Repair of truncus arteriosus: a considered approach to right ventricular outflow tract reconstruction

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Abstract

Objective: In repair of truncus arteriosus the accepted methods of establishing right ventricle (RV) to pulmonary artery (PA) continuity utilize an allograft or xenograft valved conduit. Alternatively, the PA confluence may be directly anastomosed to the RV with anterior patch augmentation, which may allow growth and delay or avoid subsequent RVOT obstruction. These methods of RVOT reconstruction were evaluated in infants undergoing truncus arteriosus repair. Methods: A retrospective analysis of 61 infants undergoing repair of truncus arteriosus between November 1988 and June 2000 was performed. Median age was 34 days (range 1 day to 6.4 months). The patient cohort was subdivided into two groups (1) Valved conduit group: RV to PA continuity performed with a conduit in 38 patients using allograft (28) or xenograft (10). (2) Direct anastomosis group: direct RV–PA anastomosis performed in 23 patients, augmented anteriorly with monocusp (15) or simple pericardial patch (eight). Results: There were eight hospital deaths (13%, 95% confidence limits 5–21%). Hospital mortality did not differ significantly between group 1 and 2 (three patients (8%) versus five patients (22%) respectively, \( P = 0.23 \)). By multivariate analysis, low operative weight (\( P = 0.023 \)), severe truncal regurgitation (\( P = 0.022 \)) and major coronary abnormalities (\( P = 0.018 \)), were independent risk factors for hospital death. Hospital survivors were followed-up from 1.3 months to 11.8 years (mean 4.2 ± 3.4 years). There were eight late deaths with survival of 73 ± 6% at 2 years and beyond. Survival was not influenced by method of RVOT resection (Conduit versus direct RV–PA anastomosis, 2.76 ± 7%, 63 ± 10%, respectively, \( P = 0.23 \)). Freedom from surgical RVOT reintervention was 56 ± 10% in group 1 and 89 ± 10% in group 2 at 10 years (\( P = 0.023 \)). The use of a xenograft conduit was an independent risk factor for reintervention (\( P < 0.001 \)). Conclusions: In truncus arteriosus repair, RV to PA continuity established by a direct anastomosis was associated with a low incidence of surgical RVOT re-intervention. This technique has the potential for RVOT growth and may be a useful alternative when an appropriate allograft is unavailable, particularly in the neonate where the risk of pulmonary hypertension are lower. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Truncus Arteriosus; Conduit

1. Introduction

The first successful correction of truncus arteriosus was performed in 1965 by McGoon, Rastelli and Ongley [1], in which a valved aortic allograft was used to establish right ventricular to pulmonary artery continuity. Since then there have been many advances in the surgical management with a clear trend to perform primary repair in early infancy [2,3], thereby avoiding the complications of pulmonary hypertension. A number of challenges still remain including the management of associated lesions or anatomical subgroups and the optimum method of establishing right ventricular to pulmonary artery continuity.

Reconstruction of the right ventricular outflow tract with the allograft valved conduit has been generally accepted as the method of choice in neonatal truncus repair due to the good haemodynamic profile and tissue handling properties [2,3]. However, the use of allografts is limited by their lack of availability, particularly in the smaller sizes appropriate for the neonatal population and delaying the procedure until a suitable allograft becomes available increases the risk to the patient. An alternative is the xenograft valved conduit and Dacron porcine conduits have been used successfully in the repair of truncus arteriosus [2,4]. However, compared with the allograft, the porcine valve is associated with a higher rate of re-intervention due to early obstruction [5].

In 1990 Barbero-Marcial described a method of recon-
struction the RVOT by creating a direct anastomosis between the right ventricle and pulmonary artery confluence, and thus avoided the use of an extracardiac conduit [6]. By utilizing the patient’s tissues this reconstruction has the potential to grow and thus may avoid or delay re-operation [7]. The principal concern with the method is the absence of a functioning trileaflet pulmonary valve in the setting of repaired truncus arteriosus, with the risk of postoperative pulmonary hypertension. Previous studies have indicated the technique is associated with higher operative mortality, and should be avoided [4].

In recognising the fact that the perfect conduit for RVOT reconstruction in infant truncus repair does not exist, we consider that the choice of method of establishing right ventricle to pulmonary artery continuity should be individualized for each patient, based on conduit availability and underlying anatomy. In general we have favoured the pulmonary allograft conduit as the first choice, and have employed the direct right ventricle-pulmonary artery RV–PA anastomosis in the neonate or younger infant where the risks of pulmonary hypertension are lower.

This study analyzes the outcome of 61 consecutive patients who underwent repair of truncus arteriosus, in which RVOT reconstruction was accomplished by either conduit or direct RV–PA anastomosis. The effect of RVOT reconstruction on hospital mortality, survival and re-intervention is presented.

