Quantitative assessment of homograft function 1 year after insertion into the pulmonary position: impact of in situ homograft geometry on valve competence

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Aims
To prospectively evaluate homograft function with cardiac magnetic resonance (CMR) imaging 1 year after insertion into the pulmonary position, and to assess the impact of in situ homograft geometry, surgical factors, and ‘intrinsic’ homograft properties on early valve incompetence.

Methods and results
A total of 60 patients (mean age 21 ± 10 years; 35 females) with congenital heart disease underwent pulmonary valve replacement with homograft insertion and were prospectively enrolled into a study protocol that included serial echocardiography and CMR 1 year after surgery. None of the patients had homograft stenosis but 10 (17%) had significant homograft incompetence (i.e. pulmonary regurgitation fraction ≥ 20% on CMR). A higher incidence of ‘eccentric’ pulmonary forward flow pattern (P < 0.001, Fisher’s exact test), more acute ‘homograft distortion angle’ (P < 0.001), larger relative ‘annular’ size (P < 0.01), and greater pre-homograft right ventricular outflow tract (RVOT) diameters (P = 0.01) at CMR was seen in those with worse homograft function. In a backward multivariate linear regression model, ‘eccentric’ pulmonary forward flow pattern (r_part = 0.36, P < 0.001), ‘homograft distortion angle’ (r_part = 0.31, P = 0.001), and pre-homograft RVOT diameter (r_part = 0.19, P = 0.03) were independently associated with the degree of pulmonary regurgitation (in %) at 1 year.

Conclusion
Using CMR, in this prospective cohort study, we have shown that significant valve incompetence is present in one-sixth of patients after homograft insertion into the pulmonary position, and that alterations in the in situ homograft geometry were associated with the likelihood of developing valve incompetence. These findings imply that mechanical factors may have an important impact on homograft performance.

Keywords
Congenital heart disease • Surgical pulmonary valve replacement • Conduit function • Magnetic resonance imaging

Introduction
Pulmonary regurgitation has been recognized as an important clinical problem in patients with congenital heart disease, and it can be associated with major adverse events (i.e. arrhythmia and death).¹,² Therefore, homografts are frequently inserted as early valve replacements (PVR) conduits to protect the right ventricle from detrimental volume overload.³–⁶

Previous studies have retrospectively assessed the effectiveness of homograft placement related to the freedom from re-intervention, secondary to conduit degeneration.³–⁷ However, only limited data exist on the actual valve function,³ which would more specifically
indicate whether the inserted homograft has successfully abolished pulmonary regurgitation. Furthermore, none of the studies to date have addressed the impact of mechanical factors associated with the right ventricular outflow tract (RVOT) morphology and the role of adaptive surgical techniques on homograft function over time. This information would be of particular clinical interest as it may influence the choice of PVR conduit (e.g., homograft, ContegraTM, Hancock) and surgical technique (e.g., RVOT aneurysm resection, plication) at the time of the PVR procedure in the field of congenital heart surgery.

In this study, we prospectively assessed homograft function with cardiac magnetic resonance (CMR) imaging and echocardiography, 1 year after homograft PVR. Furthermore, the impact of in situ homograft geometry, surgical factors, and ‘intrinsic’ homograft properties on early valve incompetence were studied.

**Methods**

**Patients**

Between June 2004 and February 2007, 60 patients (mean age 21 ± 10 years; 35 females) underwent PVR with homograft insertion and were prospectively enrolled into a study protocol that included serial echocardiography and CMR 1 year after surgery (Table 1). The clinical indication criteria for PVR have been described previously. The local research ethics committees approved the study protocol. Written informed consent was obtained from patients and parents/guardians as appropriate.

