Functional outcomes after the Ross (pulmonary autograft) procedure assessed with magnetic resonance imaging and cardiopulmonary exercise testing


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Functional outcomes after the Ross (pulmonary autograft) procedure assessed with magnetic resonance imaging and cardiopulmonary exercise testing

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ABSTRACT
Objective To assess autograft, homograft and ventricular function, as well as exercise capacity, in adult patients who have undergone the Ross procedure.

Setting Single centre paediatric and adult congenital heart disease unit.

Patients 45 subjects (24.6 years, range 16.9—52.2 years) who underwent the Ross procedure between 1994 and 2006 (8.1 years after the Ross operation, range 2.0—14.0 years).

Interventions Cardiovascular magnetic resonance imaging, echocardiography and cardiopulmonary exercise testing.

Main outcome measures Autograft and homograft stenosis, and regurgitation. Autograft size. Biventricular function, scar volume and exercise capacity.

Results Mean autograft regurgitation was 6.8%±8.3% (trivial regurgitation) and diameter was 40.0±7.0 mm. Mean homograft velocity was 2.4±0.8 m/s (mild-moderate stenosis) and regurgitation was 6.1%±8.3% (trivial regurgitation). Biventricular systolic function was normal (LV EF 63.1±6.4% and RV EF 60.1±7.6%). In 38% of cases there was evidence of LV scar, mostly noted within the inter-ventricular septum. The mean exercise capacity achieved was 87%±22% of predicted. There was no correlation between exercise capacity and ventricular function or scar.

Conclusion This study demonstrates minor autograft and homograft dysfunction in the majority of patients after the Ross procedure, associated with good ventricular function and exercise capacity. In addition, minor scar was present in a third of patients with no functional consequences.

INTRODUCTION
In the Ross procedure, aortic valve replacement is performed using the patient’s own pulmonary valve (autograft). A biological valve, such as a homograft, is then placed in the right ventricular outflow tract.1 The Ross procedure overcomes problems such as anticoagulation for mechanical prostheses and the limited durability of homograft valves in the aortic position.2 This makes it an attractive approach for the management of younger, active patients with aortic valve disease.

Meta-analysis of the Ross procedure suggests low re-intervention rates for both the autograft and the homograft.3 Nevertheless, several studies have demonstrated minor degrees of homograft and autograft dysfunction, which resulted in mild ventricular impairment.4 5 The functional consequence of this level of ventricular impairment has not been well described.

The primary aim of this study was to comprehensively assess homograft and autograft function in a large group of adult patients after the Ross procedure and relate it to (i) biventricular function and (ii) exercise performance. Homograft, autograft and biventricular function were assessed using a combination of cardiovascular magnetic resonance (MR) imaging and echocardiography. Exercise performance was determined using cardiopulmonary exercise testing (CPEX). The secondary aim of this study was to identify and quantify myocardial scar due to cardiac surgery in patients after the Ross procedure and assess its effect on ventricular function and exercise performance.

METHODS
Patient recruitment
Forty-five patients were recruited from a total of 75 patients who underwent the Ross procedure at our institution between February 1994 and June 2006 and are locally followed up. Inclusion criteria: (i) contactable by telephone for recruitment (n=72). Exclusion criteria were: (i) age ≤16 or severe developmental delay (n=16), (ii) internal cardiac defibrillator or permanent pacemaker (n=5), (iii) concurrent non-cardiac illness preventing MR or CPEX (n=5), (iv) claustrophobia (n=1), (v) unwilling to participate in the study (n=2). The study was carried out between July 2007 and June 2008. The local research ethics committee approved the study, and all subjects (and a parent/guardian in patients aged <18) gave informed consent.

Cardiovascular magnetic resonance imaging
MR imaging and post processing was performed using a 1.5 T MR scanner (Avanto, Siemens Medical Systems, Erlangen, Germany) and a dedicated MR workstation (Leonardo, Siemens Medical Systems, Erlangen, Germany).
Aortic and homograft dimensions
Single phase three-dimensional balanced steady-state free precession (b-SSFP) imaging was used to assess anatomy. This sequence was triggered in diastole and utilised a respiratory navigator. Vascular dimensions were measured from multi-planar reformats of the 3D data. Aortic dimensions were measured at the sinus of Valsalva and sino-tubular junction (ST). Homograft dimensions were measured proximally (immediately after the valve) and distally (immediately before the bifurcation).

