Comparison of bovine jugular vein with pulmonary homograft conduits in children less than 2 years of age

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Abstract

Objectives: The optimal pulmonary valved conduit for infants and small children remains controversial. This report compares the initial insertion outcome of small caliber bovine jugular vein (BJV) (12–14 mm) with pulmonary homografts (PHs) (10–15 mm) in patients under age 2.

Methods: From December 1998 to August 2009, 84 children (mean age 8.4 ± 8.5 months) received BJV (n = 51) or PH (n = 32) conduits. Mean Z score for BJV was 2.2 (range: −0.8 to 3.3) and for PH 2.1 (range: 0.8–4.2; P = 0.2). The two cohorts were similar with respect to age, BSA, conduit indication, bypass and cross-clamp time. Graft dysfunction is defined as right ventricular outflow tract obstruction with peak echo-Doppler gradient >40 mmHg and/or grade III/IV conduit valve regurgitation. Graft failure is defined as need for conduit replacement or need for catheter or surgical re-intervention. Follow-up was greater in number in homografts (BJV, 90% at 85% vs PH, 71% and 24% P = 0.05). Results: Early and late mortality were similar (BJV, 80%; PH 88%; P = 0.55). No death was graft related. Freedom from dysfunction was improved at 5 and 10 years with BJV (BJV, 90% at 85% vs PH, 71% and 24% P < 0.05). Conduit failure trended higher in the PH cohort at 5 and 10 years (BJV, 85% and 67% vs PH, 75% and 45%; P = 0.06). Freedom from explantation was significantly better for BJV patients (BJV, 85% vs PH, 47% P < 0.001). Freedom from distal conduit stenosis was similar (BJV, 52% vs PH, 44% P = 0.36). Conclusions: This study suggests that the early performance of small BJV may be more advantageous than homografts. A BJV conduit is an appropriate first choice for conduit replacement in patients less than 2 years of age.

Keywords: Pulmonary valve replacement; Conduits; Pulmonary homograft; Outcomes

1. Introduction

The optimal valved conduit to establish right ventricle to pulmonary artery continuity for certain cono truncus congenital malformations involving hypoplasia or atresia of the right ventricular outflow tract (RVOT) has not been established. Pulmonary allograft, the long-standing prosthesis of choice, has been plagued by limited availability and premature obstruction secondary to calcific stenosis, notably in sizes appropriate for neonates and infants [1]. This observation has prompted the quest for an alternative biological valved conduit. The bovine jugular vein conduit (BJV, C, Contegra, Medtronic, Inc., Minneapolis, Minnesota, USA), first introduced into clinical practice in 1999, is a heterogenous graft containing a trileaflet venous valve with three natural sinuses slightly larger than the lumen. This conduit is stored in buffered glutaraldehyde without anti-calcification treatment and is available in sizes as small as 12 mm.

In contrast to homografts, BJV offers five important technical advantages: (1) off-the-shelf availability in small sizes; (2) the ability to easily displace the conduit valve leftward and posterior by shortening the outflow end to avoid potential distortion from the ascending aorta or compression by the sternum; (3) completion of the right ventricular anastomosis exclusively with the inflow end of the conduit, avoiding a hood of additional prosthetic material; (4) glutaraldehyde preservation, which likely renders the prosthesis non-antigenic and (5) a larger effective orifice area secondary to the absence of stent and the structure of the valve leaflets as they insert directly on the wall of the jugular vein.

Recent clinical reports, albeit with follow-up of less than 2 years, have demonstrated clinically equivalent performance of BJV and pulmonary homografts (PHs) for neonatal repair of truncus arteriosus and in children receiving BJV within the first 4 years of life [2,3].

The purpose of this report is to compare BJV and PH conduits with respect to patient survival, conduit durability...
and performance in all neonates and infants less than 2 years of age requiring first-time RVOT reconstruction with a follow-up exceeding 4 years.

