Midterm Results of the Ross Procedure Preserving the Patient’s Aortic Root

Hans-H. Sievers, MD; Gerlinde Dahmen, MSc; Bernhard Graf, MD; Ulrich Stierle, MD; Andreas Ziegler, PhD; Claudia Schmidtke, MD

Background—Since the early 1990s, the pulmonary autograft is predominantly implanted as a freestanding root for less aortic valve regurgitation is reported. However, there is a certain risk of dilatation of the root over time potentially impairing valve function. We favor since 8 years the original subcoronary or inclusion technique to preserve the root of the patient as a restrain to dilatation.

Methods and Results—Between June 1994 and May 2002 the subcoronary (n = 228) and inclusion technique (n = 17) were performed in 245 patients (191 male, 54 female), mean age 45.7 ± 13.4 (15–70) years. The underlying aortic valve disease was an aortic insufficiency in n = 83, stenosis in n = 48, a combined aortic valve disease in n = 111 and an acute endocarditis in n = 19 patients. Previous aortic valve surgery was performed in n = 23. Last follow-up investigations (within last year) including echocardiography was performed at a mean follow-up of 29.4 ± 24.7 months (553.7 patient years). Hospital mortality was n = 2, late mortality n = 4 (all noncardiac). Two patients were lost to follow-up (99% complete clinical follow-up). Reoperations were necessary in n = 7 valves (autograft: endocarditis n = 1, malpositioning n = 1, leaflet prolapse n = 1; homograft: stenosis n = 2, insufficiency n = 2). Autograft insufficiency (AI) was AI 0 in n = 154, AI I n = 66, AI II n = 8. The maximum/mean pressure gradient across the autograft was 6.6 ± 3.4 (2.1 to 25.9)/3.6 ± 1.8 (1.2 to 13.2) mm Hg, respectively. Homograft insufficiency was 0 in n = 167, I in n = 54, II in n = 9, and III in n = 1. Maximum and mean transhomograft pressure gradients were 11.7 ± 6.8 (2.2 to 42.6)/6.2 ± 3.8 (1.2 to 24.5) mm Hg. Most patients were NYHA class I (n = 214), class II (n = 19), class III (n = 2). Significant aortic root dilatation was not observed.

Conclusions—Aortic valve replacement with a pulmonary autograft in the subcoronary or inclusion technique provides excellent hemodynamics with no root dilatation at least in a mid term postoperative period. Transhomograft pressure gradients are slightly increased. Longer term results with special emphasis on the pulmonary homograft are necessary. (Circulation. 2003;108[suppl II]:II-55-II-60.)

Key Words: valves ■ prosthesis ■ surgery

The pulmonary autograft has emerged as an attractive alternative substitute for aortic valve replacement with promising hemodynamic and clinical results. This surgical method was originally inaugurated by D. Ross in 1967 who implanted a pulmonary autograft in subcoronary position.1 During the last decade the autograft is implanted preferentially as a freestanding root which was reported to have the lowest occurrence rate of aortic insufficiency postoperatively.2–5 Although the intermediate term results of this technique are excellent concern increases about the neoaoartc root to sustain systemic pressure over time without dilatation and subsequent insufficiency.6 Especially in patients with congenital aortic valve disease progressive dilatation was observed when complete root replacement was used.6,7 This kind of risk is less inherent in the subcoronary and root inclusion techniques, however, the autograft needs to be adapted to the anatomy of the patient’s aortic root rendering these techniques more susceptible to implantation related malfunction. Nevertheless, the obvious advantage of a lower risk of dilatation with these techniques that preserve the patient’s aortic root is appealing, provided excellent results concerning the autograft can be achieved over time. Longer term follow-up studies are necessary for further evaluation of these types of the Ross procedure. Therefore, we present our series of the subcoronary autograft and the root inclusion procedure during the last 8 years.

Patients and Methods
From June 1994 until May 2002 245 patients (Table 1) were consecutively operated on using the subcoronary (n = 228) or root inclusion technique (n = 17). The mean age was 45.7 ± 13.4 years, ranging from 15 to 70 years. Last follow-up investigation in the outpatient clinic included echocardiography and was performed during the last year at a mean follow-up of 29.4 ± 24.7 months (range 0.1 to 84.5) months (553.7 patient years). Two patients were lost to follow-up comprising 99% completeness. Exclusion criteria for the
Ross procedure were severe calcification of the aortic root, significantly reduced left ventricular ejection fraction (<40%), more than 2 vessel coronary artery disease and anatomical or structural defects of the pulmonary valve.