Autograft and homograft flow
Autograft and homograft flow data were acquired using velocity-sensitive phase contrast mapping. Imaging planes were located at the sinotubular junction and at the midpoint of the homograft. Analysis of phase contrast data was performed as previously described.2 Autograft and homograft regurgitation fraction (RF) was calculated as percentage backward autograft/homograft flow over forward autograft/homograft flow. Trivial regurgitation was <10%, mild regurgitation was 11–20%, moderate regurgitation was 21–55% and severe regurgitation was >55%.

Ventricular volumes and function
Ventricular volumes were measured using b-SSFP cine imaging of the ventricles in the short axis. Measurement of end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), ejection fraction (EF) and myocardial mass was performed as previously described.4, 5, 7

Assessment of myocardial scar: late enhancement imaging
Scar imaging within the myocardium was performed with a phase-sensitive inversion recovery sequence 5–10 minutes after the administration of intravenous contrast (0.1 mmol/kg of gadolinium pentatate, Magnevist).8 Imaging included the short-axis stack and long-axis planes. The volume of scar within the myocardium was expressed as a percentage of the total left ventricular (LV) myocardial volume.

Echocardiography
Peak velocity across the autograft and homograft was measured using continuous wave Doppler (Vivid 7 GE system, General Electrics, Milwaukee, WI, USA). Diastolic function was assessed using pulse wave Doppler measurements of the peak early diastolic (E) and late diastolic (A) velocities at the mitral and tricuspid valve leaflet tips. Myocardial diastolic (E, A) velocities were measured using tissue Doppler imaging of the basal segments of the respective lateral/free walls.

Cardiopulmonary exercise testing
Cardiopulmonary exercise (CPEX) testing was performed on an electronically braked bicycle ergometer or standard treadmill with respiratory gas exchange analysis. A ramp protocol comprising an initial period of loadless cycling to permit equilibration was used. A period of active recovery (slow cycling) followed maximal exertion. Treadmill testing followed the Bruce protocol. Heart rate, blood pressure and oxygen saturation were monitored in all subjects for the duration of the test. Peak heart rate, blood pressure and workload (W) achieved were recorded. Continuous ECG monitoring for arrhythmia and ST-T wave changes was performed.

Statistical analysis
All data are presented as mean±SD or median and range. Statistical comparison was performed using an unpaired Student t test. Correlation between variables was assessed with Pearson’s test. Multivariate regression analysis (using a backwards model) was used to assess independent predictors of aortic size. A value of p<0.05 was considered statistically significant. Statistical testing and data analysis were performed with SPSS Version 16 (SPSS Inc).

RESULTS
Patient characteristics
In all cases, the pulmonary autograft was inserted into the LV outflow tract as a free standing root replacement and a pulmonary homograft was inserted into the RV outflow tract. Median age was 24.6 (range 16.9–32.2), median age at Ross procedure was 18.3 years (range 5.7–43.5), and median years after the Ross procedure was 8.1 (range 2–14.0). Original pathology was aortic stenosis in 78% of subjects, regurgitation in 15% and mixed valve disease in 9%. In 49% of patients the original valve was bicuspid. Ten of the 45 patients required re-intervention before this study (intervention on homograft n=4, autograft n=5, both n=1). Two patients underwent re-intervention after the study (homograft n=2).

Autograft anatomy and function
The mean aortic sinus diameter was 40.0±7.0 mm (<40 mm n=23, 40–50 mm n=18, >50 mm n=4). Aortic ST junction diameter was 39.2±7.2 mm (<40 mm n=25, 40–50 mm n=17, >50 mm n=5). On multivariate analysis the only independent predictors of aortic root dimensions (sinus and ST junction) were age at Ross procedure (negative predictor) and body surface area (positive predictor) (table 1). There was no statistically significant difference in aortic root dimensions between subjects whose original pathology was bicuspid (49%) versus tricuspid (51%) aortic valve disease (p=0.26 and p=0.27, respectively). The mean autograft RF was 6.8±8.3%. Trivial regurgitation was demonstrated in 37 (82.2%) subjects, mild regurgitation in 6 (13.3%) subjects, moderate regurgitation in 1 (2.2%) subject and severe regurgitation in 1 (2.2%) subject. The peak velocity across the autograft was 1.5±0.4 m/s (gradient 7.7±4.9 mm Hg). Forty-three (95.5%) subjects had a peak autograft velocity<2 m/s (normal), the remaining two (4.5%) subjects had a peak velocity between 2-5 m/s (mild—moderate stenosis). Aortic root size positively correlates with aortic RF (sinus r=0.49, p=0.001; ST r=0.55, p=<0.001).

