Right ventricular outflow tract reconstruction using Contegra® valved conduit: natural history and conduit performance under pressure

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Abstract

Objective: To assess the performance of the bovine Contegra® valved conduit used for right ventricular (RV) outflow tract reconstruction, particularly in relation to post-operative RV pressure.

Methods: Follow-up study of 64 consecutive right ventricular to pulmonary artery-conduit implants in 62 patients between January 2000 and April 2003. The majority of cases were forms of pulmonary atresia/VSD (n = 24, 39%) or Fallot’s tetralogy (n = 13, 21%). Thirteen cases (21%) had aortic atresia, truncus arteriosus or discordant connections with pulmonary atresia/VSD. Twelve cases (19%) were conduit replacements. Echocardiography was performed for a median follow-up of 14 months (range 0—38 months). Results: Median age at implantation was 13.8 months (range 0.1—244 months) and median weight was 8.9 kg (range 2.1—84.1 kg). Thirty-eight patients (59.4%) were <10 kg at the time of surgery. Early mortality was 6.4% (n = 4). During follow-up there were four explantations (one for endocarditis and three for conduit dilatation) and 16 (28.6%) catheter interventions. Overall freedom from intervention at 1 and 3 years was 71%/6% and 53%/61%, respectively. Freedom from conduit-specific reintervention was 66%/61% at the end of the study period. Reintervention was associated with small conduits (p = 0.04), age < 1 year (p = 0.04) and with high RV/LV pressure ratio in the immediate post-operative period (p = 0.0003). On multivariate analysis, the RV/LV pressure ratio was the strongest single factor predicting the overall reintervention (OR 5.45). Acquired distal conduit stenosis at suture line was the commonest indication for conduit-specific reintervention and was associated with the smaller conduits. The conduits explanted for dilatation showed neointimal proliferation, thrombosis, calcification and chronic inflammation.

Conclusions: The Contegra conduit is widely applicable to RVOT reconstruction with satisfactory mid-term results. However, there is a significant incidence of conduit-related complications, particularly with the smaller conduits. Adverse performance was strongly associated with high RV/LV pressure ratio at completion of surgery. We would recommend cautious use of the conduits in patients with predicted high RV/LV pressure ratios, where careful monitoring of conduit performance is crucial. There is some element of unpredictability, which adds to the importance of close follow-up. Further studies are needed to explore the issues of thrombogenicity, degeneration, possible ‘rejection’, and the potential role of anti-platelet and anti-inflammatory modulation.

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Keywords: Bovine Contegra® valved conduit; Right ventricular outflow tract reconstruction; Right ventricular to left ventricular pressure ratio

1. Introduction

It is 40 years since Ross and Somerville [1] reported the successful use of human tissue graft valves, which broadened the scope for a possible durable conduit for right ventricular (RV) outflow tract reconstruction. A variety of prosthetic conduits have since developed, although homografts continue to be regarded as the most reliable option; they have, however, shown early degeneration and calcification, particularly in very young patients [2]. The lack of small-size availability, overall scarcity, and the concerns about their long-term outcome [3—5], have led to the development and evaluation of a pliable biological conduit.

The recently developed Contegra® valved bovine conduit (Medtronic Inc., Minneapolis, MN, USA) has been advocated for its ‘off-the-shelf’ availability, small sizes, surgical pliability and encouraging short-term success in experimental animal studies [6—10].
2. Methods

2.1. Patients

This is a prospective study of 64 consecutive, Contegra valved conduits used in RVOT reconstruction in 62 patients over the 38 months between January 2000 and April 2003 at Birmingham Children’s Hospital. Two patients had a second operation involving the insertion of a further Contegra conduit. Both patients therefore appeared twice in the conduit statistics.

Aspirin was used if additional patching had been performed on the pulmonary arteries; in 46 out of the 64 conduits (71.8%).