**Operative Data**

Extracorporeal circulation with moderate hypothermia (26°C nasopharyngeal temperature) was used in all cases. In the first 5 years crystalloid cardioplegia and thereafter cold blood cardioplegia was applied. Explanation of the autograft and implantation in a subcoronary technique as well as root inclusion technique were accomplished as described previously in detail. The autograft was trimmed at the base in a scalloped fashion to match the proximal suture line with the remnants of the leaflet attachments of the excised patient’s valve as close as possible. The proximal anastomosis was performed with single 4 to 0 polyfilament and the distal anastomosis with 2-dimensional echocardiography at 3 different levels: the supraaortic annulus at the level of the autograft leaflet hinges, the sinus of Valsalva at the largest antero-posterior diameter, and the supraaortic ridge level at the distal rim of the sinuses of Valsalva as described by Roman et al. Measurements of diameters where taken perpendicular to the long axis of the aorta, maximum velocities across the aortic valve and the homograft where obtained by continuous wave Doppler imaging transducer. To assess aortic and pulmonary regurgitation pulsed wave Doppler and color flow Doppler imaging where used. Trace aortic insufficiency defined as tiny regurgitant jet in early diastole was grouped to aortic insufficiency grade 0. Grade I regurgitant jets (mild aortic insufficiency) were present only in the left ventricular outflow tract immediately below the valve, grade II and III (moderate aortic insufficiency) extended to the tips of mitral leaflet and papillary muscles, respectively. Peak systolic pressure gradient was calculated according to the modified Bernoulli equation (delta P [mm Hg]=4v^2 [m/s]) where v is the peak systolic velocity.

**Statistical Analysis**

Categoric data are given as total numbers and relative frequencies. Continuous data are given as mean±SD. Time related events such as survival and reoperation also included events during the primary hospital stay and were calculated by the Kaplan Meier method. Morbidity and mortality are reported according to the guidelines of Edmunds et al.

**Results**

**Mortality**

Hospital mortality was n=2 (0.8%). One patient died because of refractory arrhythmias 3 days after the procedure and another patient who was operated on for acute aortic valve endocarditis died from thrombotic occlusion of the left main coronary artery 1 day before discharge. There were 4 nonvalve related late deaths (n=1 cranial bleeding, n=1 esophageal varices bleeding, n=2 cancer). Thus, freedom from valve related and overall death was 99% and 94%, respectively, after 7 years (Figure 1).

**Morbidity**

**Structural Valvular Deterioration**

According to Edmunds et al structural valve deterioration is defined as any change in function of an operated valve servers from video recorded studies. The average value of 5 consecutive beats was taken. Autograft diameters were measured with 2-dimensional echocardiography at 3 different levels: the annulus at the level of the autograft leaflet hinges, the sinus of Valsalva at the largest antero-posterior diameter, and the supraaortic ridge level at the distal rim of the sinuses of Valsalva as described by Roman et al. Measurements of diameters where taken perpendicular to the long axis of the aorta, maximum velocities across the aortic valve and the homograft where obtained by continuous wave Doppler imaging transducer. To assess aortic and pulmonary regurgitation pulsed wave Doppler and color flow Doppler imaging where used. Trace aortic insufficiency defined as tiny regurgitant jet in early diastole was grouped to aortic insufficiency grade 0. Grade I regurgitant jets (mild aortic insufficiency) were present only in the left ventricular outflow tract immediately below the valve, grade II and III (moderate aortic insufficiency) extended to the tips of mitral leaflet and papillary muscles, respectively. Peak systolic pressure gradient was calculated according to the modified Bernoulli equation (delta P [mm Hg]=4v^2 [m/s]) where v is the peak systolic velocity.

**Statistical Analysis**

Categoric data are given as total numbers and relative frequencies. Continuous data are given as mean±SD. Time related events such as survival and reoperation also included events during the primary hospital stay and were calculated by the Kaplan Meier method. Morbidity and mortality are reported according to the guidelines of Edmunds et al.

