Mid-term experience with the Hancock porcine-valved Dacron conduit for right ventricular outflow tract reconstruction†

André Rüffer*, Johannes Wittmann†, Sergej Potapov*, Ariawan Purbojo*, Martin Glöckler†, Andreas Max Koch†, Sven Dittrich† and Robert Anton Cesnjevar‡

* Department of Paediatric Cardiac Surgery, University Hospital Erlangen, Erlangen, Germany
† Institute of Medical Informatics, Biometry and Epidemiology, Friedrich Alexander University Erlangen-Nuremberg, Erlangen, Germany
‡ Corresponding author. Department of Paediatric Cardiac Surgery, University Hospital Erlangen, Loschgestrasse 51, 91054 Erlangen, Germany.

Tel: +49-9131-8541962; fax: +49-9131-8534011; e-mail: andre.rueffer@uk-erlangen.de (A. Rüffer).

Received 31 August 2011; received in revised form 29 December 2011; accepted 4 January 2012

Abstract

OBJECTIVES: Surgical reconstruction of the right ventricular outflow tract (RVOT) often requires implantation of a valved conduit. A single-centre 10-year experience with the Hancock porcine-valved Dacron conduit was retrospectively assessed.

METHODS: The records of 63 patients who underwent RVOT reconstruction with Hancock conduit implantation between August 2000 and July 2010 were retrospectively reviewed. The median age was 13 years (range, 4 months to 64 years) and the median weight 44 kg (range, 6.5–75 kg). Fifty-one patients (83%) had previous cardiac surgery, and conduit replacement was performed in 31 patients (49%). Patient and conduit survivals with respect to factors precipitating conduit degeneration were analysed. Conduit failure was defined as severe conduit regurgitation or stenosis with a main pulmonary artery systolic gradient over 60 mmHg.

RESULTS: Early mortality was 4.8% and not related to conduit failure. Follow-up was complete with a mean duration of 3.5 ± 2.6 years. Patient survival after conduit implantation was 93 [95% confidence interval (CI), 87–100], 90 (95% CI, 81–100) and 85% (95% CI, 74–98) after 1, 3 and 5 years, respectively. Conduit failure occurred in six patients after a median of 5.6 years (range, 2.7–9.0 years). Freedom from conduit failure was 100, 96 (95% CI, 89–100) and 83% (95% CI, 62–100%) after 1, 3 and 5 years, respectively. Mean systolic gradient over the stenotic conduit valve was 87 ± 11 mmHg. Neither RVOT-aneurysm formation nor distal conduit stenosis occurred. Univariate analysis revealed younger age and absent pulmonary valve syndrome as risk factors for conduit failure (P = 0.01 and P < 0.01). Stepwise logistic regression identified higher white blood cell count at postoperative day 8 as a significant risk factor for conduit failure (odds ratio, 0.7; 95% CI, 0.52–0.89; P < 0.01).

CONCLUSIONS: The Hancock conduit is a valuable option for pulmonary valve replacement. It is not associated with RVOT-aneurysm formation or distal conduit stenosis. A persisting perioperative inflammatory reaction may be a predictor for later conduit failure.

Keywords: Right ventricular outflow tract • RVOT • Hancock conduit • Pulmonary valve replacement • Porcine valve

INTRODUCTION

Surgical reconstruction of the right ventricular outflow tract (RVOT) often requires implantation of a valved conduit. An ideal conduit should be easy to implant, resist valvular degeneration or calcification and should be associated neither with distal obstruction at the pulmonary artery bifurcation nor with proximal aneurysm formation at the right ventricular infundibulum. The conduit should grow with the patient and survive without anticoagulation. Unfortunately, grafts with all those features do not exist.

Since the late eighties, cryopreserved pulmonary homografts became the gold standard for pulmonary valve replacement [1]. However, their use is restricted to homograft banks, and the number of available allograft valves in stock is usually not sufficient to respond to all the surgeons’ requests, especially regarding smaller valve sizes [2]. Moreover, even homograft valves undergo degenerative changes like any xenograft, which finally leads to functional deterioration [1, 3–5].