**Table 1 Patient characteristics**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient group (n = 60)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at homograft insertion (years), mean</td>
<td>21 ± 10</td>
</tr>
<tr>
<td>Sex (female/male), n</td>
<td>35 / 25</td>
</tr>
<tr>
<td>Body surface area (m²), mean</td>
<td>1.56 ± 0.31</td>
</tr>
<tr>
<td>Principal diagnosis, n</td>
<td></td>
</tr>
<tr>
<td>Tetralogy of fallot</td>
<td>45</td>
</tr>
<tr>
<td>Pulmonary atresia with VSD</td>
<td>6</td>
</tr>
<tr>
<td>Pulmonary atresia with intact ventricular septum</td>
<td>3</td>
</tr>
<tr>
<td>Critical pulmonary stenosis, ASD</td>
<td>3</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>2</td>
</tr>
<tr>
<td>Double outlet right ventricle</td>
<td>1</td>
</tr>
<tr>
<td>Number of previous surgeries, median (range)</td>
<td>1 (1–3)</td>
</tr>
<tr>
<td>Right ventricular outflow tract, n</td>
<td></td>
</tr>
<tr>
<td>Transannular patch</td>
<td>48</td>
</tr>
<tr>
<td>Homograft conduit</td>
<td>6</td>
</tr>
<tr>
<td>Pulmonary valvotomy, infundibular resection</td>
<td>5</td>
</tr>
<tr>
<td>Monocusp valve</td>
<td>1</td>
</tr>
</tbody>
</table>

Continuous data are presented as mean ± standard deviation.

**Table 2 Homograft details**

<table>
<thead>
<tr>
<th>Parameter</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Size (mm), mean</td>
<td>22 ± 2</td>
</tr>
<tr>
<td>Anatomic origin (pulmonary/aortic), n</td>
<td>59/1</td>
</tr>
<tr>
<td>Age of donors (years), mean</td>
<td>46 ± 14</td>
</tr>
<tr>
<td>ABO compatibility with recipient, n</td>
<td></td>
</tr>
<tr>
<td>Match</td>
<td>35</td>
</tr>
<tr>
<td>Mismatch</td>
<td>14</td>
</tr>
<tr>
<td>Unknown</td>
<td>11</td>
</tr>
<tr>
<td>Rhesus compatibility with recipient, n</td>
<td></td>
</tr>
<tr>
<td>Match</td>
<td>48</td>
</tr>
<tr>
<td>Mismatch</td>
<td>10</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
<tr>
<td>Total ischaemic time (in h), mean, n = 51</td>
<td>35 ± 18</td>
</tr>
<tr>
<td>Overall judgment of homograft quality, n</td>
<td></td>
</tr>
<tr>
<td>‘Fair’</td>
<td>1</td>
</tr>
<tr>
<td>‘Good’</td>
<td>22</td>
</tr>
<tr>
<td>‘Very good’</td>
<td>37</td>
</tr>
</tbody>
</table>

Continuous data are presented as mean ± standard deviation.

**General aspects of surgical technique**

Homograft insertion was performed by four cardiothoracic surgeons under routine cardiopulmonary bypass (CPB, 91 ± 33 min) on the beating heart with ascending aortic and bicaval cannulation at 31 ± 2°C. Aortic cross clamping with cold blood cardioplegia was used in five (8%) patients (resection of residual trabeculations, extensive enlargement of pulmonary arteries, closure of a patent foramen ovale, tricuspid valve annuloplasty, and aortic valve replacement).

The native main pulmonary trunk was dissected out and the branch pulmonary arteries were sized. A longitudinal incision was made into the proximal RVOT and any hypertrophied muscular trabeculations in the sub-junctional region were divided. In patients with aneurysmal RVOT patch, this area was excised (RVOT ‘re-fashioning’). Prior to insertion, every individual homograft (59 pulmonary, 1 aortic) was inspected by the operating surgeon and judged according to quality, also by challenging the leaflets with an injection of sterile saline solution (see Supplementary material, Video clip). The homograft was tailored in length and sutured to the distal pulmonary trunk using 5–0 Prolene. The proximal end of the homograft was inserted using continuous 4–0 Prolene.