**Results**

**Mortality**

Hospital mortality was n=2 (0.8%). One patient died because of refractory arrhythmias 3 days after the procedure and another patient who was operated on for acute aortic valve endocarditis died from thrombotic occlusion of the left main coronary artery 1 day before discharge. There were 4 non-valve related late deaths (n=1 cranial bleeding, n=1 esophageal varices bleeding, n=2 cancer). Thus, freedom from valve related and overall death was 99% and 94%, respectively, after 7 years (Figure 1).

**Morbidity**

**Structural Valvular Deterioration**

According to Edmunds et al structural valve deterioration is defined as any change in function of an operated valve servers from video recorded studies. The average value of 5 consecutive beats was taken. Autograft diameters were measured with 2-dimensional echocardiography at 3 different levels: the annulus at the level of the autograft leaflet hinges, the sinus of Valsalva at the largest antero-posterior diameter, and the supraaortic ridge level at the distal rim of the sinuses of Valsalva as described by Roman et al. Measurements of diameters where taken perpendicular to the long axis of the aorta, maximum velocities across the aortic valve and the homograft where obtained by continuous wave Doppler imaging transducer. To assess aortic and pulmonary regurgitation pulsed wave Doppler and color flow Doppler imaging where used. Trace aortic insufficiency defined as tiny regurgitant jet in early diastole was grouped to aortic insufficiency grade 0. Grade I regurgitant jets (mild aortic insufficiency) were present only in the left ventricular outflow tract immediately below the valve, grade II and III (moderate aortic insufficiency) extended to the tips of mitral leaflet and papillary muscles, respectively. Peak systolic pressure gradient was calculated according to the modified Bernoulli equation (delta P [mm Hg]=4v^2 [m/s]) where v is the peak systolic velocity.

**Statistical Analysis**

Categoric data are given as total numbers and relative frequencies. Continuous data are given as mean±SD. Time related events such as survival and reoperation also included events during the primary hospital stay and were calculated by the Kaplan Meier method. Morbidity and mortality are reported according to the guidelines of Edmunds et al.
because of intrinsic abnormalities leading to stenosis or regurgitation resulting in a decrease of one New York Heart Association (NYHA) functional class or more. 7 patients reported on a decrease of functional capacity (n=5 NYHA class I to II, n=2 NYHA class II to III). However, only 2 of these 7 patients showed an impairment of valve function (n=1 aortic valve insufficiency increase grade 0 to grade I and n=1 transhomograft maximum pressure gradient increase from 6.6 to 17.6 mm Hg). Nevertheless, without change of NYHA functional class 7 patients presented an increase of aortic valve insufficiency (n=5 grade I to II and n=2 grade 0 to II) and 24 patients an increase of maximum pressure gradient across the homograft of more than 20 mm Hg (range 20 to 42.6, 26.2±6.3 mm Hg). Structural valvular deterioration as verified by reoperation occurred in 4 homografts (n=2 with significant stenosis, n=2 with moderate pulmonary insufficiency) and one autograft due to insufficiency grade II because of leaflet prolapse.

Nonstructural Dysfunction
There was one nonstructural dysfunction of an autograft due to inappropriate positioning as determined by reoperation one day after primary procedure.

Valve Thrombosis
There was no valve thrombosis.

Embolism
Neurological events occurred in 4 patients: n=1 transient ischemic attack, n=1 reversible ischemic neurologic deficit, n=2 stroke (one with new onset of atrial fibrillation) in patients 68 and 69 years of age.

Bleeding Event
There were 6 major bleedings (n=4 with gastrointestinal bleeding, n=1 cerebral bleeding, n=1 esophageal varices bleeding) in patients without anticoagulation.

Operated Valvular Endocarditis
There was one autograft that had to be reoperated because of severe aortic insufficiency after healed endocarditis.

Reoperations
Seven valves were reoperated: n=3 autografts (due to endocarditis, malpositioning, leaflet prolapse) and n=4 homografts (n=2 stenosis, n=2 insufficiency). The freedom from reoperation at 7 years was 98% for the autograft and 97% for the homograft (Figures 2 and 3). There were no complications because of reoperation.

Autograft Function
Aortic regurgitation ≤ grade I was observed in 96%, grade II in 3.7%. There was no patient with more than grade II insufficiency. The maximum/mean pressure gradient across the autograft was 6.6±3.4 (range 2.1 to 25.9) mm Hg and 3.6±1.8 (range 1.2 to 13.2) mm Hg, respectively (Table 3).