Compared with pulmonary homografts, xenografts are available in all sizes off the shelf. The Hancock porcine-valved conduit (Medtronic Inc., Minneapolis, USA) was first used by Bowman et al. [6] in 1973 and has been on the market since then [7–15]. With some newer xenografts, a significant incidence of conduit-related complications, like distal conduit stenosis or right ventricular aneurysm formation, has been described [16–22]. Modern alternatives like tissue-engineered xenogenous valves seem still far away from being able to provide reliable implantation data [18, 20]. We recently reported our unsatisfying early results with a decellularized porcine pulmonary valve conduit [18].
The aim of this study was to analyse the durability of the ‘traditional’ Hancock conduit in the modern era at a single centre and to recognize determinants of conduit failure.

**MATERIALS AND METHODS**

**RVOT-conduit selection**

Our institution’s strategy regarding RVOT-conduit selection depends on individual surgical experience and has been focused on the Hancock conduit since 2009. Earlier, different types of valves and conduits, including the Hancock conduit, had been selected for RVOT reconstruction. Other valves implanted into the RVOT during the study period were: SupraAnnular SAV, \( n = 17 \) (Edwards Lifesciences LLC, Irvine, USA); Perimount CEP, \( n = 10 \) (Edwards Lifesciences LLC); Pericarbon MORE, \( n = 5 \) (Sorin Group, Milan, Italy); Pericarbon SOPRANO, \( n = 4 \) (Sorin Group); Mitroflow, \( n = 4 \) (Sorin Group); Pulmonary homograft, \( n = 4 \) (Eurotransplant, Leyden, the Netherlands); Contegra conduit, \( n = 34 \) (Medtronic GmbH, Meerbusch, Germany); Pulmonary valve conduit, \( n = 15 \) (Shelhigh Inc., Millburn, USA); Matrix P, \( n = 8 \) (AutoTissue GmbH, Berlin, Germany); Matrix P Plus, \( n = 16 \) (AutoTissue GmbH).

**Patient characteristics**

Between August 2000 and July 2010, 63 patients (41 males) underwent RVOT reconstruction with the Hancock conduits at the University Hospital Erlangen, Germany (Fig. 1). The median age at operation was 13 years (range, 4 months to 64 years), including four patients younger than 1 year and 10 patients older than 18 years. The median weight was 44 kg (range, 4.5–85 kg).

Cardiac diagnoses and indications for conduit implantation, or replacement, are given in Table 1. Nineteen patients (30%) received primary corrective surgery during the Hancock conduit implantation followed in this study. Sixteen patients (25%) had pulmonary stenosis or regurgitation on the basis of a previously implanted RVOT patch. Two patients developed pulmonary valve stenosis for other reasons: one patient with anomalous origin of the left coronary artery from the pulmonary artery following Takeuchi repair and another patient with an unbalanced atrio-ventricular canal after pulmonary artery banding.

Previous cardiac surgery had been performed in 52 patients (83%); most of the patients undergoing reoperation for Hancock conduit implantation in this study had their third sternotomy (\( n = 22; \) 42%). Thirty-one patients (49%) were scheduled for conduit replacement; previously implanted valves for RVOT reconstruction were: six Contegra Bovine Jugular Vein (Medtronic GmbH), two S.A.V. Aortic Porcine (Edwards Lifesciences LLC), two Carpentier-Edwards Perimount Pericardial Aortic (Edwards Lifesciences LLC), six Matrix P Plus (AutoTissue GmbH), eight Hancock conduits, two Pericarbon Freedom Stentless Aortic (Sorin Group), four homografts and one undefined bioprosthesis. Median survival of these explanted conduits was 5.9 years (range, 2 months–14.9 years).