Homograft anatomy and function
The mean proximal and distal homograft dimensions were 19.7±3.4 mm and 23.5±3.2 mm, respectively. Pulmonary artery dimension did not correlate with age, age at Ross procedure, years after the Ross procedure or body surface area (BSA) (absolute r<0.2, p>0.4). Mean homograft RF was 6.1%±8.3%.

Table 1: Predictors of aortic root size by univariate and multivariate analysis

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Univariate p value</th>
<th>Multivariate p value</th>
<th>ST junction Univariate p value</th>
<th>Multivariate p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.18</td>
<td>–</td>
<td>0.003</td>
<td>–</td>
</tr>
<tr>
<td>Age at Ross</td>
<td>0.001</td>
<td>0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Year post Ross</td>
<td>0.14</td>
<td>–</td>
<td>0.25</td>
<td>–</td>
</tr>
<tr>
<td>BSA</td>
<td>0.005</td>
<td>0.003</td>
<td>0.003</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

BSA, body surface area; ST, sino-tubular.
Cardiovascular surgery

Trivial regurgitation was demonstrated in 37 (82.2%) subjects, mild regurgitation in 4 (8.8%) subjects, and moderate regurgitation in 4 (8.8%) subjects. The peak velocity across the homograft was $2.4 \pm 0.6$ m/s (gradient $25.2 \pm 13.0$ mm Hg) on echocardiography. Twelve (26.6%) subjects had a peak velocity <2 m/s (normal), 26 (57.7%) subjects had a peak velocity between 2-3 m/s (mild–moderate stenosis) and 7 (15.5%) subjects the peak velocity was >3 m/s (moderate–severe stenosis).

### Ventricular dimensions and function

Left ventricular volumes and mass were slightly increased compared to published normal data (table 2), and moderately correlated with aortic RF (LVEDV: $r = -0.51$, $p < 0.001$, LVESV: $r = -0.52$, $p = 0.001$, LVEF: $r = -0.41$, $p = 0.005$, LV mass: $r = -0.51$, $p = 0.03$). Parameters of LV systolic and diastolic function were within the normal range (table 2). However, aortic RF negatively correlated with LVEF ($r = -0.34$, $p = 0.02$) and positively correlated with E/E_m ratio ($r = 0.35$, $p = 0.02$).

Right ventricular volumes and function were within normal limits (table 2). However, there was a moderate positive correlation between homograft RF and RVESV ($r = 0.35$, $p = 0.02$) and RVSV ($r = 0.51$, $p = 0.04$). Echocardiographic measures of RV diastolic function were normal. Indexed RV mass was slightly increased compared to published data (table 2) and correlated with homograft RF ($r = 0.31$, $p = 0.04$), peak velocity ($r = 0.30$, $p = 0.04$) and tricuspid E/A ratio ($r = -0.35$, $p = 0.05$).

### The presence of LV scar

Left ventricular scar was present in 17 (38%) subjects (table 3) with a mean volume of $5.7 \pm 4.9$ cm$^3$ (3.5%± 2.8% of LV myocardial volume). Septal scar was associated with dyskinesia in all cases. Comparing subjects with and without scar, there was no significant difference in aortic cross-clamp time for the Ross procedure ($p = 0.5$), LVEDV ($p = 0.9$), LVESV ($p = 0.8$), LVEF ($p = 0.65$), baseline ECG abnormalities ($p = 1.0$) or workload achieved on exercise testing ($p = 0.83$).

### Cardiopulmonary exercise testing

Five patients did not successfully complete CPEX testing (1 patient NYHA III, 4 patients NYHA I). Mean workload achieved was $213 \pm 64$ W (107%± 52% of predicted). Mean VO$_2$ max achieved was $31.5 \pm 9.1$ ml/kg/min (87%± 22% of predicted). Exercise capacity correlated with RV volumes (table 4), but not with homograft/autograft dysfunction, LV volumes or biventricular systolic or diastolic function (absolute $r < 0.25$, $p > 0.19$).