2. Materials and methods

The medical records of all patients less than or equal to 2 years of age undergoing RVOT reconstruction with the first-time insertion of a PH or Contegra conduit from December 1998 to August 2009 at James Whitcomb Riley Children’s Hospital at Indiana University, Indianapolis, Indiana, USA and Cardinal Glennon Children’s Hospital at St. Louis University, St. Louis, Missouri, USA were retrospectively reviewed. The Institutional Review Board (IRB) at St. Louis University and Indiana University approved this study and both IRBs waived the need for patient consent.

Fifty-two paediatric patients, who had BJV implanted, were compared with 32 patients receiving PH. The choice of conduit was made preoperatively or in the operating room by the surgeon without randomisation.

All homografts were obtained from Cryolife (Marietta, GA, USA). No patient received a prosthesis that was undersized to accommodate a larger prosthesis and to avoid conduit stringing. When applicable, the incision in the pulmonary artery was extended leftward onto the left pulmonary artery to position the conduit valve at the distal anastomosis well to accommodate a larger prosthesis and to avoid conduit stringing. When applicable, the incision in the pulmonary artery bifurcation was performed with continuous suturing. If the distal anastomosis to the pulmonary artery bifurcation was performed with continuous suturing, the proximal anastomosis was then performed with the cross-clamp removed and the heart beating during rewarming. All BJV conduits were continuously agitated for 5 min in four, 500 ml normal saline baths to remove glutaraldehyde. The distal anastomosis to the pulmonary artery bifurcation was performed with continuous Prolene suture tied at two locations to prevent purse stringing. When applicable, the incision in the pulmonary artery was extended leftward onto the left pulmonary artery to accommodate a larger prosthesis and to avoid conduit distortion by the ascending aorta and the sternum [3]. The outflow end of the BJ conduit was cut as short as possible to position the conduit valve at the distal anastomosis well to the left of the sternum. The inflow end of the prosthesis was opened posteriorly and sewn to a vertical incision in the right

2.1. Definitions

Graft dysfunction is considered to be present if the peak echo Doppler gradient is greater than 40 mmHg by two-dimensional echocardiography at any level within the RVOT and/or grade 2 or 3 conduit valve insufficiency.

Conduit failure is defined as the need for surgical repair, replacement and/or intervention with balloon dilatation or stent placement. Excluded are catheter interventions for peripheral pulmonary artery stenosis not involving the distal conduit anastomosis.

Non-mutually exclusive indications for conduit replacement include: (1) graft dysfunction; (2) right ventricular pressure greater than or equal to two-thirds of systemic pressure; and (3) unsuccessful percutaneous intervention.

Fig. 1 shows the distribution and frequency of conduit insertion. Homografts were more frequently inserted within the first 3 years of our study; thereafter, Contegra was more commonly used to establish right ventricle to pulmonary artery continuity.

The patient demographics are summarised in Table 1. There were no significant differences in age, sex, body surface area or preoperative diagnoses. Truncus arteriosus and tetralogy of Fallot with pulmonary atresia were the most common indications for conduit insertion, with the BJ conduit more frequently employed in the latter morphology. DiGeorge syndrome was the most frequent non-cardiac anomaly, while the majority of prior operations were interim, modified, Blalock shunts.

2.2. Operative technique

All patients underwent median sternotomy, bicaval cardiopulmonary bypass, left ventricular venting and moderate hypothermia (28–32 °C). Intracardiac repair was performed during aortic cross-clamping with intermittent cold blood cardioplegia, while conduit insertion was performed with the cross-clamp removed and the heart beating during rewarming. All BJV conduits were continuously agitated for 5 min in four, 500 ml normal saline baths to remove glutaraldehyde. The distal anastomosis to the pulmonary artery bifurcation was performed with continuous Prolene suture tied at two locations to prevent purse stringing. When applicable, the incision in the pulmonary artery was extended leftward onto the left pulmonary artery to accommodate a larger prosthesis and to avoid conduit distortion by the ascending aorta and the sternum [3]. The outflow end of the BJ conduit was cut as short as possible to position the conduit valve at the distal anastomosis well to the left of the sternum. The inflow end of the prosthesis was opened posteriorly and sewn to a vertical incision in the right

![Fig. 1](https://academic.oup.com/ejcts/article-abstract/38/3/318/431346/fig)
ventricle with continuous Prolene suture and no additional prosthetic material.