**Surgical technique**

The surgical technique for RVOT-conduit implantation and postoperative management has been described previously [18]. In the absence of intracardiac communications or concomitant corrective surgery, conduit implantation or exchange was performed on the beating heart. Myocardial protection, if necessary, was achieved by intermittent cold crystalloid cardioplegia. The

**Table 1: Cardiac diagnoses and indications for conduit implantation or replacement**

<table>
<thead>
<tr>
<th>Cardiac diagnosis</th>
<th>N</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>TOF/DORV</td>
<td>17</td>
<td>27</td>
</tr>
<tr>
<td>PA-VSD</td>
<td>15</td>
<td>24</td>
</tr>
<tr>
<td>PS</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>APV</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>TAC</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>PA-IVS</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>TGA-PS</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>D-TGA-PS</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>L-TGA-PS</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Miscellaneous PS</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

APV: absent pulmonary valve syndrome; AV: Aortic valve pathology (aortic stenosis or regurgitation); PA-IVS: pulmonary atresia with intact ventricle septum; PA-VSD: pulmonary atresia with ventricular septal defect; PS: pulmonary stenosis; TAC: truncus arteriosus communis; TGA/PS: transposition of the great arteries with pulmonary stenosis; TOF/DORV: Tetralogy of Fallot or double outlet right ventricle.
conduit metallic ring around the Dacron tube securing the stented porcine valve was left in place in all patients.

Ten patients (16%) had primary RVOT surgery, five of them in the context of a Ross procedure. The Hancock conduit was placed between the right ventricle and the pulmonary artery bifurcation in all except four patients with corrected transposition, ventricular septal defect and pulmonary stenosis, where it was sutured to the left ventricle during conventional correction. The choice of conduit size was adapted to the patient's anatomy, age, body surface and matched PA diameter. The median conduit size was 22 mm (range, 12–25 mm; Fig. 2). All patients received i.v. heparin after implantation of RVOT conduits in the early postoperative period. Oral low-dose antiplatelet medication with aspirin 2 mg/kg/day was started on the first postoperative day and continued for 3 months.

**Follow-up**

Following Hancock conduit implantation, all patients received echocardiography including evaluation of conduit function before hospital discharge. Follow-up was complete, with a mean duration of 3.5 ± 2.6 years (median, 2.6; range, 0.3–10.7 years) in March 2011 (patient-years) and was accomplished by routine control echocardiography in our hospital or by direct contact with the referring cardiologist.

Pulmonary valve regurgitation was classified as trivial, moderate or severe according to features of the jet, and assessed with pulsed flow or colour Doppler in the parasternal short-axis view. In the case of significant pulmonary valve, incompetence calculated indexed right ventricular volume load was measured by magnetic resonance imaging. Conduit stenosis was allocated to its origin (muscular, valvular or at the PA bifurcation) and quantified by measuring the peak velocity flow through the conduit with the continuous-wave Doppler technique in the parasternal short-axis view. Conduit failure was attributed to the date of diagnosis by echocardiography or catheter and was defined as severe conduit regurgitation or stenosis with a main pulmonary artery systolic gradient over 60 mmHg. Additionally, aneurysm formation in the subpulmonary ventricle was assessed.

Significant conduit stenosis assessed by echocardiography was first addressed by invasive cardiac catheterization and consecutive balloon dilatation whenever eligible. In the case of further prevalence of a significant gradient over the conduit, interventional or surgical valve replacement was scheduled depending on patients' anatomy: distal conduit stenosis was referred to surgery, whereas conduit stenosis on a valvular level has been treated by interventional transcatheter pulmonary valve implantation since 2008 (Melody, Medtronic Inc.).

**Laboratory analysis**

Blood levels of systemic inflammation or autoimmune reaction [C-reactive protein (CRP) and white blood cells (WBCs)] were measured preoperatively and for two weeks after surgery.