Homograft Function
Homograft insufficiency was ≤ grade I in 95.7%, grade II in 3.9%, grade III in 0.4%. Maximum and mean transhomograft pressure gradients were 11.7±6.8 (range 2.2 to 42.6) mm Hg and 6.2±3.8 (range 1.2 to 24.5) mm Hg, respectively, with an increase early postoperatively (Table 3, Figure 4).

NYHA Functional Class
Most patients (91.1%) were in NYHA class I, 8.1% in class II and 0.9% in class III.

Dimensions of the Aortic Root
The mean diastolic diameter of the aortic annulus was 21.3±3.4 mm, at the level of the sinus of Valsalva 28.9±4.1 mm and at the supraaortic ridge 26.0±4.2 mm.
Discussion
This study with up to 8 years experience with the Ross procedure preserving the patient’s aortic root provides some evidence that the original subcoronary and also the root inclusion technique lead to favorable midterm results warranting its further consideration.

Mortality
The hospital mortality of 0.8% (n=2) is comparable to that reported in the International Ross Registry with 2.5%.4 No patient in our series died late due to valve related complications. All late deaths were caused by noncardiac events reflecting the excellent survival of this operative technique. The overall survival rate at 7 years postoperatively was 94%. Chambers et al12 reported on a survival rate of 85% after 10 years and 61% after 20 years whereas Elkins et al13 described a survival rate after the Ross procedure using the freestanding root technique of 87% after 10 years in a group of patients ranging in age from 1 week to 62 years with a mean age of 22 years. These results are comparable to those of this study (Figure 1) keeping in mind, however, that the mean patient age was essentially higher with 45.7 (range 15 to 70) years. This indicates that the survival rate after the subcoronary technique is at least comparable to that after the freestanding root technique.

Structural Valvular Deterioration
Structural valvular deterioration is the most difficult item to define precisely but one of the most important to describe durability of valve function. Applying the guidelines of Edmunds et al11 only 4 homografts and 1 autograft fulfilled the criteria of valvular deterioration as verified by reoperation. Interestingly, 2 homografts failed for insufficiency and 2 for stenosis which was not related to leaflet calcification but to shrinkage of the conduit confirming the result of Niwaya.14 These authors also stated that the shrinkage of the homograft conduit significantly contributes to valve failure. The question as to whether an immunological response is involved in this process is still not settled.15 There is, however, a growing body of evidence that a gradually variable response resulting in some increase in pressure gradient occurs in the majority of patients early after implantation of the homograft. We and others14 found a significant increase in pressure gradient in the immediate period after implantation. Fortunately, this pressure gradient did not change significantly over the following years (Figure 4) indicating some stability of this postimplantation reaction. This transhomograft pressure gradient did not lead to impairment in NYHA classification in our patients although there were 24 patients with a maximum transhomograft pressure gradient of more than 20 mm Hg at last follow-up investigation. Irrespective of the question whether these findings fulfill the criteria of valvular deterioration the long-term behavior of the homograft remains the Achilles’ heel of any kind of the Ross procedure and close follow-up of patients with elevated transhomograft pressure gradients is necessary. Concerning the autograft we found 7 patients with an impairment of the aortic valve insufficiency to grade II without a decrease of functional state. Only one patient presented an impairment of NYHA class I to II who also showed increase of aortic insufficiency from grade 0 to I. Whether this is related to the aortic valve insufficiency remains speculative since the left ventricular function was

<table>
<thead>
<tr>
<th>TABLE 3. Data of Last Follow-up Investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Insufficiency</td>
</tr>
<tr>
<td>I°</td>
</tr>
<tr>
<td>II°</td>
</tr>
<tr>
<td>III°</td>
</tr>
<tr>
<td>IV°</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Pressure gradient (mm Hg)</td>
</tr>
<tr>
<td>maximum</td>
</tr>
<tr>
<td>mean</td>
</tr>
<tr>
<td>NYHA</td>
</tr>
<tr>
<td>I</td>
</tr>
<tr>
<td>II</td>
</tr>
<tr>
<td>III</td>
</tr>
<tr>
<td>IV</td>
</tr>
</tbody>
</table>

Figure 4. Mean pressure gradient across the homograft during follow-up. Only patients with 4 consecutive echocardiographic follow-up studies (n=30) are considered. The gradient increase from postoperative investigation (up to 1 month postoperatively) was statistically significant when compared with 1st follow-up study and did not change thereafter statistical significantly.
still excellent without indication for reoperation. Important to note is that three of these patients showed preoperatively signs of active rheumatoid disease indicating that these patients probably are not ideal candidates for the Ross procedure. 16 In most patients, however, the function of the autograft was excellent and stable over the years even at exercise. 17

In summary determination of the structural valvular deterioration of the homograft and the autograft in the Ross procedure is difficult and probably needs more precisely defined and international accepted criteria.