2.2. Study group

The patient group encompassed a broad spectrum of conditions and diagnoses. The patient details are given in Table 1, dividing patients into the main diagnostic categories. Of note, 18 patients (29%) with PA/VSD/MAPCA were included in the study, reflecting the referral pattern at this institution.

2.3. Surgical technique

Conduits were inserted using moderate hypothermic cardiopulmonary bypass and aortic cross-clamping, with intermittent doses of cold crystalloid cardioplegia for myocardial protection. The conduit was rinsed in saline according to the manufacturer’s guidelines prior to implantation. The native pulmonary arteries was extended if necessary to at least the same diameter as the chosen conduit. Distal anastomosis was performed using 6/0 continuous running polypropylene sutures with an evert technique. The proximal suture line (at the ventriculotomy) was performed with a layer of continuous 5/0 or 6/0 polypropylene suture (depending on the conduit size) reinforced with interrupted pledgetted sutures at the heel of the anastomosis. At chest closure a Gore-Tex® pericardial membrane was placed behind the sternum to reduce the likelihood of adhesion of conduit to the sternum.

Over half (60%) of the implanted conduits were of the smallest sizes (38% were 12 mm in size) (Fig. 1).

2.4. Post-operative follow-up

Data collection was from the clinical notes and from serial echocardiograms performed prospectively according to the study protocol (see below). Cardiac catheterisation was performed only when indicated by clinical and echocardiographic findings except for the PA/VSD/MAPCA patients who have an elective catheter at 3–6 months according to

<table>
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<th>Study group demographics</th>
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<tbody>
<tr>
<td><strong>Female/male</strong></td>
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<tr>
<td><strong>Age (months)</strong></td>
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<tr>
<td><strong>Weight (kg)</strong></td>
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<tr>
<td><strong>Diagnosis</strong></td>
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<tr>
<td><strong>Primary repair</strong></td>
</tr>
<tr>
<td><strong>Complex PA/MAPCA</strong></td>
</tr>
<tr>
<td><strong>PA/VSD with good size pulmonary arteries</strong></td>
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<tr>
<td><strong>Aortic atresia</strong></td>
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<tr>
<td><strong>Truncus Arteriosus</strong></td>
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<tr>
<td><strong>TGA/PA</strong></td>
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<td><strong>Fallot’s tetralogy</strong></td>
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<tr>
<td><strong>Pulmonary valve atresia</strong></td>
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<td><strong>Absent pulmonary valve</strong></td>
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<tr>
<td><strong>Anomalous LAD</strong></td>
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<tr>
<td><strong>Disconnected LPA</strong></td>
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<td><strong>Severe bilateral branch PA stenosis</strong></td>
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<tr>
<td><strong>Anomalous LAD + disconnected LPA</strong></td>
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<tr>
<td><strong>Conduit change</strong></td>
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<tr>
<td><strong>Total</strong></td>
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<td><strong>FU complete</strong></td>
</tr>
</tbody>
</table>

Table 1

for elective catheterisation. The RV/L V pressure ratio was persisted in the early post-operative period were put forward.

Gradient across the valve was considered significant. Pressure ratio/C21

When catheterisation was performed. Patients with RV/L V assessed at each echocardiographic examination based on 24 h post-operatively. Subsequently, RV pressure was directly in theatre and echocardiographically as above within In the immediate post-operative period, it was measured just distal to the Contegra just distal to the Contegra conduit valve/C21. R F/C21 was measured at the level of the valve, above the valve and below the valve. A mean of the three measurements was taken and was expressed as the percentage of diameter difference between the actual diameter and the manufacturer-labelled dimensions at implantation (DD%).

The degree of conduit regurgitation was qualitatively assessed by colour flow Doppler, graded 1–4 regarding grades 3 and 4 as significant. Regurgitant fraction (RF) was quantitatively assessed by pulsed wave Doppler at a point just distal to the Contegra conduit valve/C21. RF > 40% was considered significant.

Conduit valve stenosis was diagnosed if the mean pressure gradient across the valve was >30 mmHg.