Homografts were implanted similarly except that the prosthetic valve was positioned at the distal end of the right ventriculotomy, and the proximal anastomosis was augmented anteriorly using a hood of autologous pericardium or Gore-Tex.

Stenosis in a branch pulmonary artery was relieved employing a patch of bovine or autologous pericardium. To facilitate sternal re-entry, Gore-Tex membrane pericardial substitute (W.L. Gore & Company, Flagstaff, AZ, USA) was sewn to the native pericardium in all patients at the time of sternal closure to cover the conduit and all anterior cardiac structures.

Postoperatively, patients in the BJV cohort received heparin, which was transitioned to aspirin (10 mg kg\(^{-1}\) day\(^{-1}\)) at discharge. Patients receiving homografts were given ibuprofen postoperatively and transitioned to lifetime aspirin (10 mg kg\(^{-1}\) day\(^{-1}\)) at discharge.

2.3. Operative variables

The operative variables are summarised in Table 2. Concomitant procedures were nearly equally distributed between groups and consisted of closure of atrial and ventricular septal defects, repair of tricuspid or mitral valves, patch enlargement or reduction of the pulmonary arteries and, in one patient, repair of cardiac total anomalous pulmonary venous return.

The time on the extracorporeal circuit and the global ischaemic times were similar. The mean conduit size was 13 mm for both cohorts, but a significantly greater number of patients received PHs in sizes 10 and 11 mm, while BJV patients were more likely to receive a 12-mm conduit (\(P < 0.05\)).

The mean conduit Z score at first implantation was two standard deviations above normal and was not significantly different between cohorts.

2.4. Follow-up

All surviving patients were examined by their referring cardiologist in the immediate postoperative period and re-examined with serial transthoracic echocardiography every 6 months or 1 year until August 2009.

Follow-up was 100% in both cohorts. For PH, the mean follow-up was 5.9 ± 3.3 years, ranging from 3 months to 10 years, with a median of 7 years. The mean follow-up for BJV was significantly lower at 4.4 ± 3 years, ranging from 2 months to 10 years, with a median of 4 years.
2.5. Statistical analysis

Data are presented as the mean ± SD. Continuous variables were assessed using Student’s t-test and categorical variables using the chi-square test. Variables for the two cohorts were compared using two-tailed unpaired t-test. The log-rank test was used to estimate the statistical significance of differences between survival curves for actuarial survival, freedom from conduit dysfunction or failure, interventional or surgical re-intervention and conduit replacement, respectively. The chi-square test was used to compare using two-tailed unpaired t-test. Kaplan–Meier curves for actuarial survival, freedom from conduit dysfunction or failure, interventional or surgical re-intervention and conduit replacement, respectively. The log-rank test was used to estimate the statistical significance of differences between the two types of conduits. The significance level was set at a P value of ≤ 0.05.

3. Results

3.1. Early mortality

There were three early deaths, each in the BJV cohort (BJV, 3/52 6% vs PH, 0/32 0%; P = 0.28). A 1-month-old neonate with truncus arteriosus type 2, single coronary artery and major aortopulmonary collateral arteries underwent biventricular repair employing a 12-mm BJV for RVOT reconstruction. This patient expired 2.5 weeks postoperatively secondary to sepsis and multi-organ system failure. Autopsy demonstrated disseminated cytomegalovirus infection and pulmonary candidiasis. The second mortality occurred in a 2-week-old neonate with truncus arteriosus type 3 and interrupted aortic arch type A, who underwent truncus arteriosus and interrupted aortic arch repair with insertion of a 12-mm BJV. The patient expired on the third postoperative day secondary to massive pulmonary hemorrhage with disseminated intravascular coagulation (DIC), while on extracorporeal membrane oxygenation for acute right ventricular failure secondary to pulmonary hypertension. The final mortality occurred in a 6-month-old patient with double outlet right ventricle and a remote ventricular septal defect (VSD) who underwent Rastelli repair with implantation of a 12-mm BJV. The patient died 1 week postoperatively from low cardiac output and multi-organ system failure.