Reoperation
One of the main potential risks of the Ross procedure is the reoperation especially for homograft dysfunction. The linearized rate was 1.15% per patient year in our series for autografts and homografts together. Similar data are reported by Chambers et al in a pioneer series with a freedom from reoperation of 88% and 75% for autografts and 89 and 80% for homografts after 10 and 20 years, respectively. 12 These data are comparable to those of our series with 2% reoperation rate for autografts and 3% for homografts after 7 years. Reoperation of the autograft was necessary in three patients within the first 2 years after the operation (Figure 2) because of endocarditis, malpositioning and leaflet prolapse, reasons that are less likely to be related to degeneration. In concert with the stable function of the autograft over time it becomes evident that the degeneration rate of the autograft at least in this midterm follow-up seems to be low.

Function of the Pulmonary Autograft
The function of the autograft regarding the pressure gradient is excellent simulating almost normal conditions in almost all patients. This is also confirmed during exercise with no undue increase of pressure gradient. 13 The International Ross Registry reports on 86% of patients with aortic valve insufficiency grade I or less, 9% insufficiency grade II, 3% insufficiency grade III and in 2% grade IV. 4 The completeness of the follow-up of the International Registry is about 70% and thus, the conclusion limited. In our series the results were similar reflecting 99% completeness of follow-up. In most patients we could not find an increase of aortic insufficiency during follow-up. The function of the neoaortic valve seems to be stable over the years, even in the subcoronary technique.

Function of the Pulmonary Homograft
Most patients developed some kind of moderate pressure gradient early after implantation reflecting a yet undefined reactive mechanism (Figure 4). This pressure gradient is well tolerated in most patients but does increase during exercise. 17 Whether this will impair right ventricular function over time remains to be established and carefully followed. Until now we did not observe clinical or echocardiographic signs of right ventricular dysfunction in patients with moderate pressure gradients across the homograft. Nevertheless, maximum transhomograft pressure gradients of more than 20 mm Hg require careful follow-up. Further research in the Ross procedure should aim on the performance of the homograft and how to improve the results.

Dilatation of the Aortic Root
Primary concern as to whether the freestanding pulmonary root would sustain systemic pressure over time could be destroyed by the excellent results with the freestanding root technique of Stelzer et al, Kouchoukos et al, and Elkins et al. 3,5,13 Nevertheless, there are some reports indicating that especially patients with congenital aortic valve disease with aortic insufficiency may develop some kind of dilatation of the neoaortic root 6,7 resulting in aortic valve insufficiency and reoperation. 7 Technical details are probably useful to prevent autograft dilatation when the freestanding root is used. 18 Obviously, there exists a special risk for unphysiological dilatation of the pulmonary autograft after the Ross procedure in special cases which was not found when the subcoronary technique is used. 8

Limitations of the Study
It must be taken into consideration that this is a retrospective observational study with a relatively short median follow-up period. Furthermore, the observed number of events is too low for the analysis of possible factors influencing overall survival and reoperation rates. This is also causative for the observed negligible differences between the Kaplan Meier and cumulative incidence analysis. We therefore presented only the Kaplan Meier curves.

Conclusions
The Ross procedure in the subcoronary or root inclusion technique preserving the patient’s aortic root provides excellent mid-term results in this observational study. Autograft function is comparable to that reported in the literature for the freestanding root without dilatation of the aortic root. Whether the valve function of the autograft and especially that of the homograft and thus, the durability of the valves remains favorable over years remains to be determined. Future research should be focused on improvement of right ventricular outflow tract reconstruction.

References


Midterm Results of the Ross Procedure Preserving the Patient's Aortic Root
Hans- H. Sievers, Gerlinde Dahmen, Bernhard Graf, Ulrich Stierle, Andreas Ziegler and Claudia Schmidtke

_Circulation_. 2003;108:II-55-II-60
doi: 10.1161/01.cir.0000087443.84392.32
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2003 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/108/10_suppl_1/II-55