**Statistical analysis**

Pre- and postoperative data were retrospectively collected. Descriptive data for continuous variables are presented as the mean ± standard deviation or as median with range; categorical variables are presented as numbers or percentages. The probabilities of overall survival and freedom from conduit failure were estimated according to the Kaplan–Meier method. The potential predictive factors of the Hancock conduit failure were identified using the log-rank test for categorical variables and Cox proportional hazard analysis for continuous variables. In addition, patients with conduit failure were compared with those without conduit failure regarding laboratory levels of inflammatory response, and the difference in mean or median levels was calculated for each day. The 'last observation carried forward' method was applied to complete missing levels of systemic inflammation. To exclude the influence of acute perioperative inflammatory response triggered by extracorporeal circulation, the association between late postoperative (days 7–14) levels of inflammation and conduit failure was calculated by stepwise logistic regression. The strength of associations was expressed as the odds ratio (OR) with 95% confidence interval (CI). All statistical analyses were conducted using the software R version 2.12.2 (R Foundation for Statistical Computing, Vienna, Austria). Statistical significance was defined at a P-value of <0.05.

**RESULTS**

**Patient survival**

Early mortality was 4.8% (n = 3) and not related to conduit failure. Mean hospital stay of survivors was 13 ± 8 days. Three patients died late; in one patient, death was associated with conduit failure. Patient survival after conduit implantation (including early mortality) was 93 (95% CI, 87–100), 90 (95% CI, 81–100) and 85% (95% CI, 74–98) after 1, 3 and 5 years, respectively (Fig. 3A). Causes of death are given in Table 2.

**Conduit failure**

Conduit failure occurred in six patients after a median duration of 5.6 years (range, 2.7–9.0 years). Freedom from conduit failure was 100, 96 (95% CI, 89–100) and 83% (95% CI, 62–100) after 1, 3 and 5 years, respectively (Fig. 3B). During a 10-year study
Figure 3: The Kaplan–Meier 5-year analysis. Broken lines indicate the 95% confidence bands. (A) Patient survival and (B) actuarial freedom from conduit failure.

Table 2: Cardiac diagnoses and causes of death

<table>
<thead>
<tr>
<th>Cardiac diagnosis</th>
<th>Operation</th>
<th>Conduit size (mm)</th>
<th>Age (years/months)</th>
<th>Death (days after operation)</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOF</td>
<td>Conduit exchange, PA plasty</td>
<td>18</td>
<td>3/9</td>
<td>1</td>
<td>Thrombosis of sinus venosus, brain dead</td>
</tr>
<tr>
<td>First operation: AP shunt, PA plasty</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second operation: correction with</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>conegra (14 mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interventions: RPA stent, LPA stent,</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dilatation of distal conduit stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOF-MAPCAs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First operation: AP shunt</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second operation: RVOT patch</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First intervention: RPA stent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DORV, coronary artery, Cantrell</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>syndrome, exomphalos</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First operation: AP shunt</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second operation: AP shunt</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tracheostoma and ventilationLPA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>hypoplasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d-TGA, VSD, PS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First operation: Rastelli at the age of 11 years (abroad)</td>
<td>Winter才</td>
<td>25</td>
<td>21</td>
<td>93</td>
<td>Unknown reason (abroad)</td>
</tr>
<tr>
<td>First operation: Rastelli at the age of 11 years (abroad)</td>
<td>Winter才</td>
<td>25</td>
<td>21</td>
<td>93</td>
<td>Unknown reason (abroad)</td>
</tr>
<tr>
<td>TOF-absent pulmonary valve, PA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aneurysm, tracheomalacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cc-TGA, VSD, PS, moderate TR,</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>complete heart block and DDD-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pacemaker, glomerulonephritis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conventional correction, LVOT conduit</td>
<td></td>
<td>16</td>
<td>64</td>
<td>1435</td>
<td>Renal failure</td>
</tr>
</tbody>
</table>

AP: aortopulmonary; cc-TGA: congenitally corrected transposition; d-TGA: dextro-transposition of great arteries; DORV: double outlet right ventricle; ECMO: extra-corporal membrane oxygenation; LPA: left pulmonary artery; PA: pulmonary artery; PS: pulmonary stenosis; RPA: right pulmonary artery; RVOT: right ventricular outflow tract; TOF: tetralogy of Fallot; VAD: ventricular assist device; VSD: ventricular septal defect.
period, the total incidence of Hancock conduit failure among hospital survivors was 10%.