3.2. Late mortality

There were seven BJV and four PH late deaths (BJV, 7/52 13% vs PH, 4/32 12%; P = 0.73). All but one mortality occurred in the first postoperative year and is summarised in Table 3. No death was attributed to structural failure of the conduit. The actuarial survival including hospital deaths for BJV and PHs at 5 (BJV, 80%; PH, 88%) and 10 years (BJV, 80%; PH, 88%) was similar for both cohorts (P = 0.35) (Fig. 2).

3.3. Morbidity

Operative morbidity was not significantly different between cohorts. Post bypass extracorporeal membrane oxygenation was required in six patients (BJV, three patients; PH, three patients) because of low cardiac output or right ventricular dysfunction secondary to pulmonary hypertension. Twelve patients (BJV, seven patients; PH, five patients) required delayed sternal closure and one patient (PH) required left diaphragm plication for phrenic nerve paresis. No patient in either cohort developed a conduit aneurysm or endocarditis.

3.4. Freedom from conduit dysfunction

Among the 81 hospital survivors, 30 patients (BJV, 8/49 16%; PH, 22/32 69%; P = 0.001) had evidence for significant conduit dysfunction at the most recent follow-up or before conduit replacement. The mode of dysfunction was characterised as stenosis with regurgitation, 17 patients; stenosis or insufficiency alone in nine and four patients, respectively. Freedom from conduit dysfunction was significantly worse in the homograft cohort (71% and 24% at 5 and 10 years, respectively) as compared with BJV (90% and 85% at 5 and 10 years, respectively, P < 0.001) (Fig. 3(A)).

Table 3

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (days)</th>
<th>Time (days)</th>
<th>Diagnosis</th>
<th>Conduit/procedures</th>
<th>Cause of death</th>
</tr>
</thead>
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<tr>
<td>1</td>
<td>25</td>
<td>90</td>
<td>TA, Cornelia de Lange’s syndrome</td>
<td>11 mm PH</td>
<td>Unknown</td>
</tr>
<tr>
<td>2</td>
<td>19</td>
<td>648</td>
<td>TA</td>
<td>11 mm PH</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>3</td>
<td>21</td>
<td>62</td>
<td>TA, mild truncal valve insufficiency, horseshoe kidney</td>
<td>12 mm PH</td>
<td>LCO</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>86</td>
<td>TOF, APV, LSVC, absent corpus colliosus, left lung hypoplasia, ventilator dependence</td>
<td>12 mm PH</td>
<td>LCO</td>
</tr>
<tr>
<td>5</td>
<td>20</td>
<td>72</td>
<td>TA, discontinuous PAs, MAPCAs, DiGeorge</td>
<td>12 mm BJV</td>
<td>Bronchopneumonia</td>
</tr>
<tr>
<td>6</td>
<td>28</td>
<td>181</td>
<td>TA</td>
<td>12 mm BJV</td>
<td>Sepsis, meningitis</td>
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<tr>
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<td>185</td>
<td>192</td>
<td>PA/VSD/MAPCAs, s/p BTS, left MAPCAs ligation</td>
<td>12 mm BJV</td>
<td>Sepsis</td>
</tr>
<tr>
<td>8</td>
<td>242</td>
<td>110</td>
<td>PA/VSD/LSVC, s/p BTS</td>
<td>12 mm BJV</td>
<td>Multiple system failure</td>
</tr>
<tr>
<td>9</td>
<td>24</td>
<td>64</td>
<td>TA, moderate truncal valve insufficiency, IAA (type A)</td>
<td>12 mm BJV</td>
<td>Multiple system failure</td>
</tr>
<tr>
<td>10</td>
<td>176</td>
<td>40</td>
<td>DORV, remote VSD, s/p PAB</td>
<td>12 mm BJV</td>
<td>LCO, intracranial haemorrhage</td>
</tr>
<tr>
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<td>2499</td>
<td>104</td>
<td>PA/VSD/MAPCAs, S/P right and left unifocalisation, s/p BTS</td>
<td>12 mm BJV</td>
<td>Multiple system failure</td>
</tr>
</tbody>
</table>