Homograft details

Individual data on the homografts were obtained from four heart-valve banks based in the UK. Details included size of homograft, age, sex, and blood group of the donors (ABO and Rhesus). Information on the ‘total ischaemic times’ (i.e. time from cardiac asystole to antibiotic sterilization) and overall homograft quality were provided (Table 2). All homografts were stored in a dimethyl sulphoxide (DMSO) solution and were cryopreserved.
Review of patient charts
Perioperative data on the patients were reviewed (Table 3). To assess the patients’ inflammatory response to surgical homograft insertion, serial blood levels of C-reactive protein and white blood cell (WBC) counts were obtained in the early post-operative period.

Echocardiographic follow-up
Echocardiography was performed early post-operatively (100% completeness) and at 1 year (88% completeness) to assess homograft valve competence with colour flow Doppler. In addition, the peak RVOT and tricuspid regurgitation (TR) jet velocities were obtained from continuous-wave Doppler traces (VIVID 7, GE, Medical Systems, Milwaukee, WI, USA).9

Cardiac magnetic resonance imaging
Cardiac magnetic resonance was performed at 1 year post-procedure using a 1.5 T MR scanner (Avanto; Siemens Medical Systems, Erlangen, Germany). Data on pulmonary flows were acquired by use of a flow-sensitive gradient-echo sequence as described previously. The pulmonary regurgitation fraction (PRF) was calculated as per cent back-flow over forward flow (Argus; Siemens Medical Systems). A PRF value of >20% was considered to be significant homograft incompetence.

Indicators of homograft geometry in situ were obtained. First, the pattern of pulmonary forward flow was assessed. The area of pulmonary forward flow was outlined on the phase images and subsequently divided by the ‘annular area’ obtained from the respective modulus images (Open Source Osirix software). If the ratio was ≤0.5, the pattern of flow was considered to be abnormal and termed ‘eccentric’ (Figure 1). Second, retrospective-gated steady-state free precession cine MRIs were used in diastole to measure the angle between the proximal and distal anastomosis (homograft distortion angle) from the sagittal long-axis RVOT image plane (Figure 2). Thirdly, dimensions were measured at the proximal, mid- and distal level of the homograft, and in the RVOT just below the homograft from two perpendicular long-axis RVOT image planes. The corresponding diameters in situ were calculated using the following equation:

\[
\text{diameter} = 2\sqrt{\frac{sagittal\ dimension^2 + (axial\ dimension)^2}{8}}
\]

Subsequently, the proximal homograft diameter in situ (equivalent to ‘annular’ diameter) was divided by the original heart- valve bank diameter (i.e. ‘annular’ diameter measured by inserting a Hegar dilator) to determine relative alterations in ‘annular’ size.

Assessment of right ventricular (RV) volumes was performed by manual segmentation of short-axis cine images at end diastole and end systole (Argus; Siemens Medical Systems), and from these volumes, the RV ejection fraction (RVEF) was calculated.9

Statistics
Data are expressed as mean ± SD unless otherwise specified. Parametric data were compared using unpaired Student’s t-test and non-parametric data with Mann–Whitney U test. Categorical data were compared using Fisher’s exact test.