Early postoperative and last follow-up echocardiographic gradients over the conduit are shown in Fig. 4. There was no correlation between elevated follow-up gradients and early postoperative data.

The reason for conduit failure was conduit stenosis in all patients. Mean systolic gradient over the stenotic conduit valve was $87 \pm 11$ mmHg. Site of obstruction was found to be exclusively at the valvular level. Neither RVOT-aneurysm formation nor distal conduit stenosis at the pulmonary artery bifurcation occurred.

Only two patients with conduit failure had to be reoperated on. In the first patient, conduit replacement was performed with a Shelhigh (Shelhigh Inc., Union, USA) porcine pulmonary valve before the Melody transcatheter pulmonary valve implantation technique was commenced in our hospital. The reoperation indication in the second patient receiving the Hancock conduit again was stated to be due to an additional residual ventricular septal defect. Explanted conduits showed massive calcification of valve leaflets without signs of tubular narrowing or stenosis of the anastomotic site (Fig. 5). Histological examination revealed strongly calcified cusps with focal fibrinous and haemorrhagic signs of inflammation. In two patients, interventional implantation of a Melody transcatheter pulmonary valve was successful. One patient died because of a malignant arrhythmia during diagnostic cardiac catheterization in another institution. One patient with corrected transposition and conventional correction developed elevation of gradients over the LV-PA conduit which was well tolerated. Induction of septal shifting positively influenced right ventricular geometry and prevented deterioration of tricuspid valve regurgitation.

**Risk factors for conduit failure**

Diagnosis-related conduit survival was significantly worse for patients with absent pulmonary valve syndrome (log-rank, $P < 0.01$). Additionally, there was a non-significant trend towards the higher risk of conduit failure among patients with conduit size ≤16 mm (log-rank, $P = 0.056$). Cox proportional hazard analysis revealed younger age as a significant risk factor for conduit failure ($P = 0.01$; HR, 0.8; CI, 0.66–0.96). Four patients were younger than 1 year receiving a 14- ($n = 2$), 16- and 18-mm valve; conduit failure has occurred in only three patients after 1051 days, so far. Ten patients had conduit sizes ≤16 mm and the mean follow-up was similar to those of our whole study cohort; two died early and two of the remaining eight patients were diagnosed with conduit failure after 1051 and 2177 days during follow-up. There was no influence of other types of cardiac malformation, weight and operative parameters on conduit failure. Stepwise logistic regression identified higher WBC count at postoperative day 8 as a significant risk factor for conduit failure (OR, 0.7; 95% CI, 0.52–0.89; $P < 0.01$), whereas there was no significant difference regarding postoperative median CRP levels between patients with or without conduit failure (Fig. 6).

![Figure 4](image4.png)

**Figure 4:** Development of echocardiographic gradients (mmHg) over the conduit measured in each patient postoperatively before discharge and at the last follow-up (calculated from the velocity time integral). The horizontal line at 60 mmHg marks the threshold of conduit failure.

![Figure 5](image5.png)

**Figure 5:** The explanted Hancock conduit after 4.9 years in a patient with conduit stenosis on the valvular level and additional residual VSD. Leaflet calcifications as seen from above (A) and below (B) the valvular level.
DISCUSSION

This study presents our mid-term experience using the ‘traditional’ Hancock conduit for RVOT reconstruction in the current era. It is important to note that we identified conduit failure as a follow-up gradient higher than 60 mmHg between the subpulmonary ventricle and the distal pulmonary artery branches. Since an increase in right ventricular pressure is well tolerated in most patients usually, indication and timing of conduit replacement are very sensitive matters and differ between institutions [11]. Moreover, the possibility of transcatheter valve implantation decreases the need for surgery. The probability of conduit replacement after implantation of a Hancock conduit in this study remains relatively, low affecting only two of all hospital survivors (3.3%) during a 10-year study period. Freedom from conduit failure was 85% at 5-year follow-up; one late death occurred during cardiac catheterization in a patient with suprasystemic right ventricular pressures. These results are consistent with contemporary results from France where no graft-related late death occurred, and survival with freedom from conduit reoperation was 81% at 5 years [7]. Older reports regarding 5 years freedom from porcine-valved Dacron conduit replacement ranged from 70 to 94% [10–12, 14].

