	0	0
Late thromboembolic event	0	0	0
Autograft related reoperation	0	4 (3.3)[a]	1 (1.2)[a]
Homograft related reoperation	1 (2.2)[a]	0	0
Autograft endocarditis (early)	0	2 (1.6)[a]	0
Homograft endocarditis (late)	0	1[b] (0.8)[a]	0

[a] Values within parentheses are percentages; [b] Same patient.

homograft valves were calculated by Doppler velocity with the use of the modified Bernoulli equation. Aortic annulus dimensions, diameters of Valsalva's sinus, and sinotubular junction were measured from parasternal long-axis views of the aortic root, using the inner wall distances during early systole from three to four consecutive cardiac cycles.

Statistical Analysis

Data are presented as mean ± standard deviation (unless non-normally distributed or otherwise indicated). The χ^2 test was used for comparison of frequencies between groups. With respect to continuous data in general the Kruskal-Wallis test was used as a global test. In cases of significance, considered at a p value < 0.05 for all tests, individual group differences were determined post hoc using the Wilcoxon rank-sum test. Time-to-event analyses were performed using Kaplan-Meier methods.

Results

There were no operative or early deaths. Early autograft endocarditis was suspected by echocardiography in 2 patients from group 2 with negative blood cultures in both. These 2 patients were successfully treated intravenously with antibiotics. All major complications are summarized in Table 3.

With regard to the 246 survivors, the follow-up was 95% complete within 16 months of the closing of the study date. The echocardiographic follow-up results are presented in Table 4. Long-term no thromboembolic or hemorrhagic event occurred.

Autograft Complications

For the whole series we had 5 patients requiring reoperation for autograft failure. Four of these patients are from group 2.

A 44-year-old woman with grade one regurgitation at discharge after the Ross procedure developed slow progressive autograft insufficiency. At reoperation after 52 months, a left coronary cusp tethered by a stitch was detected and her autograft was successfully repaired.

A 49-year-old male patient showed progressive annular dilatation with incompetence after the Ross operation. At reoperation after 26 months, fibrotic shrinkage of the autografts' right coronary cusp was found. He requested and received an aortic homograft as replacement.

A 30-year-old man with bicuspid aortic valve, on whom we had also reduced the aortic annulus from 33 to 25 mm at the initial operation and had also supported the annulus with an untreated autologous pericardial strip, presented after 32 months with autograft regurgitation and left ventricular dilatation. At reoperation the aortic

Table 4. Echocardiographic Profile

	p[a]	Group 1	p (Group 1 Versus 2)	Group 2	p (Group 2 Versus 3)	Group 3	p (Group 1 Versus 3)
Autograft							
Systolic peak gradient (mm Hg)	0.426	5.7 ± 2.6		5.1 ± 2.6		5.4 ± 2.6	
Systolic mean gradient (mm Hg)	0.877	3.0 ± 1.0		3.1 ± 1.4		3.2 ± 1.4	
		n		n		n (%)	
AI ≥ II°		0		0		1 (1.2)	
Homograft							
Systolic peak gradient (mm Hg)	< 0.001	23.6 ± 17.8	0.002	14.6 ± 8.3	0.002	11.5 ± 6.5	< 0.001
Systolic mean gradient (mm Hg)	< 0.001	13.4 ± 10.2	0.005	8.6 ± 5.4	0.001	6.6 ± 4.2	< 0.001
		n (%)		n (%)		n (%)	
Systolic peak gradient ≥ 40 mm Hg		8 (17.4)		3 (2.4)		0	

[a] Global test: Kruksal-Wallis test.

AI = autograft regurgitation.