Linear regression analyses were performed to determine parameters that were associated with homograft incompetence (i.e. degree of PRF, in %) at 1 year. First, a univariate linear regression analysis was performed. Secondly, backward linear multivariate modelling was performed (probability of F-to-remove ≥0.10) only with the parameters that reached a P-value of ≤0.20 on the univariate analysis, after verifying the normal P-P plot of the regression-standardized residuals. Independent factors analysed included CMR indicators of homograft geometry in situ (eccentric pulmonary forward flow pattern; ‘homograft distortion angle’; homograft diameters; relative alterations in ‘annular’ size; pre-homograft RVOT diameter) and surgical parameters (patient’s age, sex, and body surface area (BSA); presence of either of the following diagnoses: pulmonary atresia, double outlet right ventricle (DORV) or truncus arteriosus; RVOT ‘re-fashioning’; additional procedures; CPB time; sequence order of homograft insertions (i.e. ‘learning curve’); post-operative WBC counts at Day 1). Additionally, homograft-related data were included to account for potential ‘intrinsic’ determinants of early valve incompetence (homograft size related to BSA of recipient; Rhesus compatibility; ‘total ischaemic time’ during tissue harvesting; overall homograft quality). Partial (also known as semi-partial) correlation coefficients (\(r_{ps}\)) were used to describe the actual associations.

In light of the large number of covariates, a P-value of <0.01 was used to infer statistical significance in the univariate linear regression analysis.

For all other tests, a P-value of <0.05 was considered statistically significant. Statistical analysis was performed on SPSS 15.0 (SPSS Inc., Chicago, IL, USA) and GraphPad Prism 5 (GraphPad Software, San Diego, CA, USA). The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results
Patient characteristics
In this series, 60 patients underwent homograft insertion into the pulmonary position (Table 1). Patients had undergone a median of
one previous cardiac surgery (one to three). The most common previous repair was transannular patch placement \((n = 48, 80\%)\), and most patients had aneurismal RVOTs \((n = 44, 73\%)\). Significant pulmonary regurgitation was present in 58 (97\%) patients.

**Operative details**

Homografts were placed in anatomical position in 58 (97\%) patients. RVOT ‘re-fashioning’ was performed in 43 (72\%) patients. Plication of the RVOT—to help create a better fit for the proximal anastomosis at the ventriculo-arterial junction—was mentioned in 15 (25\%) patients. For the same purpose, a hood extension of the RVOT with additional patch material was performed in six (10\%). In most patients \((n = 57, 95\%)\), the muscular rim of the homograft was sutured in a sub-annular fashion for functional support. In three cases, the operating surgeons indicated issues that complicated homograft insertion: ‘short homograft’ \((n = 2)\) and ‘size discrepancy’ \((n = 1)\).

At the time of homograft surgery, ten additional procedures were performed to the pulmonary arterial (PA) tree. These procedures included patch enlargement of the pulmonary bifurcation \((n = 4)\), plasty of the right \((n = 2)\) and left \((n = 2)\) pulmonary arteries (both, \(n = 1)\), and left pulmonary artery dilatation with a 10 mm Hegar dilator \((n = 1)\).

**Serial echocardiographic assessment**

On the first documented echocardiography after surgical homograft insertion, 12 (20\%) patients showed ‘mild’ and 3 (5\%)}
showed more than ‘mild’ PR (2 ‘moderate’ and 1 ‘severe’). At 1 year after surgical homograft insertion, 7 (13%) patients showed ‘mild’ and 10 (19%) showed more than ‘mild’ PR. The proportion of patients with significant homograft incompetence (i.e. more than ‘mild’ PR on echocardiography) was greater at 1 year when compared with early post-operation (10 of 54 vs. 3 of 60 patients, \( P = 0.0365 \), Fisher’s exact test).

There was no indication of homograft stenosis at 1 year; on average, the peak RVOT and TR-jet velocities were low (1.9 ± 0.5 and 2.7 ± 0.5 m/s, respectively).

**Cardiac magnetic resonance assessment of homograft valve**

One-year after homograft insertion into the pulmonary position, 10 (17%) patients had significant valve incompetence (i.e. PRF >20% on CMR). A higher incidence of ‘eccentric’ pulmonary forward flow pattern (\( P < 0.001 \), Fisher’s exact test; Figure 1), more acute ‘homograft distortion angle’ (\( P < 0.001; \) Figure 2), larger relative ‘annular’ size (\( P < 0.01 \)), and greater pre-homograft RVOT diameters (\( P = 0.01 \)) at CMR was seen in those with worse homograft function. Furthermore, the RV end-diastolic volumes were higher in these patients (\( P < 0.01 \)), while the RV ejection was not significantly different when compared with patients with well-functioning homografts (\( P = 0.39 \); Table 4).