Comparing these results with larger historical series with homografts, one could be tempted to suggest that the reported mid-term performances of the latter grafts do not seem to be much superior. Tweddell et al. [3] and Stark et al. [1] reported that freedom from homograft failure with the need for reoperation was 74 and 84% at 5 years after implantation, respectively. For the same period regarding non-Ross patients, even 60% homograft dysfunction in the RVOT was published [4]. On the other hand, the long-term results of a recent study including 509 allografts revealed that freedom from conduit replacement was 89% at 10 and 81% at 15 years [5].

Examining both homo- and heterografts, and taking valve diameter into account, Lange et al. [8] reported similar conduit exchange rates for diameters of 12–14 mm. However, the same group underlined superior 10-year survival for larger homografts. Comparing the contegra conduit with homografts recently, different experiences have been published: Urso et al. [22] concluded that the contegra conduit in the RVOT is an independent risk factor for graft replacement, whereas Fiore et al. [23] suggested that the early performance of small contegra conduits in children <2 years of age may be more advantageous than homografts. The latter study leads us again to the suggestion that smaller homografts may not be advantageous over xenografts. Considering these results, we do not hesitate to use the Hancock conduit even in newborns with elevated pulmonary vascular resistance where implantation of a valved RVOT conduit is necessary. Especially in this patient group, alternative xenografts have shown the risk of proximal RVOT-aneurysm formation, thrombosis or distal conduit stenosis at the PA bifurcation [16–21]. None of these complications were observed in our study population.

Univariate risk analysis of the present study group revealed higher probability of conduit failure among patients with absent pulmonary valve syndrome. Moreover, according to the observation of others [8, 11, 12, 15], age was a significant risk factor for increased conduit failure. The literature has pointed out the diagnosis of truncus arteriosus as a risk factor for conduit failure or late mortality [4, 8]. In addition, conduit size is an independent predictor of early conduit-related intervention, most likely resulting from patient outgrowth of the implanted valve [4, 8, 15]. We admit that the high amount of larger conduit sizes (81% of all implanted conduits were larger than 18 mm) may be responsible for the favourable outcome with a very low reoperation rate [11]. Consistent with the observation of Belli et al. [7] reporting that oversizing was more likely to be associated with longer conduit life, our policy is to implant the biggest diameter as possible. However, it is of major importance to place the conduit with the containing valve away from the sternum in order to prevent squeezing of the conduit by sternal compression [7, 9, 12–14]. Therefore, we routinely open the left pleural space allowing the conduit to shift to the left side away from the midline. The conduit valve is placed as distally as possible, without removing the metallic ring. Thus, neither sternal compression nor coronary artery compression has been observed. It is important to note that transcatheter pulmonary valve implantation is facilitated in the Hancock conduit by the presence of the metallic ring that generally avoids prestenting of the conduit.
In contradiction to observations on with homograft failure, and consistent with other studies examining Hancock conduit performance, valve regurgitation during follow-up was absent or mild [7, 11]. The main reason for Hancock conduit dysfunction was degeneration or calcification of the valve with development of valvular stenosis [7, 9, 12, 13]. The type and location of conduit dysfunction is of major importance regarding the management of conduit reintervention [24]. Valvular Hancock conduit stenosis is easily accessible for future transfemoral valve implantation. Since 2008, we have performed implantation of the melody transcatheter pulmonary valve in our institution. In all patients with Hancock conduit dysfunction and no other indication for surgery (n = 2), interventional valve implantation was successful. The technique implies a short window of conduit diameter between 16 and 22 mm. Due to the development of a fibrous peel in the conduit [9], the effective valve diameter shrinks over time. Therefore, we prefer to implant even larger sizes (25 mm) with respect to the valve-in-valve concept. In contrast to other authors [9, 14], we did not observe the haemodynamic relevance of intraluminal fibrous peel formation in the conduit. The routine prescription of low-dose aspirin after conduit implantation may possibly be one reason for the diminished formation of obstructive fibrous peel by inhibited platelet aggregation between the peel and the conduit.