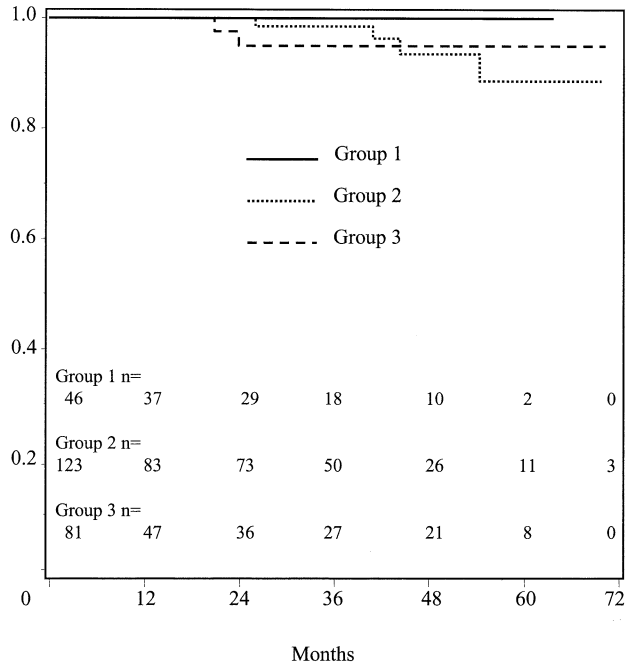

Fig 1. Kaplan-Meier curve of freedom from pulmonary autograft reoperation or AI ≥ II°. (AI = autograft regurgitation.)

annulus had again dilated to 35 mm, the acoronary cusp prolapsed, and also the posterior cusp was perforated. He received autograft replacement with an aortic homograft.

Finally, for group 2, a 29-year-old man with a bicuspid aortic valve and history of a previously operated coarctation of the aorta at childhood, presented with aortic incompetence, annular and ascending aorta dilatation. At the Ross procedure the annulus was plicated down from 35 to 27 mm. A potentially stretchable felt strip was used in an attempt to support the aortic annulus. In addition his pulmonary autograft showed three small perforations judged as insignificant. At reoperation performed after 40 months because of incompetence, the annulus was found to have also redilated to 37 mm, the left coronary cusp prolapsed, and the ascending aorta had dilatated (2 cm above the original autograft to the ascending aortic anastomosis). He requested a mechanical valved conduit as a replacement.

In group 3, a 50-year-old man with a previous bicuspid aortic valve and a pericardial strip as support of a similarly plicated, dilatated aortic annulus, also developed progressive annular dilatation. At reoperation performed after 20 months, the annulus had dilatated from 26 to 38 mm with irreparable prolapse of the left coronary cusp, and he received an aortic homograft.

In the same age group we have been closely following up a 62-year-old patient with arterial hypertension, but interestingly there was no aortic annulus plication at the original operation and only trace autograft regurgitation at discharge. The incompetence subsequently progressed to an asymptomatic grade 2. This has remained stable for more than 1 year under strict afterload reduction.

Kaplan-Meier estimates of freedom from autograft failure (Fig 1) defined as autograft related reoperation or autograft regurgitation ≥ II° for groups 1, 2, and 3 at 48 months were 100%, 93.5 ± 7% (standard error 0.038) and 95.0 ± 7% (standard error 0.035), respectively.

Pulmonary Homograft Complications

An 8-year-old boy from group 1 with a previous homograft aortic valve replacement, 7 years before his Ross operation, developed rapid progressive pulmonary homograft stenosis and he was treated with an outflow tract widening patch at 8 months.

We have 8 patients from group 1 and three patients from group 2 under close observation for elevated pulmonary homograft gradients of 40 mm Hg and more.

Estimates of freedom from homograft failure (Fig 2) defined as homograft related reoperation or a peak gradient of greater than or equal to 40 mm Hg across the homograft valve at 48 months were 65.9 ± 23% (standard error 0.119), 95.8 ± 6% (standard error 0.031), and 100% for groups 1, 2, and 3, respectively.

Late Mortality

Overall, 4 patients died during follow-up, 3 from group 3 and 1 from group 2. A 44-year-old man died at 25 months from massive hemoptysis due to suppurative pneumonia and with unsuspected pulmonary homograft endocarditis found at autopsy.