RV pressure was assessed regularly throughout the study. In the immediate post-operative period, it was measured directly in theatre and echocardiographically as above within 24 h post-operatively. Subsequently, RV pressure was assessed at each echocardiographic examination based on tricuspid regurgitation velocity and measured invasively when catheterisation was performed. Patients with RV/L V pressure ratio ≥60% at the end of the operation that persisted in the early post-operative period were put forward for elective catheterisation. The RV/L V pressure ratio was divided into quartiles from 1 to 4 which were equivalent to ratio of <0.4 for quartile 1 through to a ratio of >0.6 for quartile 4. Pulmonary artery pressure was the pressure measured distal to the conduit and was divided into four quartiles, the highest being quartile 4 (equivalent to 38–88 mmHg).

2.6. Cardiac catheterisation

This was performed either electively for the PA/VSD/MAPCA group or when there was evidence of RVOT obstruction, branch pulmonary artery stenosis, significant conduit dilatation or unexplained impairment of RV function. The site of stenoses was classed as either conduit-related or branch pulmonary artery-related (i.e., distally). Further localisation of any stenosis within the conduit is described in Section 3.

The explanted conduits were sent for detailed histological examination by an independent pathologist who had specific experience with the Contegra valves and who was anonymous to the patients and the purpose of the study.

2.7. Data analysis

Actuarial survival, freedom from reintervention based on age, diagnostic group, RV/LV pressure ratio and PAP were estimated by the Kaplan–Meier method, using SPSS for Windows (version 11 Inc.). Sigma-plot software was used to plot survival curves.

Continuous variables were expressed as mean ± SD or median range, and binomial or ordinal data were expressed as percentages. Comparative univariate analyses were performed with the χ²-test, two-sided Fisher exact test or binomial logistic regression, as appropriate. A probability value p < 0.05 was taken to represent a statistically significant difference between groups.

The effect of pre-operative, operative and post-operative variables on the outcome was tested by univariate and multivariate analyses. Univariate analysis of early outcome measures was done using the tests as above and variables with p ≤ 0.1 were included in a stepwise logistic regression model. Results of the multivariate analysis were expressed as odds ratios (OR) with 95% CI for variables with p < 0.05.

2.8. Outcome measures

Outcome measures were early mortality (defined as death within 30 days of surgery), late death, survival, freedom from reintervention and NYHA functional status.

3. Results

Median age at operation was 13.8 months (range 0.1–244 months) and median weight was 8.9 kg (range 2.1–84.1 kg). Thirty-eight patients (59.4%) were <10 kg at the time of surgery. Clinical outcome was available for all patients with complete echocardiographic follow-up on 93.5% of survivors. Median follow-up was 14 months (range 0.3–38 months).

Early (30-day) survival was 94% (four deaths) and actuarial survival at 3 years was 84 ± 7% (three late deaths).
The majority of patients (94.5%) were in NYHA class I at final follow-up (Fig. 2).

3.1. Mortality

There were 4 (6.5%) early and 3 (4.8%) late deaths of which only one death was shown to be conduit-related.

Early deaths: case 1 was a 3-week neonate with PA/VSD/MAPCAs who underwent repair with a 12 mm conduit and arrested suddenly on day 5 with profound hypoxia and was found to have a completely thrombosed conduit at post-mortem. Case 2 with PA/VSD/MAPCAs was 17 months old who died on post-operative day 7 from necrotising enterocolitis, gut perforation and sepsis. Case 3 was a 5.3 kg patient with Fallot’s tetralogy, disconnected LPA and acquired pulmonary atresia that arrested on day 1 with low cardiac output. Case 4 was a patient with ccTGA, aortic atresia and severe ebsteinoid tricuspid valve who underwent Damus—Kaye/Senning/VSD closure with an RV-PA conduit of size 12 mm who died on day 1 from low cardiac output and RV failure. The Contegra® conduits in these three patients were patent with no thrombus.