TA, truncus arteriosus; DORV, double outlet right ventricle; VSD, ventricular septal defect; MAPCAs, major aortopulmonary collateral arteries; BTS, modified Blalock–Taussig shunt; PA, pulmonary atresia; PAB, pulmonary artery banding; LSVC, left superior vena cava; PAs, pulmonary arteries; APV, absent of pulmonary valves; LCO, low cardiac output; PH, pulmonary homograft; BJV, bovine jugular vein.
3.5. Freedom from conduit failure

3.5.1. Pulmonary homograft

At latest follow-up, 19 patients (59%) in the PH cohort exhibited conduit failure (15 patients, explantation; two patients, percutaneous intervention; and two patients for both) secondary to stenosis and/or valve dysfunction. The indications for re-operation included a mean RV-PA gradient of $51 \pm 22$ mmHg (range: 20—90 mmHg), distal supravalvar or branch pulmonary artery stenosis greater than 40 mmHg and/or grade 2 or 3 pulmonary insufficiency. Five patients required a re-operation during the first 3 postoperative years, while 14 patients required re-operation 2—9 years postoperatively. The mean time to re-operation in the homograft cohort was $4.5 \pm 2.3$ years (range: 8 months to 9 years). Percutaneous interventions were required in four patients (balloon dilatation, two patients and stent implantation, two patients); of these, two patients required conduit explantation at 1—2 years post intervention. The BJV conduit was used to replace all explanted homografts.

3.5.2. Bovine jugular vein

Conduit failure was observed in 16 of 52 BJV patients (31%) (percutaneous, eight patients; surgical, five patients; and both, three patients). The mean RV—PA gradient was $48 \pm 18$ mmHg (range: 20—80 mmHg). Surgical procedures included seven explants and one distal anastomotic patch angioplasty. The majority of percutaneous interventions (balloon dilatation, eight patients and stent implant, three patients) occurred in the first 2 postoperative years. The mean time to re-operate for BJV was $4.4 \pm 3.4$ years (range: 6 months—9 years) and was not significantly different from the PH cohort ($P = 0.68$). All explanted BJVs were replaced with a BJV prosthesis.

At 5 and 10 years post-implantation, the freedom from conduit failure trended higher in the BJV cohort, but the difference was not significant ($P = 0.06$; Fig. 3(B)). As shown in Fig. 4, the fate of BJV was significantly better than PH with a freedom of explantation at 5 and 10 years of 95% and 85%, respectively ($P < 0.0001$).

The frequency of supravalvar and/or proximal branch pulmonary stenosis was similar (BJV, 21/52; 40% vs PH, 16/32; 50%; $P = 0.26$). The actuarial freedom from distal stenosis at 10 years was not significantly different between cohorts (BJV, 56% vs PH, 44%; $P = 0.36$).

3.5.3. Conduit retention and function at last follow-up

Among the 70 late survivors (BJV, 42/52, 81% vs PH, 28/32 82%, $P = 0.55$), 47 patients retained their initial conduit (BJV, 35/52 67% vs PH, 12/32 37%; $P = 0.001$).

In the BJV group, PR was trivial in 15 patients (43%), mild in 13 (26%) and moderate in seven patients (14%). One patient with moderate regurgitation had a distal anastomotic peak gradient of 55 mmHg, while four patients had transconduit gradients above 30 mmHg (35, 40, 40 and 64 mmHg).

Conduit regurgitation among the 12 PH patients was trivial in four, mild in three and moderate in five patients. At latest follow-up, four patients (33%) in the PH group were being
followed up with a transconduit gradient above 40 mmHg (43, 50, 55 and 55 mmHg).