2. Patients and methods

2.1. Patients

The study group consisted of 61 consecutive patients (31 female, 30 male) who underwent surgical correction of truncus arteriosus by a single surgeon, (W.J.B.), at the Birmingham Children’s Hospital between November 1988 and July 2000. Primary repair was performed in the majority (n = 58, 95%) with three patients having undergone a prior palliative procedure (main pulmonary artery band, n = 1, left modified Blalock–Taussig shunt with right pulmonary artery banding, n = 1 and bilateral branch pulmonary artery banding, n = 1). Associated cardiovascular and extra-cardiac anomalies are listed in Table 1. The median age at complete repair was 34 days (range 2 days to 6.4 months) and 49% of patients (n = 30) were less than 30 days of age. The median weight of patients at the time of operation was 3.2 kg (range 1.7–8.6 kg) and 25 patients (41%) weighed less than 3 kg. Truncus types according to the Van Praagh classification [8] were type A1 or A2 (n = 50, 82%), type A3 (n = 4, 7%) and type A4 (with interrupted arch, n = 7, 11%).

2.2. Operative procedures

Repair of truncus arteriosus was performed with deep hypothermic cardiopulmonary bypass. Intermittent periods of circulatory arrest were employed in 54 patients (89%) to optimize visualization during pulmonary artery excision, VSD exposure and repair of interrupted arch. Myocardial protection was achieved by cold crystalloid cardioplegia and topical ice slush in all patients. The median cross-clamp time was 76 min (range 51–154 min). The median circulatory arrest period was 49 min (range 3–108 min).

The pulmonary arteries were excised from the ascending aorta by careful dissection to prevent injury to the truncal valve or left coronary artery. The truncal valve was then inspected to determine morphology and function. The defect remaining in the ascending aorta was then closed either directly or with a patch when it was considered direct closure would potentially distort the truncal valve or compromise the coronary ostia.

A vertical right ventriculotomy was then made and the ventricular septal defect closed. Two types of VSD were recognized: those with a complete muscular border (muscular n = 32, 53%) and those in which the VSD was in continuity with the tricuspid valve annulus (perimembranous, n = 29, 47%). In eight patients the VSD was considered restrictive by intra-operative assessment and was enlarged towards the left side (one patient developed transient postoperative heart block). The VSD was closed with bovine pericardium in the neonates and Dacron patch in the older children using interrupted Teflon buttressed sutures.

2.3. Right ventricular outflow tract reconstruction

A variety of methods were used to establish right ventricle to pulmonary artery continuity, based on allograft availabil-

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Truncal valve insufficiency</td>
<td>17</td>
</tr>
<tr>
<td>Mild</td>
<td>5</td>
</tr>
<tr>
<td>Moderate</td>
<td>7</td>
</tr>
<tr>
<td>Severe</td>
<td>1</td>
</tr>
<tr>
<td>Hypoplastic or stenotic</td>
<td>5</td>
</tr>
<tr>
<td>pulmonary arteries</td>
<td></td>
</tr>
<tr>
<td>Right aortic arch</td>
<td>11</td>
</tr>
<tr>
<td>Interrupted aortic arch</td>
<td>7</td>
</tr>
<tr>
<td>Persistent left superior vena cava</td>
<td>5</td>
</tr>
<tr>
<td>Coronary artery abnormalities</td>
<td>9</td>
</tr>
<tr>
<td>Intramural left coronary artery</td>
<td>3</td>
</tr>
<tr>
<td>LAD or RCA conal branch</td>
<td>3</td>
</tr>
<tr>
<td>crossing infundibulum</td>
<td></td>
</tr>
<tr>
<td>Abnormal ostia position</td>
<td>3</td>
</tr>
<tr>
<td>DiGeorge syndrome</td>
<td>14</td>
</tr>
<tr>
<td>Major Sketelal abnormality</td>
<td>4</td>
</tr>
<tr>
<td>Imperforate anus</td>
<td>1</td>
</tr>
<tr>
<td>Necrotising enterocolitis</td>
<td>1</td>
</tr>
<tr>
<td>Pre-operative mechanical ventilation</td>
<td>13</td>
</tr>
</tbody>
</table>

* LCA, Left coronary artery; RCA, right coronary artery.
ity and underlying anatomy (Table 2). From this, two groups were defined for subsequent analysis, based on the presence of an extra-cardiac conduit in the reconstructed RVOT. Group 1, designated conduit repair, comprised patients in whom allograft and xenograft valved conduits were used. Group 2, direct anastomosis repair, the RVOT was reconstructed by a direct anastomosis between the posterior wall of the pulmonary artery and the right ventriculotomy.

**Group 1.** In 28 infants a cryopreserved allograft valved conduit was used comprising 16 aortic and 12 pulmonary allografts, ranging in size from 8 to 18 mm. The conduit was anastomosed to the infundibulotomy either directly or by constructing a hood to ‘roof-over’ the anterior portion of the anastomosis to prevent conduit distortion. The hood consisted of anterior mitral valve remnant in the aortic allograft and bovine pericardium in the pulmonary allografts.

In ten patients a variety of xenografts valved conduits were used: porcine valve housed in a Dacron conduit \( (n = 1) \) (Hancock, Medtronic, Inc., Minneapolis, MN), porcine pulmonary valve (Shellhigh, Inc., \( n = 2 \), TissueMed \( n = 3 \)), gluteraldehyde fixed lamb’s pulmonary valve \( (n = 3) \) [9] and bovine jugular conduit (VenPro, Contegra\textsuperscript{TM} \( n = 1 \)) [10], ranging in size from 12 to 15 mm.