Systolic (>140 mm Hg) and/or diastolic (>90 mm Hg) hypertension at rest was detected in five subjects. Eleven subjects had a hypertensive response to exercise (systolic blood pressure>210 mm Hg). Peak systolic BP correlated with BSA ($r = 0.61$, $p < 0.001$) but not with age ($r = 0.14$, $p = 0.35$) and on multivariate analysis (including age at Ross and BSA) was also an independent predictor of sinus and ST junction diameter ($p = 0.08$, 0.06 respectively). Three subjects had changes consistent with coronary ischaemia (n=2) or arrhythmia (n=1) at peak exercise.

### DISCUSSION

The Ross procedure is a viable alternative to metallic aortic valve replacement. However, there are concerns regarding midterm and long-term homograft and autograft integrity. In this study, an adult population who had previously undergone the Ross procedure were assessed with a combination of MRI, echocardiography and CPEX. Only adult patients were recruited into this study to improve the homogeneity of the study population. The main findings were: (i) aortic root dilation associated with

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**Table 2** Ventricular volumes and parameters of biventricular systolic and diastolic function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>LVEDV (cm$^3$)</th>
<th>RVEDV (cm$^3$)</th>
<th>LVEF (%)</th>
<th>RVVEF (%)</th>
<th>LV mass (g)</th>
<th>RV mass (g)</th>
<th>E/a</th>
<th>E/E_m</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV</td>
<td>82.4±21.4</td>
<td>86.1±17.3</td>
<td>30.1±11.1</td>
<td>34.3±9.1</td>
<td>51.4±12.4</td>
<td>51.7±11.9</td>
<td>1.5±0.4</td>
<td>7.7±3.4</td>
</tr>
<tr>
<td>LVESV</td>
<td>31.0±11.1</td>
<td>34.3±9.1</td>
<td>51.4±12.4</td>
<td>51.7±11.9</td>
<td>82.4±18.1</td>
<td>19.1±8.5</td>
<td>1.3±0.3</td>
<td>7.1±2.9</td>
</tr>
<tr>
<td>LVSV</td>
<td>51.4±12.4</td>
<td>51.7±11.9</td>
<td>63.1±6.4</td>
<td>60.1±7.6</td>
<td>82.4±18.1</td>
<td>19.1±8.5</td>
<td>1.3±0.3</td>
<td>7.1±2.9</td>
</tr>
</tbody>
</table>
| LVEF            | 82.4±18.1       | 19.1±8.5       | E/a      | E/E_m     | E, peak early diastolic (E) to late diastolic (a) velocity ratio; E/E_m, E to early myocardial velocity ratio; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; LV, left ventricle; RV, right ventricle; SV, stroke volume.

**Table 3** Scar volume pattern and surgical aspects in patients with scar identified on MRI

<table>
<thead>
<tr>
<th>Scar volume (cm$^3$)</th>
<th>Scar as % of LV volume</th>
<th>Pattern within LV</th>
<th>Surgical aspects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.9</td>
<td>1.8</td>
<td>Apex (SE)</td>
<td>NA</td>
</tr>
<tr>
<td>9.1</td>
<td>9.9</td>
<td>Mid to distal septum (SE), apex (SE)</td>
<td>NA</td>
</tr>
<tr>
<td>3.3</td>
<td>1.9</td>
<td>Basal septum (FT)</td>
<td>Bleeding at autograft harvest</td>
</tr>
<tr>
<td>3.1</td>
<td>3.0</td>
<td>Basal septum (SE)</td>
<td>NA</td>
</tr>
<tr>
<td>1.9</td>
<td>1.4</td>
<td>Apex (FT)</td>
<td>NA</td>
</tr>
<tr>
<td>7.8</td>
<td>4.8</td>
<td>Basal septum (FT)</td>
<td>Bleeding at autograft harvest</td>
</tr>
<tr>
<td>6.3</td>
<td>4.5</td>
<td>Basal septum (FT) and apex (FT)</td>
<td>NA</td>
</tr>
<tr>
<td>2.9</td>
<td>1.4</td>
<td>Apex (FT)</td>
<td>NA</td>
</tr>
<tr>
<td>0.9</td>
<td>0.5</td>
<td>Mid septum (SE)</td>
<td>Anomalous coronary anatomy, no bleeding</td>
</tr>
<tr>
<td>2.5</td>
<td>1.9</td>
<td>Basal lateral wall (FT)</td>
<td>NA</td>
</tr>
<tr>
<td>19.0</td>
<td>10.2</td>
<td>Antero-septal (FT)</td>
<td>Small branch coronary vessel divided at autograft harvest</td>
</tr>
<tr>
<td>6.9</td>
<td>3.9</td>
<td>Basal septum (FT) and apex (SE)</td>
<td>Bleeding at autograft harvest</td>
</tr>
<tr>
<td>1.9</td>
<td>1.1</td>
<td>Apex (SE)</td>
<td>Minor bleeding at autograft harvest</td>
</tr>
<tr>
<td>4.0</td>
<td>2.8</td>
<td>Apex (SE)</td>
<td>NA</td>
</tr>
<tr>
<td>7.2</td>
<td>4.4</td>
<td>Antero-septal (FT)</td>
<td>Small branch coronary vessel divided at autograft harvest</td>
</tr>
<tr>
<td>4.9</td>
<td>3.1</td>
<td>Inferior-posterior (SE)</td>
<td>NA</td>
</tr>
<tr>
<td>6.7</td>
<td>3.5</td>
<td>Inferior (SE)</td>
<td>NA</td>
</tr>
</tbody>
</table>