We examined the freedom from conduit dysfunction combined with explanation at latest follow-up. Among the 52 original BJV conduits, eight were dysfunctional and seven explanted, while in the 32 PH patients, four were dysfunctional and 17 explanted. Freedom from dysfunction combined with explanation was 71% (37/52 patients) in the BJV cohort, but only 34% (11/32 patients) in the PH cohort (P < 0.01).

We also compared the performance of size 14 mm conduits inserted primarily in patients less than 1 year of age (BJV, 24 patients; PH, 10 patients). Survival was 100% in both cohorts. Explanation (BJV 4/24; 17%; vs PH 4/10; 40%; P = 0.19) and failure (BJV 6/24; 25% vs PH 5/10; 50%; P = 0.23) trended lower in the BJV cohort, but did not reach statistical significance. Conduit dysfunction was significantly higher in the PH group (BJV 6/24; 25%; vs PH 7/10; 70%; P = 0.02).

4. Discussion

This study is a retrospective, non-randomised, bi-institutional comparison of BJV and PHs for RVOT reconstruction with respect to durability and conduit function as evaluated by echocardiography in neonates, infants and children less than 2 years of age. We have demonstrated that, at intermediate follow-up, actuarial survival is similar (88% at 10 years), but conduit dysfunction and the need for graft explantation are higher among patients receiving pulmonary homografts. Our results also suggest that conduit durability as defined by the freedom from surgical or percutaneous intervention trended higher in those patients receiving the Contegra prosthesis.

In this report, the cohorts were quite comparable with respect to age, frequency of neonatal operations and implant conduit Z score. The number of homograft implants is less, in part, because of the limited availability in small sizes (Fig. 1).

Age less than 2 years is the most challenging environment to test any RVOT conduit. At this time, neonates and infants experience rapid growth and weight gain, which can lead to early somatic outgrowth of the prosthesis.

Another concern is the immunologic reaction known to occur with valve allografts implanted in patients at this age. Cryopreservation does not eliminate the antigenic expression in homografts [4]. A vigorous immune response as evidenced by elevated human leucocyte antigen (HLA) antibodies to viable endothelial cells and fibroblasts on the surface of the cryopreserved conduit has an impact significantly on allograft dysfunction and failure [5]. In addition to the humoral response, Hoekstra and associates have demonstrated a cell-mediated immune reaction to cryopreserved homografts. T cells have been directly implicated in the inflammatory process that is associated with early and late allograft valve failure [6,7]. An important advantage of Contegra is storage in dilute, buffered glutaraldehyde, which retains leaflet- and conduit-wall compliance. Based on the long-established experience with xenograft valves, glutaraldehyde preservation likely renders Contegra non-antigenic.

Aortic homografts placed in the pulmonary position, perhaps because of decreased elastin, are more prone to early calcification resulting in stenosis and reduced durability. When compared with PHs in children less than 2 years of age, aortic allografts have demonstrated a significant higher degree of valve regurgitation and conduit stenosis [8].

Tweddell and associates in a study analysing risk factors for homograft failure noted that outgrowth was the primary aetiology for explanation, and inserting an allograft with a Z score of +1 to +3 was associated with the greatest freedom from failure [9].

PHs are the current conduit of choice for RVOT reconstruction. Greater elastin content may render PH more compliant than aortic allografts and resistant to early calcification, which leads to stenosis, regurgitation and explantation [10]. Currently, in the United States, the availability of PHs in sizes appropriate for patients less than 2 years of age is severely limited. To accommodate this deficiency, the BJV conduit was introduced and may offer many attractive advantages over pulmonary allografts. Recent reports have demonstrated favourable haemodynamics and excellent durability in small-size Contegra conduits, which are readily available for neonates and infants [11–13].

We believe the size of the implanted prosthesis is critically important. Karamiou and associates have confirmed in PHs the observation made by Tweddell (noted above) for aortic homografts [14]. The ideal implant size is a Z score of +1 to +3. In this report, the mean Z score for the implanted valve was 2.0. Larger conduit insertion, particularly in certain anatomic subtypes (e.g., transposition with pulmonary atresia) predisposes the prosthesis to sternal compression and valve dysfunction. In our series, the index Z value of the implanted conduit was similar in both cohorts and therefore, the observed advantage of the Contegra conduit cannot be due to a discrepancy in implant size.