Late deaths: the first patient was 6 months old with right atrial isomerism who died from overwhelming pneumococcal sepsis at 2 months post-operative (despite penicillin prophylaxis). The second patient was 2 months old following neonatal biventricular repair of aortic atresia/VSD who also had tracheo-oesophageal fistula repair and died suddenly on the paediatric ward; ECG and echocardiography had been satisfactory and post-mortem was declined. The third patient was 10 years old who had received a redo conduit replacement for PA/VSD and 22 months earlier and had poor right ventricular function and normal conduit performance. She suffered a sudden arrhythmic death.

3.2. Freedom from reintervention

Overall freedom from reintervention (both catheter and surgical) was 53 ± 11% at 38 months. Reinterventions could be divided into those that were conduit-related and those that were related to distal branch pulmonary artery stenoses (exclusively in the PA/VSD/MAPCA group). There were 10 catheter reinterventions for distal (branch) pulmonary artery stenoses with no surgical reinterventions.

3.3. Conduit-specific freedom from reintervention

Conduit-specific freedom from reintervention was 66 ± 11% at 38 months. Six of these were percutaneous catheter interventions for acquired conduit stenosis and four were surgical replacements—one for (early) endocarditis and three for a combination of aneurismal dilatation with distal conduit stenosis.

Univariate analysis identified RV/LV pressure ratio at the end of the operation to be the strongest single factor associated with the need for reintervention (p = 0.0003).

Smaller conduit size (12 and 14 mm, p = 0.04), young age (<1 year, p = 0.04) and severe conduit regurgitation (regurgitant fraction > 40%, p = 0.016) were the only other significant factors. The only diagnostic group that had a significantly higher reintervention rate was the PA/VSD/MAPCA group which was mainly related to distal branch PA stenosis (p < 0.002, Fig. 3c).

On multivariate analysis, the RV/LV pressure at the end of the operation was the only significant factor for reintervention (ratio > 0.6 gave OR 5.45, CI 2.0–14.6, p = 0.001, Fig. 3b). When considering the conduit-specific reinterventions alone, the RV/LV pressure ratio ceased to be significant although the absolute PA pressure continued to be a risk factor for reintervention (PA pressure in 4th quartile, 38–88 mmHg, p = 0.016).

3.4. Conduit stenoses

All stenosis requiring reintervention was an acquired stenosis in the distal conduit involving the site of the distal anastomosis to the branch pulmonary arteries. In addition, there were five conduits (8.6%) that developed mild to moderate valvular stenosis during the study. None required reintervention and none progressed during the period of the study.

3.5. Conduit dilatation and regurgitation

A total of 16 conduits (27.5%) developed significant dilatation during the period of the study (defined by DD% > 30%). All of these conduits developed severe regurgitation which appeared to be a secondary phenomenon to the dilatation as the valve cusps themselves were intact and there had not been any regurgitation prior to the dilatation. A high RV/LV pressure ratio was associated with the dilatation in 11 patients, 10 of whom required subsequent reintervention. Three of these patients had such severe dilatation (DD% > 60%) that it was regarded as the primary indication for urgent conduit replacement. All were associated with high RV pressures and stenosis in the distal conduit.

The remaining five conduits that developed dilatation in the absence of high RV pressures have not required any reintervention to date, but remain under close review. The relationship between conduit dilatation and time is shown in Fig. 4, showing no significant correlation. Dilatation appeared to be influenced by RV/LV pressure rather than by any other factor.
3.6. Conduit thrombosis

There was one fatal incidence of complete conduit thrombosis which occurred in the early post-operative period. In addition, there were five conduits in which thrombus developed in one of the valve cusps, occurring at a median time interval of 78 days (range 12—210 days) after conduit insertion. All cases were receiving aspirin at 2.5 mg kg$^{-1}$ day$^{-1}$, and all were associated with moderate degree of regurgitation. All cases resolved with continuation of aspirin therapy.