Another 57-year-old male patient, operated on for predominant aortic stenosis, but who also had essential

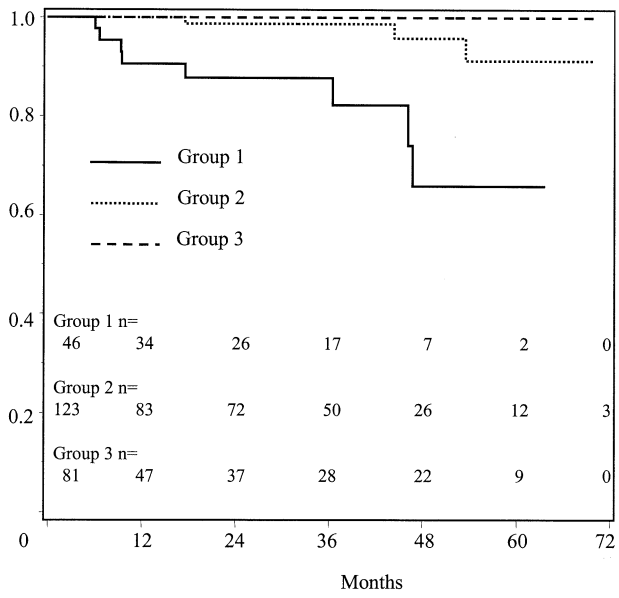

Fig 2. Kaplan-Meier curve of freedom from homograft reoperation or a peak gradient ≥ 40 mm Hg across the homograft.

arterial hypertension, died suddenly of an unknown-cause after 5 months. Unfortunately, autopsy was denied.

Another 56-year-old man expired from a suspected acute descending thoracic aorta dissection 40 months after the Ross procedure. The diagnosis was made by transesophageal echocardiography and the relatives denied autopsy.

The fourth patient who died late in our series, a 57-year-old man, expired after weeks with severe neurological deficit following late resuscitation for suspected ventricular arrhythmia at 25 months.

Comment

The superior hemodynamic properties of the Ross procedure, as aortic valve replacement in patients of all ages independent of their age groups, are confirmed by the near-physiologic gradients across the pulmonary autograft found in all of our patients in this series. As shown in several other studies, the physiologic hemodynamic properties of the pulmonary autograft enable normal exercise tolerance for patients until the Ross operation when compared with a healthy control group [6, 10]. These factors, together with the absence of thromboembolic or hemorrhagic events without anticoagulation, encouraged us to extend the indications for the Ross operation.

However, we did experience autograft failure during a follow-up of 20 to 52 months after the Ross operation, and all patients (except 1 from group 3) were from group 2. Whether this complication is related to a specific age group has not been adequately addressed, particularly after adulthood. Almost all patients with autograft failure were operated on early in this series and received no support, or in retrospect inadequate support, of an aortic annulus plicated to match the size of the autograft. In these cases the autograft failure may reflect a technical learning curve, as discussed in detail in a previous report [7]. After this evolutionary process and revision of our technique, to date, no further autograft reoperation has been necessary in any group.

Three of those patients with autograft failure had a bicuspid aortic valve and 2 had aortic insufficiency as the preoperative diagnoses, both speculative factors associated with autograft failure [1, 12, 13]. In contrast, and despite the fact that aortic insufficiency as preoperative diagnosis and a bicuspid aortic valve were found, respectively, in 40 and 50 percent of patients from group 1, surprisingly we had no case of autograft failure in this age group. This is despite concern about the ability of the pulmonary autograft to adapt to the higher pressures in the left ventricular outflow tract in these younger patients, and also despite speculation about desired growth of the autograft in younger patients being a combination of real somatic autograft growth and passive dilatation [4]. It may be that growth of the younger patients could compensate for passive autograft or aortic annulus dilatation.

On the other hand, for the older patient some skepticism exists whether the pulmonary autograft has the potential of an appropriate aortic valve substitute with satisfying long-term results, because of a decline in its elastic integrity [14]. This can only be answered in our patient group by continued close follow-up, but for this study the results are extremely encouraging.

In 2 patients we observed excessively high arterial blood pressure partly caused by inadequate antihypertensive medication (1 patient from the middle-aged group 2 with autograft failure and fixed arterial hypertension after coarctation repair in childhood with residual moderate stenosis, and the other patient from the older group 3 with progressive autograft regurgitation). In the latter patient, adequate medical therapy seems to have preserved the patient's autograft function at a stable level of grade 2 insufficiency for 1 year, although further deterioration is not excluded. According to a study from Carr-White and colleagues [15], who investigated the mechanical behavior of the pulmonary artery, there is evidence that a systolic blood pressure in excess of 200 mm Hg is more likely to lead to disruption of elastic fiber and breakage in the pulmonary artery wall than in the aortic wall. The authors emphasize the importance of treating hypertension in patients receiving a pulmonary autograft.