Inflammation seems to play an important role in the pathogenesis of conduit degeneration as patients with elevated WBC count at 1 week after Hancock conduit insertion were at higher risk of later conduit failure, or more precisely, porcine valve degeneration. It has been well described that the implantation of bioprosthesis induces a xenograft-specific immune response. Both evidences for a ‘non-specific’ immune reaction with macrophages and granulocytes and ‘specific’ immune reaction to xenograft protein with lymphocytes and plasma cell rich infiltration have been described [17–19]. The persisting elevated WBC count beyond 1 week after conduit insertion in our patient group with future xenograft failure suggests a primarily ‘non-specific’ immune reaction. Konacki et al. [25] showed that the α-Gal epitope on porcine valves may be responsible for a specific immune response. The study is intriguing as it suggests the completely genetic manipulation of α-Gal-deficient xenograft or pretreatment with α-galactosidase, might reduce the immune response against bioprostheses and extend durability.

Limitations of this study include the typical disadvantages of retrospective single-centre studies with a limited patient population. Nevertheless, we encourage using the Hancock porcine valve for RVOT reconstruction as a good alternative to cryopreserved homografts, especially as a scaffold for future transfemoral pulmonary valve replacement leading to a decreased need for reoperation.

Conflict of interest: none declared.

REFERENCES
APPENDIX. CONFERENCE DISCUSSION

Dr M. Danton (Glasgow, UK): As you state, the Hancock valve conduit is nearing its fourth decade. It is well known. It is not perfect, which would suggest the alternatives are not perfect either. Therefore, RV-PA conduit reconstruction remains a contemporary, important, and relevant issue. There are some limitations of this study: 65 patients is perhaps a small study; the follow-up is short at 3.5 years mean, and there is a heterogeneity of the congenital lesions and position of the conduit. Nevertheless, you have raised some interesting issues. Six patients developed conduit failure resulting in 80% freedom from reoperation at 5 years, and this accords with other studies. But this may give an overly optimistic view of the situation. We might expect the attrition rate to increase in the five to ten year period. Also, this was a predominantly adolescent age group, and the challenges of outgrowth are probably less.

It is interesting that all conduits failed by stenosis. You had no regurgitation, and a possible explanation for this may be the stented valve, stented both by the ring and by the semi-rigid Dacron, which does reinforce the position of the valve. It makes it a little bit more resilient to the constraints of the implantation. And this contrasts with the pure biological valves like homograft and Contegra which are easier to use and fit the space, but the valve is not supported and these failed both by regurgitation and stenosis. I have three questions.

Turbulence or lack of laminar flow leads to energy loss and may incite a local inflammatory reaction at the site of the valve. Did you find any relationship between the postoperative echo, velocity time integral, and the conduit failure?

Dr Rüffer: We tried to find a correlation, but the data were not complete enough to make a statistical analysis meaningful. But, of course, there was a trend. In the beginning, the gradient was the same as postoperatively, and then increased in the later years.

Dr Danton: Yes. You have covered this in your presentation to a certain extent, but I would like a little bit more detail. In the conduit failures, you reintervened on four patients. That is a small number. But, nevertheless, can you describe to us some of your experience. Two underwent standard conduit replacement, and two underwent a Melody valve implantation. In those patients who had the Melody valve implantation, how successful was it in terms of what was the original conduit size, what size valve were you able to implant, and what was the change in the postoperative haemodynamics?

Dr Rüffer: We oversize intentionally in order to make the Melody valve implantation possible in case of future conduit failure over the next 10 years. It is well known that in the Hancock conduit, some peeling formation may occur, leading to a tubular narrowing. Therefore it is better to have a little bit larger conduit, at least 22 mm. If the diameter shrinks, then a 20 mm valve will be accessible. Does that answer your question?

Dr Danton: I was trying to find out the direct experience of putting the valve in and how that changed postoperative gradients. Because putting a valve inside an already stenotic valve may not be a perfect solution, whereas replacing it with a fresh conduit may give you a better haemodynamic result. I am just interested to know what your experience was.