**Factors associated with homograft valve incompetence at 1 year**

On univariate linear regression analysis, five factors were associated with the PRF at 1 year (Table 5). Three variables were related to the homograft geometry in situ: ‘eccentric’ pulmonary forward flow pattern (\( r_{part} = 0.61, P < 0.001 \)), ‘homograft distortion angle’ (\( r_{part} = 0.59, P < 0.001 \)), and pre-homograft RVOT diameter (\( r_{part} = 0.26, P = 0.004 \)); two variables were related to surgical parameters: sequence order of homograft insertions (i.e. ‘learning curve’; \( r_{part} = -0.39, P = 0.002 \)); and post-operative WBC counts at Day 1 (\( r_{part} = 0.35, P = 0.007 \)).

After inputting all variables that reached a \( P \)-value of <0.20 on univariate linear regression analysis into a backward multivariate linear regression model, three variables were independently associated with the degree of PRF (in %) at 1 year: ‘eccentric’ pulmonary forward flow pattern (\( r_{part} = 0.36, P < 0.001 \)), ‘homograft distortion angle’ (\( r_{part} = 0.31, P = 0.001 \)), and pre-homograft RVOT diameter (\( r_{part} = 0.19, P = 0.03 \)).

We have further statistically tested potential interactions between these three parameters by building interaction terms. In this analysis, there was a stronger relationship (approximately five times) between pre-homograft RVOT diameter and subsequent degree of PRF (in %) in those who also showed ‘eccentric’ pulmonary forward flow pattern (\( P = 0.02 \)). Furthermore, the strength of the relationship between pre-homograft RVOT diameter and PRF increased incrementally according to the degree of ‘homograft distortion angle’ (\( P < 0.001 \)). No significant interaction was seen between ‘eccentric’ pulmonary forward flow pattern and ‘homograft distortion angle’ (\( P = 0.32 \)).

**Pre-operative right ventricular outflow tract morphology**

In a subset of patients (\( n = 27 \)), three-dimensional reconstructions of their RVOTs were acquired with CMR prior to homograft insertion. There was no visual difference in the pre-operative RVOT morphology (subjective assessment, examples shown in Figure 3) between patients who subsequently developed significant homograft incompetence and those who did not.

**Follow-up events**

During mean follow-up of 40 ± 10 months after homograft insertion, two patients with significant valve incompetence underwent redo PVR (one, homograft; one, Hancock prosthesis) and one patient underwent percutaneous pulmonary valve implantation (PPVI).\(^{11}\) Seven patients are under continuous medical follow-up with yearly CMR and cardiopulmonary exercise studies. To date, these patients have not fulfilled our clinical criteria that justify re-intervention.\(^{9}\)

**Discussion**

Using CMR, in this prospective cohort study, we have shown that significant valve incompetence is present in one-sixth of patients