In addition to its availability in smaller sizes, compliance of Contegra is structurally suitable for neonatal implantation. The venous wall is ideal for suturing and the risk of valve thrombosis is exceedingly rare. However, isolated reports of cusp thrombosis have been documented [15].

Biological conduits cross-linked with glutaraldehyde are potentially thrombogenic and therefore, all of our patients are anticoagulated postoperatively with lifetime aspirin.

Experimental reports suggest that the BJV conduit is resistant to early calcification particularly in smaller sizes [16,17]. Herijgers and associates reported the resistance of Contegra to calcification when implanted in juvenile sheep for 5 months [18]. We reviewed the histology of all explanted BJV conduits in this series and noted that of the seven explants, none had calcium in the valve leaflets or conduit wall. Two patients demonstrated isolated, microscopic calcium deposits within blood vessel walls at the distal anastomotic suture line. Explanted PHs, however, exhibited extensive calcification along the conduit wall and valve leaflets.

In this report, 5- and 10-year freedom from conduit dysfunction observed in pulmonary allografts at a Z score above 2 is 71% and 24%, respectively. This is nearly identical to that reported by Sinzobahlmvya and associates in a series of 76 PHs ranging in size from 8 mm to 13 mm [19]. The 5- and 10-year freedom from dysfunction in the BJV cohort was significantly better at 90% and 85%, respectively. Hickey and
associates, in a study comparing Contegra with PH in patients with truncaus arteriosus, noted a greater increase in peak instantaneous conduit gradient and conduit valve regurgitation in those patients receiving pulmonary allografts [20].

Conduit explantation, perhaps the most stringent measure of performance, was significantly better at 5 and 10 years with BJV conduits. This is in agreement with the recent work of Boethig and associates, who demonstrated a freedom from explantation at 5 years for Contegra at 90% and PHs at 70% \( (P = 0.02) \) [21].

The distal anastomosis is the principal site of BJV conduit obstruction. Several aetiologies have been proposed including inadequate glutaraldehyde removal, kinking from excessive outflow length, distortion by the ascending aorta from rightward implantation on the pulmonary artery and improper suturing technique leading to anastomotic stenosis. We have addressed each of these technical issues (in Section 2). The echocardiographic analysis of distal obstruction must be evaluated cautiously. Differentiation of an anastomotic gradient from proximal pulmonary artery stenosis can be problematic. Central to the mechanism of stenosis is neo-intimal proliferation. Meyns and associates noted excessive neo-intimal ingrowth at the distal anastomosis resulting in the need for endovascular intervention in 17 patients (29%) and conduit explantation in seven (12%) [22]. In our report, the 10-year freedom from distal obstruction at the anastomosis or in the proximal pulmonary artery was not significantly different between Contegra (56%) and PH (44%). Among our seven explanted BJV conduits, distal anastomotic obstruction was observed in five patients (71%).

In addition to distal anastomotic obstruction, two other complications, valve thrombosis and conduit dilatation, have been reported in patients receiving BJV prosthesis [23, 24]. Glutaraldehyde can hinder and delay neo-intimalisation of the conduit tube resulting in early thrombosis. In a canine model, Chang and associates reported no endothelialisation in glutaraldehyde-preserved grafts at 6 months [25]. For this reason, all of our patients are kept on lifetime aspirin.

Aneurysmal dilatation of this prosthesis has been reported in patients with hypoplastic pulmonary arteries or with high pulmonary vascular resistance and right ventricular pressures exceeding 75—80% systemic [26]. The dilatation aetiology is unknown, but this conduit is harvested from the bovine venous system and even following glutaraldehyde fixation may not withstand pulsatile blood flow at or near systemic levels. For this reason, Contegra conduit insertion in this patient population mandates close echocardiographic and clinical long-term follow-up.