3.7. Conduit endocarditis

There was one episode of endocarditis occurring just 2 weeks after implantation. This required urgent conduit replacement (with a second Contegra™ conduit) and went on to make an uneventful recovery. The organism was a Staphylococcus aureus that was isolated on blood culture and subsequently from the conduit with no clearly apparent source.

3.8. Summary

Smaller conduit size (12 and 14 mm), young age at operation (<1 year), conduit dilatation/regurgitation and high RV/LV pressure ratio at the end of the procedure (>0.6) were the most significant factors in predicting the need for reintervention. The latter two are probably interrelated.

3.9. Histopathology of the explanted conduits

The three conduits explanted for aneurismatic dilatation showed similar histopathological findings. There was a generalised layer of thrombus covering the neo-intima, degeneration of elastic fibers within the conduit itself, with additional infiltration with inflammatory and giant cells and diffuse but mild calcification. The patient whose conduit is illustrated in Fig. 5 had PA/VSD/MAPCAs and had undergone single stage repair at 8 months of age (14 mm conduit). RV/LV pressure ratio at the end of the operation was 60%. Elective cardiac catheterisation with balloon angioplasty of distal branch PA stenosis was undertaken 3 months later but there was progressive dilatation of the conduit (diameter difference 93%), and RV/LV pressure ratio increased to 80% leading
to explantation at 9 months. Superficial external examination of the conduit was relatively unremarkable, with apparent mild intimal thickening superior to one valve cusp and mild thickening of vein conduit wall. There was no obvious thrombosis seen but microscopic examination showed diffuse conduit degeneration with multifocal elastic fiber fragmentation, calcification and dropout. There was also evidence of chronic neointimal and adventitial inflammation, with necrosis, luminal surface fibrin deposition and thickening of both neointima and adventitia. The valve cusps were covered by fibrin-rich thrombus layer (of varying thickness) associated with chronic inflammatory cells. At the distal anastamotic suture line, there was focal elastic fiber calcification.

4. Discussion

Bovine jugular vein grafts were first evaluated in 1993 by Sung et al. [6]; these grafts have a retained native valve which is cross-linked with a diglycidyl ether (DE) during preparation. Short-term results showed minimal transvalvar pressure gradients and superior haemodynamic functional performance to other available conduits. In 1994, heparin binding was added in order to permit endothelialisation of the graft in a low pressure system at a year from implantation [7]. Subsequently, Herijgers et al. [10] compared bovine jugular venous conduits with Dacron conduits for RVOT replacement in young sheep at 20 weeks post-implantation; the bovine group showed good function, preserved structure and minimal calcification, whereas the Dacron conduits exhibited extensive fibrous sheathing and calcification. Since its introduction, the conduit has become increasingly popular and versatile, including the use a percutaneously deliverable system mounted on a stent [8,9].

The current commercially available bovine graft, Contegra®, is available for clinical use in diameters between 12 and 22 mm (supported and unsupported versions). Overall length is 10–12 cm but the 12 mm graft has a length of around 7 cm and a conduit valve closer to the outflow. The grafts have been treated with buffered glutaraldehyde without either additional anticalcification or antithrombogenic preparation.
Initially published clinical data in humans have been encouraging [11—14]. Breymann et al. [12] compared the performance of 71 Contegra conduits with 52 homografts and 30 porcine xenografts. Their Contegra group showed no signs of conduit or valve degeneration over 27 months. There were five reoperations for peripheral branch pulmonary stenosis although no histopathology data were available. Breymann and co-workers [18,19] have recently updated their results, with a total of 108 Contegra conduits (the largest available single institute experience) extending the mean follow-up to 4 years, and still maintaining excellent results (see Table 2) although they did report 10 cases of supravalvar stenosis (9.3%). This is similar to the incidence of acquired conduit stenosis at the distal suture line reported in this series (11.6%).