Dr Rüffer: But you have the risk of reoperation.

Dr Danton: Sure, I accept that.

Dr Rüffer: The Hancock conduit is really a very good layer for the Melody valve, and we believe that there is almost no reason not to perform this procedure.

Dr Danton: That is fair argument. Okay. My last question, you did identify an interesting association between the early white cell count and the CRP elevation and the later conduit failure, and you speculated in your discussion that there may be some immunological process taking place. Just going on from that, 16 patients had previous porcine grafts which may sensitize the patient to a later porcine implantation. Did you find any relationship between previous porcine grafts and raised white cell counts?

Dr Rüffer: Regarding the last question, despite the low number of 63 patients, as you said, we tried to perform a risk analysis. According to our results, only absent pulmonary valve was a significant marker for future conduit failure. We also expected reoperation or previous conduit implantation to be potential risk factors, but it did not come out.

Dr G. Sarris (Athens, Greece): I noticed in your patient age distribution, and rather analogously in the size distribution of the conduits implanted, that most were larger conduits.

Dr Rüffer: That is correct.

Dr Sarris: Based on your experience that you reported today, what is your current policy? What is your first choice of RV-PA conduit in the similar age group as you have described, but also, perhaps more importantly, in the younger age group, in the neonates. There was one patient I think that was six months old. What would your policy be when repairing a neonatal truncus? The Hancock conduit has been used, and we have used it for truncus repair and it is a good conduit, but there may be better tissue conduits available to do this. So what is your policy?

Dr Rüffer: We changed our policy after considering the literature and because of our results. For example, in comparing pulmonary homografts to the Hancock conduit, especially in the smaller sizes, no significant difference has been shown regarding long-term outcome, because, anyway, the patient grows out of the conduit size. Therefore, there is no reason to be reluctant to implant the Hancock conduit, a 12 mm Hancock in a truncus, for example. Currently we exclusively use Hancock conduits for RVOT reconstruction.

Dr Sarris: So you would use a Hancock conduit in a neonate to repair truncus?

Dr Rüffer: Exactly.

Dr R. Margaryan (Massa, Italy): You said you are routinely oversizing. Are you oversizing in both groups, adults and children? This is the first question. And, second, is your data supporting that oversizing is increasing the longevity?

Dr Rüffer: The bigger the conduit diameter, the greater the possibility that you can implant the Melody valve; therefore, we try to use a large conduit. There is no reason why we should implant a valve which has a smaller size. We choose the size which is adequate to the pulmonary artery bifurcation, but we do not exaggerate, for example using a 20 mm conduit in a neonate, of course not. We try to implant the largest adequate diameter conduit as possible.

Dr Margaryan: So how do you oversize, for example, a 3 kg or 5 kg patient?

Dr Rüffer: For a neonate, the 12 mm conduit is the smallest you can get, and this is already oversized.

Dr Margaryan: What about the 14 year old patient?

Dr Rüffer: In a 14 year old we would try to put in a 20 mm or even 22 if it is possible. Sometimes the patients have dilated pulmonary arteries, and then it is easier.

Dr Fragata (Miraflores-Alges, Portugal): From the German registry on the Ross operation, we have learned that when you oversize a conduit, this is indeed an inducing factor for early failure. So you do not gain a lot by oversizing. What do you think about that?

Dr Rüffer: According to the results of Belli and Serraf from last year in the Annals, analysing their experience with the Hancock conduit, the size of the implanted conduits was chosen to be as large as possible and this was not a risk for reintervention or pulmonary artery distortion or anything.

Dr Fragata: Definitely not for the homografts anyway.

Dr G. Ziener (Chicago, IL, USA): I just would like to make a comment about oversizing. I mean, we should not be too impressed by numbers like 22. A 22 mm Hancock is worse than an 18 mm homograft at best. And if we would give this talk on homografts and then 18 mm in this 14 year old, there is not so much oversizing anymore. Just as a comment.