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**Table 4 Data from cardiac magnetic resonance imaging at 1 year**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>PRF &gt;20% (n = 10)</th>
<th>PRF &lt;20% (n = 50)</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary regurgitation fraction (%), mean</td>
<td>30 ± 7</td>
<td>4 ± 5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Right ventricular end-diastolic volume (mL/m²), mean</td>
<td>119 ± 36</td>
<td>90 ± 20</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right ventricular ejection fraction (%), mean</td>
<td>51 ± 8</td>
<td>54 ± 8</td>
<td>0.39</td>
</tr>
<tr>
<td>Eccentric pulmonary forward flow pattern, ( n )</td>
<td>7</td>
<td>3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Homograft diameters in situ (mm), mean</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal homograft level</td>
<td>23 ± 4</td>
<td>20 ± 3</td>
<td>0.05</td>
</tr>
<tr>
<td>Mid homograft level</td>
<td>25 ± 5</td>
<td>23 ± 4</td>
<td>0.12</td>
</tr>
<tr>
<td>Distal homograft level</td>
<td>24 ± 4</td>
<td>23 ± 4</td>
<td>0.27</td>
</tr>
<tr>
<td>Relative alterations in ‘annular’ size (%), mean</td>
<td>11 ± 21</td>
<td>-6 ± 14</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Pre-homograft RVOT diameters (mm), mean</td>
<td>28 ± 5</td>
<td>24 ± 4</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Significant differences are given in bold. Continuous data are presented as mean ± standard deviation. PRF: pulmonary regurgitation fraction; Relative alterations in ‘annular’ size, difference between the proximal homograft diameter in situ and the original heart-valve bank diameter (in per cent of the original heart-valve bank diameter); RVOT, right ventricular outflow tract.
after homograft insertion into the pulmonary position, and that alterations in the in situ homograft geometry were associated with the likelihood of developing valve incompetence. These findings imply that mechanical factors may have an important impact on homograft performance. By taking technical factors and conduit type into account when performing PVR, it may be possible to improve surgical outcomes.

The search for the ‘optimal’ PVR conduit is an ongoing challenge in congenital heart surgery. To date, homograft insertion represents the longest and largest clinical experience (>40 years) and can be performed safely with low mortality.\(^6\) The documented freedom from homograft re-intervention varies between 61 and 90% at 5 years.\(^3\) In these studies, the most common reason for re-intervention is homograft stenosis, although homograft valve incompetence is reported to be almost ‘universally present’.\(^5\) Data from studies using other non-homograft PVR conduits (e.g. Contegra\(^\text{TM}\)) have, as yet, not shown superior results to those of homograft insertion.\(^12,13\)

The prevalence of significant homograft incompetence has probably been underreported in previous series.\(^2\) First, most studies have been retrospective, with no protocolized follow-up. And, secondly, the principal follow-up imaging modality has been echocardiography.\(^3–6\) While echocardiography evaluates valve function (i.e. leaflet coaptation) and represents a semi-quantitative method, CMR represents a truly quantitative technique to assess pulmonary incompetence.\(^14\) Furthermore, the opportunity to assess homograft geometry in situ with CMR provides novel insight into post-operative RVOT morphology.

Importantly, our findings suggest that mechanical factors may account for early homograft valve incompetence, with ‘eccentric pulmonary forward flow’ and ‘homograft distortion angle’ independently associated with this outcome. Anatomical ‘homograft kinking’ has been shown previously with X-ray angiography,\(^5\) and it has been speculated that ‘mechanical factors’ influence homograft longevity.\(^6\) However, our study is the first to systematically address homograft geometry in situ and to directly link indicators of ‘homograft distortion’ to a functional outcome. These findings suggest that attention to, and where necessary modification of,
technical issues may improve the outcome for surgical homograft insertion.

It is a recognized challenge of homograft insertion to adapt the patients’ RVOT and the homograft in such a way that the proximal anastomosis does not become distorted, particularly when RVOT restorative surgery is performed to reduce aneurismal dilatation. This is further complicated by the great heterogeneity of RVOT and pulmonary trunk morphologies seen in patients requiring PVR, which also impact on the distal homograft anastomosis. Thus, the surgical strategy for each patient has to be ‘individualized’.

In our surgical experience, following the excision of RVOT patch material, a ‘gap’ is created anteriorly between the RV and the pulmonary trunk. The most proximal part of this ‘gap’ is closed by suturing the edges of the RV together. Typically, the more distal part of the ‘gap’ (just below the homograft) is repaired by inserting a small hood, generally constructed from bovine pericardium. However, in some patients during the initial part of our series, this ‘gap’ was closed by pulling the muscular remnant of the homograft down into the gap. We believe that this manoeuvre distorted the valve annulus and may have accounted for some of the early homograft valve failures that we have observed.