Complex pulmonary atresia has not featured in previously published series, which may make comparison difficult with our current group in which 29% of cases had PA/VSD/MAPCA. This subgroup contributed to 12 (57%) out of a total of 21 reinterventions, making this diagnostic group a significant risk factor for reintervention (p = 0.002). The relatively poor performance of the Contegra conduits in this group may be related to the tendency for these patients to have a high RV/LV pressure ratio post-operatively. Nevertheless, our reintervention for conduit-related stenosis is similar to that reported by Breymann et al. [12] (11.6%).

Significant conduit dilatation was not originally reported as a concern, other than one case by Carrel et al. [14] in a series of 22 conduits and one case from Bovéjiméline et al. [20]. More recently, conduit dilation and thrombosis have featured more prominently [21,22] despite rather short follow-up. Subsequently, Meyns et al. [15] drew the attention to the neointimal proliferation at the distal anastomosis of the conduit, leading to severe stenosis, dilatation of the proximal conduits and a freedom from stenosis of only 49 ± 8% at 24 months.

We have specifically examined performance of the 12 and 14 mm conduits which are particularly valuable due to the scarcity of small homografts. Thirty-eight percent of the conduits were 12 mm and 60% were either 12 or 14 mm in diameter.

Conduit narrowing at the pulmonary anastomosis (distal suture line) was relatively common in this series, and predominantly occurring in the smaller sized conduits (12 and 14 mm). Similar findings have been reported by Meyns et al. [15], together with the development of conduit dilatation. This acquired stenosis in the distal part of the conduit may be partly related to discrepancies in circumference between the smaller conduits and the native pulmonary arteries, as it did not seem to be operator dependent, nor related to the underlying anatomy or the RV/LV pressure ratio at the end of the surgery. The histopathological findings of elastic fiber proliferation and calcification at this point, on top of the neointimal proliferation and thrombus layer covering the entire conduit, suggest an active process occurring within the neointima which may accelerate the development of stenosis and create resistance to balloon angioplasty.

Almost one third (27.5%) of the conduits developed a degree of dilatation over time in this series. In the majority of cases, including all those that became clinically significant, the dilatation appeared to be secondary to branch pulmonary artery stenosis, reflecting the much larger proportion of patients with PA/VSD/MAPCAs. Although dilatation is significantly related to high RV and intra-conduit pressure rather than to time, there is clear histological evidence of a chronic inflammatory process in the three explanted aneurysmal conduits.

However, a small number of conduits (8.5%) demonstrated an unpredictable dilatation in the absence of raised intra-conduit pressure. None of these patients have required reintervention but there is regurgitation across the conduit as a result and these patients will need to be carefully followed up. A variable degree of inflammatory response may explain why these conduits dilated, and it will be valuable to examine the histopathology of this low RV pressure group when these conduits come up for replacement. The

Table 2

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<td>41</td>
<td>22</td>
<td>28</td>
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<td>with PA/MAPCA</td>
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<td>Mean follow-up (months)</td>
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<td>25</td>
<td>12</td>
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<td>40 (37%)</td>
<td>Median = 16 mm</td>
<td>11 (50%)</td>
<td>11 (61%)</td>
<td>15 (52%)</td>
<td>17 (29%)</td>
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<td>0</td>
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<td>91.66 ± 2.6</td>
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<td>100</td>
<td>84</td>
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<td>Conduit stenosis</td>
<td>0 (0%)</td>
<td>8 (7.4%)</td>
<td>6 (14.6%)</td>
<td>1 (4.5%)</td>
<td>3 (11%)</td>
<td>8 (27.6%)</td>
<td>50%</td>
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<td>Aspirin</td>
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E: endocarditis, D: dilatation, T: thrombosis, R: regurgitation, NI: not indicated, C: conduit, Ix: intervention. The grading mild to severe in the complication box refers to the severity of the leak. *Ten had PA/VSD but not MAPCA.