**Table 4** Correlation between right ventricular volumes and parameters of exercise capacity

<table>
<thead>
<tr>
<th>Parameter</th>
<th>% VO$_2$ max</th>
<th>% Watts</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVESV</td>
<td>0.56</td>
<td>0.001</td>
</tr>
<tr>
<td>RVEF</td>
<td>0.53</td>
<td>0.001</td>
</tr>
<tr>
<td>RVSV</td>
<td>0.42</td>
<td>0.007</td>
</tr>
</tbody>
</table>

EDV, end-diastolic volume; ESV, end-systolic volume; p, statistical significance; r, Pearson’s correlation coefficient; RV, right ventricle; SV, stroke volume.

FT, full thickness; LV, left ventricle; NA, not applicable; SE, subendocardial.
age at Ross and BSA; (ii) mild autograft regurgitation leading to slight LV dilation, but normal LV function; (iii) mild homograft stenosis and regurgitation leading to increased RV mass, but normal RV volumes and function; (iv) left ventricular scar in a proportion of patients with no associated functional consequences; and (v) normal exercise capacity with no correlation to autograft or homograft dysfunction or ventricular function.

Re-intervention
Patients who had undergone re-intervention were included in this study. Therefore, the results reflect the natural history of the Ross operation (including re-interventions), rather than the outcome of the procedure alone. This has clinical importance in understanding prognosis and developing management strategies for these patients.

The high re-intervention rate compared to mechanical valve replacement does reduce the attractiveness of the Ross procedure. However, the increasing use of percutaneous valve procedures makes the prospect of re-intervention less daunting.7

The autograft and left ventricle
Unlike a homograft, an autograft is viable tissue with significant growth potential.10 Consequently, it should dilate during periods of somatic growth, particularly in childhood. However, studies have shown that the rate of dilation in childhood is out of proportion to somatic growth.11 This suggests that other factors (eg, histological damage and thinner walls compared to native aorta) accelerate autograft dilation during childhood. In this study, the only independent predictors of aortic size were age at Ross procedure, BSA and peak systolic BP. The inverse relation between aortic size and the age at Ross procedure could be explained by the fact that autografts placed in younger patients have longer exposure to dilatory stimuli released during periods of growth and are therefore more likely to dilate.12 13 The relation between aortic size and BSA may simply be due to the autograft responding to adult somatic growth. Finally, the relation between aortic size and peak systolic blood pressure may be due to repeated damage to the more fragile autograft during exercise.10 Importantly, the length of time after the Ross procedure is not a predictor of aortic size. This implies that the autograft does stabilise in adulthood.14 Nevertheless, aortic root dilation is a risk factor for dissection and does require follow-up.

In this cohort, autograft dilation is associated with mild aortic regurgitation, confirming the findings of previous studies.5 Aortic regurgitation in this population may be due to increased valve stresses, secondary to autograft dilation and stiffening.5 However, it should be stressed that the majority of patients had no or trivial levels of regurgitation.