Three of our patients with autograft failure had an aortic annulus diameter of substantially more than 30 mm before plication, which is why we have become more cautious in accepting patients for the Ross operation who have an aortic annulus diameter of more than 30 mm in which extensive autograft mismatch requires plication.

During follow-up, older patients of group 3 presented with significantly lower pulmonary homograft gradients in the right ventricular outflow tract, and no case has been documented with homograft stenosis defined as a gradient of more than 40 mm Hg across the homograft. Because the problem of right ventricular outflow tract stenosis has not been seen in the elderly, it could be concluded that the Ross procedure may offer superior results for aortic valve disease in the elderly.

When discussing the possible reasons for the problem of pulmonary homograft stenosis in younger patients, an obvious question is whether younger patients have simply outgrown their implanted homografts. This is unlikely, not only because younger patients have all received oversized grafts, but also because the gradients all increased too rapidly. The patient's body surface area served as a guide for the choice of homograft size and all patients with a body surface area greater than 1.6 m^2 received any valve larger than 23 mm, whereas patients with a body surface area less than 1.6 m^2 received any valve between 18 to 23 mm. Regarding children and adolescents with homograft stenosis, the graft diameter measured at implantation was even suitable when we compared it with the patient's actual body surface area from their latest follow-up, except in 1 adult patient who received a smaller valve because no appropriate size was available at the time of the Ross operation.

A more likely reason for the development of pulmonary homograft stenosis is an immune reaction. Carr-White and colleagues [16] suggested an early postoper-

ative inflammatory reaction with extrinsic compression and shrinkage after examination of 15 patients with echocardiographically detected pulmonary homograft stenosis by magnetic resonance imaging with velocity mapping. In the 8-year-old boy of our series who received a pulmonary homograft patch widening for rapid progressive stenosis, we found the whole homograft conduit to be symmetrically narrowed with unaffected and pliable valve cusps, supporting the hypothesis of an associated immune response. Other investigators also reported significantly elevated gradients across the right-sided pulmonary homografts in younger patients when compared with older patients, and these authors speculated whether age-related calcium turnover or the immunologic status might be responsible [17]. We try to match age of donor and recipient as much as possible.

Whether cryopreserved and decellularized pulmonary homografts with suspected decrease in the recipient's human lymphocyte antigen antibody response [18] will improve the homograft durability, especially in the young, is not clear yet. Further investigation is necessary to identify the cause of homograft deterioration. However, this report again documents an increased risk of deteriorating function of right-sided homografts in younger patients.

Of the four cases with late deaths observed in this series, three affected older group 3 patients and all appear cardiac related. Whether this observation is of clinical importance remains unclear, but older patients from group 3 were operated on later in the series as our experience grew. They were preoperatively more frequently in a worse New York Heart Association functional class and underwent more combined operations when compared to patients from other groups. Natural causes of death are also higher in older patients and comparable for this series.

As reported previously, the Ross operation in complicated constellations or as a combined procedure can be performed without an increase in risk and operative morbidity [19]. Despite this experience and the excellent hemodynamic results after the Ross operation in older patients, this age group will present with more concomitant pathology. Thus careful selection of older patients as candidates for the Ross operation is warranted, a fact also stated by others [20]. The natural life expectancy must still be normal for each candidate, and in our clinic a homograft or stentless biological valve is still chosen if we feel that this will outlast the life expectancy of an older patient or one with multiple pathologies.

In conclusion, the overall absence of perioperative deaths, low morbidity, and reoperation rate support the evolved root replacement technique and justify its further use in all age groups of this study. The absence of right ventricular outflow tract stenosis in the older group of patients in this series implies that currently the Ross procedure as root replacement offers superior surgical results for aortic disease in patients older than approximately 50 years of age. Thus for specialized centers, the Ross operation may be the ideal therapy for aortic valve replacement in middle-aged and older otherwise healthy patients with an active lifestyle and life expectancy in excess of that of an aortic homograft or other stentless biological prosthesis [11].

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