5. Conclusions

The structural integrity and conduit performance of the BJV prosthesis is superior to PHs in patients less than 2 years of age, followed up for 5 years, undergoing RVOT reconstruction for the first time. Patient and operative variables were analogous, but follow-up was longer in the PH cohort. Actuarial survival is similar, but freedom from conduit dysfunction, failure and explantation is significantly better with Contegra. In our practice, the BJV conduit is currently the prosthesis of choice to reconstruct the RVOT in neonates, infants and children.

5.1. Limitations

A number of important limitations are present in this analysis. Data were acquired retrospectively from two institutions in a non-randomised manner. The operations were performed by four surgeons and the decision for conduit type was determined by the patient’s anatomy and surgeon preference. The population sizes are small with unequal follow-up, which could increase the risk for a type 2 error. Although methodology and management strategy was standardised between both institutions, it is possible that individual differences among surgeons could influence patient outcome.

Acknowledgement

We gratefully acknowledge the expert technical assistance of Mrs Terri Wriley in manuscript preparation.

References

Appendix A. Conference discussion

Dr P. Bonhoeffer (London, UK): For me it’s a difficult issue to comment on a surgical paper. I find this a very interesting paper because there are a number of issues which have not necessarily come across into the practice of every cardiac surgical unit up to now.

It is obviously annoying that there is no way up to now to make a decision about a simple conduit, which one is the right one, and actually to be able to study that in a conclusive way.

And I think that your paper gives the answer to the question of whether there is an alternative to homograft surgery, which is a valid alternative. And I think that your paper gives the answer to the question of whether your homograft preparation is actually representative of all homografts and how they naturally occurring crosslinking agent (genipin) in a canine model. J Thorac Cardiovasc Surg 2003;23:122–4.


Dr Fiore: In response to your first question, we did not ABO match these homografts, nor are they reduced-size homografts. We don’t take a larger homograft and make it into a size 12 or 14. There are data suggesting that reduced size homografts function equally as well as non-reduced homografts, but in this series we did not employ this technique. We are unable to ABO match homografts to the donor because of size availability in patients under two years of age. And that’s really all I can answer with respect to that. Is there anything else?

Dr Bonhoeffer: No, no, that’s fine.

Dr Fiore: Let me make a comment or two about the technical aspects of implantation. We wash the homografts not in three, but four baths of 500 cc of normal saline. We continuously agitate them rather than leave them simply sitting in the normal saline solution.

We cut the outflow end of the conduit very short so that the pulmonary valve is very close to the pulmonary artery bifurcation. We then rewash the homograft in a fourth saline bath. When we make our incision in the pulmonary artery, we try to keep it leftward. We do not make any incisions in the right pulmonary artery. We think this is important to keep the curvature of the conduit to the left of the ascending aorta and posteriorly so as to avoid sternal compression.

We do use Prolene suture for the proximal and distal anastomoses. Distally, we lock a handful of stitches so as to avoid a purse-string effect. proximally we don’t use any added material. We simply split the Contegra posteriorly and fashion it to cover the incision in the right ventricle. We use continuous Prolene suture for this. In neonates, I frequently a small strip of excess Contegra placed on the epicardial surface at the heel of the ventriculotomy anastomosis for added haemostasis.

That’s all I want to say about the technical details. What was your third question?

Dr Bonhoeffer: My third question was, even I get confused here, is there still the situation where you would use a homograft and prefer it over a Contegra in a small kid?

Dr Fiore: I have to honestly answer that no. In general, we would favour Contegra over homograft even in neonates and infants. Remember that in sizes 9, 10, and 12, pulmonary grafts are difficult to find. At the present time, I believe our data and we prefer Contegra rather than pulmonary homografts.

Dr J. Amato (Chicago, Illinois): I’m just going to give you a minimal contribution. Eight years ago an infant presented with ectopia cordis and double outlet right ventricle. After repair of the ectopia cords by placing the heart within the chest and performing Blalock–Taussig shunt, in time we reoperated the child and placed a Contegra bovine homograft. Six years later we changed that bovine jugular vein to a larger size, no failure but just a larger size. He’s now eight years old and doing excellently, thanks to the bovine jugular vein.