In the majority of patients LV volumes and function were normal, which is in keeping with good autograft function. Nevertheless, aortic regurgitation was associated with mild LV dilation and reduced LVEF. This suggests that autograft dysfunction does result in ventricular remodelling and that long-term surveillance is necessary. As shown in this study, follow-up can easily be accomplished with routine MR examinations.

The homograft and right ventricle
It is well recognised that homografts are non-viable tissue, susceptible to calcific degeneration and contracture.15 This is particularly true in patients with tetralogy of Fallot and pulmonary atresia, in whom the implantation site is intrinsically abnormal. In the Ross procedure, the homograft is placed in a normal right ventricular outflow tract. This may explain the lower homograft re-intervention rate compared to patients with tetralogy of Fallot or pulmonary atresia.5 However, minor homograft dysfunction is present in patients after the Ross procedure.4 In this study, the main finding was increased blood flow velocity across the homograft. In most patients, this increase would not trigger re-intervention,7 and was probably secondary to mild valve thickening, or contracture and stiffening of the homograft. In a small proportion of patients, high homograft velocities were encountered, suggesting clinically significant homograft obstruction. Although re-intervention is necessary in this group, percutaneous pulmonary valve replacement has made this a less serious problem.7 As previously shown,4 homograft regurgitation was not a significant problem in this cohort.

However, there is evidence that homograft dysfunction did result in RV remodelling. As expected, homograft regurgitation caused mild RV dilation and a compensatory increase in RV mass. Increased velocity across the homograft only caused an increase in RV mass. In both cases systolic function was normal, suggesting that remodelling sufficiently compensated for mild homograft dysfunction. Previous studies have shown that RV hypertrophy leads to a reduced tricuspid E/a ratio.4 In this study, the opposite result is found, with E/a ratio increasing as RV mass increases. This could be explained by pseudo-normalisation of the E/a ratio in patients with high RV mass (ie, those with significant obstruction).

Myocardial scarring
In this study, myocardial scar was present in more than a third of the cohort. There are several possible explanations for scar within the inter-ventricular septum. For instance, during autograft harvest transection of small septal vessels may lead to local bleeding and ischaemia/infarction. Furthermore, methods employed to achieve haemostasis such as localised diathermy may contribute to scar formation. It has also been suggested that abnormal septal wall function after cardiac surgery is related to cardiopulmonary bypass time.16 However, in our cohort there was no significant difference in cross-clamp time that could explain the existence of scar. Apical scar probably reflects the insertion point of bypass vents.

Although myocardial scar was associated with wall dyskinesia, it was not associated with global ventricular dysfunction or reduced exercise capacity. Therefore, the small amount of scar present after the Ross procedure does not have any functional consequences. However, scar may represent a focus for arrhythmia,8 17 18 and this requires further study.

Exercise capacity
Exercise capacity was essentially normal in this cohort. Importantly, homograft and autograft dysfunction did not predict exercise intolerance. In part, this may be due to problems in detecting a correlation when the majority of patients only have mild homograft or autograft dysfunction. Nevertheless, it does seem that mild homograft or autograft dysfunction has little functional consequence. An interesting finding is the positive correlation between exercise capacity and RV volumes. This may represent a training effect, as many of the subjects in this cohort were active participants in aerobic exercise, which is known to lead to ventricular dilation.19

Exercise induced systolic hypertension was found in a proportion of patients. This was associated with increased BSA and may reflect a pathophysiological change in response to increased weight or intrinsic abnormalities of the aorta. As previously discussed, this may lead to increased autograft dilation.
Limitations
The main limitation of this study was the small cohort size that may have made it difficult to detect correlations. However, this cohort is significantly bigger than those in previous published studies and therefore incrementally provides new insights into the “post Ross” population. Another limitation is the medium-term follow-up time as it is possible that autograft/dysfunction may worsen with time. Therefore, in the future it will be useful to perform a long-term assessment of patients after the Ross procedure.

Conclusion
After the Ross procedure adult follow-up patients exhibit mild autograft and homograft dysfunction. However, this study demonstrates that autograft/homograft dysfunction is associated with compensatory ventricular remodelling and very good functional outcomes. Thus, the Ross procedure does provide a valid alternative for the management of aortic valve disease, particularly in younger patients. A further novel finding was the presence of myocardial scar in a proportion of patients after the Ross procedure. Although myocardial scar had no functional presence of myocardial scar in a proportion of patients after the studies, it is possible that autograft/dysfunction may worsen with time.

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