* Aspirin now recommended by authors for all Contegra patients.
Contegra conduit(s) in this series have been subjected to higher pressures than in most previous reports and there is no doubt that the dilatation appears to be a secondary phenomenon and not primary conduit failure. Nevertheless, this degree of dilatation is not seen in homografts or prosthetic conduits and we would recommend that these should be used in the setting where high RV pressures are predicted post-operatively.

Echocardiographic evidence of conduit thrombosis was an unexpected finding in this study, although it has been subsequently reported [21,22]. The valve cusps of the jugular vein graft are deeper than normal semilunar valve cusps, which might predispose to thrombosis in situ. Also, the histopathological study of the three explanted (dilated) conduits showed a complete layer of thrombus covering the neointima of the valve cusps and the conduit itself, which was not obvious macroscopically. Biological implants cross-linked with glutaraldehyde are potentially thrombogenic [23]. The residual glutaraldehyde released from the implant long after insertion hinders host cells from colonising the luminal layer exposing the implant to thrombogenic process [24]. Aspirin therapy is well known to reduce the risk of thrombosis in vein grafts [25] and Boudjemline et al. [21] are now prescribing an anti-aggregate therapy in all patients for at least 6–12 months post-implantation.

In this study, the non-fatal valve thromboses occurred in patients already receiving aspirin (the mean dose used was 2.5 mg kg⁻¹ day⁻¹). In all cases the thrombus had disappeared on follow-up. Although aspirin’s influence on the outcome of the conduit was not statistically significant, concerns about thrombogenicity appear genuine from clinical point of view and in combination with histological evidence of an inflammatory process. Serious consideration should be given to the evaluation of the use of more effective anti-inflammatorv/antithrombotic medication after implantation of these grafts.

The small homograft is still considered by most as the ‘gold standard’ for neonatal and infant reconstructions. However, concern was about the long-term performance (which as yet, we cannot provide for Contegra grafts) and availability [3–5] which led to the original development of the Contegra. The main advantages of the Contegra conduit is the off-the-shelf availability in a wide size range, with more versatile handling properties compared to prosthetic conduits. There is little evidence to support that even the small Contegra conduits do not perform as well as small homografts [12,18], although the tendency for the Contegra conduit to dilate (albeit in the setting of high pressure) and the incidence of valve thrombosis is not a feature of the homograft.

4.1. Limitations of the study

Follow-up is still too short to make conclusive evaluation of conduit performance or clear statements regarding durability. There is no direct control group in this study and it will be particularly valuable to evaluate these conduits against small homografts in the future, ideally as a randomised trial. The complex pulmonary atresia/MAPCAs group are a major confounding factor as to the need for non-conduit-related interventions and it is difficult to separate conduit performance from inherent problems with the branch pulmonary arteries. Although analysis may have been more straightforward by excluding these patients, it is important to evaluate Contegra performance in this difficult subset of patients.

5. Conclusion

The Contegra valved conduit is an attractive option for right ventricular outflow tract reconstruction particularly in smaller patients. Excellent tissue handling, versatility, haemostasis and off-the-shelf availability are the principal advantages. However, there is a significant incidence of conduit-related complications, the commonest of which is the development of stenosis at the distal suture line during follow-up of the smaller-sized conduits (12 and 14 mm).

High pressure in the conduit may lead to aneurysmal dilatation and resultant valvar regurgitation. However, there is also a small unpredictable risk of conduit dilatation that is unrelated to pressure and also of thrombosis in the valve sinuses, neither of which appear to be clinically important but remain a cause for concern and will need further evaluation.

The findings of this study suggest that these conduits perform poorly under higher pressure conditions (particularly with PA/VSD/MAPCAs repair). We would recommend that the conduits are used with caution in patients in whom a high RV pressure is anticipated post-operatively. The advantage of the Contegra conduit over a homograft in smaller patients (<1 year) also remains uncertain. The role of anti-inflammatory and antithrombotic therapy needs to be evaluated. At present we would recommend indefinite aspirin therapy.

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