In addition to the technical aspects described above, the fibromuscular support of the homograft may also play a role in valve competence (see Supplementary material online, Video clip). Routinely, the placement of the proximal homograft is performed within the newly reconstructed fibromuscular RVOT sleeve to make the homograft less susceptible to distortion, which can lead to better valve coaptation. However, valve leaflet distortion can occur if the homograft is stretched to fit the RVOT—as evidenced by the larger relative ‘annular’ size at 1-year CMR.

Reinforcement of the homograft valve with a rigid ring has been proposed as a surgical means to enhance annular support and to increase valve leaflet coaptation. This concept has been derived from previous experiences with the repair of atrio-ventricular valves, and also with bio-prosthetic valves, where the sowing ring acts as a rigid de-novo annulus that provides fixed ‘relative’ leaflet coaptation. To date, there are only limited data available on the use of bio-prosthetic valves in RVOT reconstruction, which do not allow for meaningful conclusions. In two of the patients in our group who had significant pulmonary regurgitation following homograft insertion (Patient 1, PRF = 38%; Patient 2, PRF = 44%), redo interventions (one, 25 mm Hancock prosthesis; one, PPVI) led to excellent valve competence at 1 year—PRF ‘none’ and 1%, respectively. These findings suggest that there may be a beneficial role of ‘stented’ conduits in RVOT reconstruction for individual patients. However, more comparative data are needed to determine their role in clinical practice. Interestingly, the current experience with PPVI suggests that the pulmonary valve competence is not a limiting factor for the mid-term success of this procedure and appears to be independent of the morphology of the implantation site.

It will be very important to follow this patient cohort in the mid- and long-term to help identify the impact of geometry/mechanical factors on homograft valve longevity. A recent study by Troost et al. reported that the echocardiographic homograft gradient at 1 month predicted subsequent valve degeneration, suggesting an interaction between flow mechanics, shear stresses, and degenerative mechanisms. Thus, unfavourable forward flow profiles secondary to altered homograft geometry in situ may, in the long-term, affect the rate of conduit degeneration. This phenomenon could also—in part—explain the reported shorter longevity of homografts placed in heterotopic positions. In a paediatric series following the Ross operation, where the homograft valve is located in a more anatomical position, the reported longevity was greater when compared with homograft valves placed in non-Ross patients.

Limitations

Homograft geometry in situ was assessed with the help of surrogate markers on CMR, though the whole complexity of three-dimensional interactions might not have been appreciated in full. Therefore, validation studies are required to corroborate our findings.

It is impossible to differentiate whether the ‘annular’ enlargement seen at the 1-year CMR was related to the homograft insertion technique or represents a consequence of an unfavourable forward flow profile, secondary to homograft distortion (‘vicious circle’). From the sequential echocardiography findings, there is some suggestion of progression in homograft valve incompetence throughout the study, favouring the latter explanation. It is of note that the original homograft measurement with the Hegar dilator might have had limited accuracy.

The subjective assessment of the pre-operative RVOT morphology was possible only in a subset of patients, leading to insufficient power to draw any quantitative conclusions.

Conclusions

This study demonstrates the moderate prevalence of early homograft valve incompetence and the impact of geometrical factors on this outcome. These findings emphasize that particular care has to be taken when inserting homografts in complex anatomies. Equally, thorough and structured follow-up assessment of patients after homograft insertion is recommended. As much as these conduits are an indispensable source for RVOT reconstruction in congenital heart surgery, they might not be the ‘optimal’ conduits for every anatomy. Further studies are required to help identify the means to enhance outcomes after homograft insertion and, equally, to individualize the use of particular PVR conduits in specific patients to improve the lifetime management of patients with RVOT lesions.

Supplementary material

Supplementary material is available at European Heart Journal online.

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Conflict of interest: none declared.

References