**Group 2.** In 23 patients right ventricular to pulmonary artery continuity was established without an extracardiac conduit. The pulmonary artery was separated from the trunk and a direct anastomosis between the pulmonary arteries and the right ventricle was performed. In three patients with type 2A anatomy, a strip of bovine pericardium was interposed between the pulmonary artery confluence and the right ventricle to construct the posterior wall of the new pulmonary trunk. The anterior wall of the RVOT reconstruction was completed with either a simple non-valved bovine pericardial patch \( (n = 8) \), or an allograft monocusp patch \( (n = 15) \). Anterior relocation of the pulmonary arteries (Lecompte manoeuvre) was performed in one patient [11].

**2.4. Truncal valve procedures**

Eight patients had moderate to severe truncal valve regurgitation as defined by echocardiogram and intra-operative evaluation. At the initial procedure six patients underwent valve repair consisting of leaflet suspension, cusp repair or commissural plication with two hospital deaths. One patient underwent truncal root replacement with hospital death. Following repair, the truncal valve function was assessed intra-operatively by delivering cardioplegia and inspecting the truncal valve from below, though the right ventriculotomy. In addition all patients had epicardial echocardiographic assessment following termination of cardiopulmonary bypass. Three patients required truncal root replacement in the early post-operative period because of severe valvular regurgitation; two patients had undergone a previous valve repair at the initial operation (post-operative interval 31 and 55 days, no deaths) while one patient had no previous procedure on the valve (post-operative interval 11 days, hospital death). All truncal root replacements were performed with a cylinder of cryopreserved allograft ranging in size from 12 to 15 mm (aortic in three, pulmonary in one). The coronary arteries were excised with a button of aortic wall and implanted into the allograft.

**2.5. Aortic arch repair**

Seven patients (11%) underwent aortic arch repair for interruption \( (\text{type A} \ n = 2, \text{type B} \ n = 4) \) or coarctation \( (n = 1) \) with one hospital death in the group (14%). Their ages ranged from 3 to 141 days (median 38 days). Following systemic cooling to deep hypothermia, the circulation was arrested, the aortic cannula removed and the arch vessels snared. The ascending and descending aorta was widely mobilized and an end to side direct anastomosis fashioned between the descending aorta and the underside of the arch. In three cases the anastomosis was supplemented with a homograft patch.

**2.6. Non-confluent pulmonary arteries**

Four patients (7%) displayed type 3A anatomy with non-confluent pulmonary arteries. In all four patients the right pulmonary artery arose from the usual position on the truncus with the left pulmonary artery arising from different positions: a normally placed ductus arteriosus \( (n = 2) \), a closed ductus arteriosus from a left innominate artery \( (n = 1) \) and from two major aorto-pulmonary collaterals arteries (MAPCAs) supplying the left lung. In three patients with a duct-derived LPA a primary anastomosis between the divided ends of the mobilized left and right pulmonary arteries was fashioned and a valved conduit (allograft, \( n = 2 \), xenograft \( n = 1 \)) was anastomosed to the newly created pulmonary confluence. One patient, in which the LPA was of diminutive size, underwent an initial left systemic to LPA shunt using a 4 mm Gore-Tex shunt and banding of the right pulmonary artery in order to develop LPA size prior to the complete repair. Successful complete repair was performed after a 10 month interval. The patient with the left MAPCAs underwent a preliminary left thoracotomy prior to the complete repair. Both MAPCAs arose from the descending left-sided thoracic aorta and passed

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**Table 2**

Methods of right ventricular outflow tract reconstruction with associated hospital mortality

<table>
<thead>
<tr>
<th>RVOT construction</th>
<th>Patients No. (%)</th>
<th>Early mortality No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1. Extracardiac conduit</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allograft</td>
<td>28 (46)</td>
<td>2 (7.1)</td>
</tr>
<tr>
<td>Aortic</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Pulmonary</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Xenograft</td>
<td>10 (16)</td>
<td>1 (10)</td>
</tr>
<tr>
<td><strong>Group 2. Direct RV to PA connection</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allograft monocusp</td>
<td>15 (25)</td>
<td>5 (33.3)</td>
</tr>
<tr>
<td>Xenograft pericardial patch</td>
<td>8 (13)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>
anterior to the oesophagus and behind the trachea before entering the hilum of the left lung; each vessel was mobilized then, through a median sternotomy on cardiopulmonary bypass the MAPCAs were divided from their aortic origin and re-routed into the pericardial cavity via a pericardial window posterior to the phrenic nerve. The vessels were then anastomosed together initially, then joined to the mobilized right pulmonary artery; the pulmonary arterial confluence was connected to the right ventricle with a 14 mm aortic valved allograft.

Delayed sternal closure was performed in 47 patients (77%) with temporary cover provided by either skin approximation or a perforated Gore-Tex membrane. Following haemodynamic stability, usually within 24 h of operation, the sternum was formally closed in the ITU. Routine sedation with continuous morphine and midazolam intravenous infusion and neuromuscular blockade was practiced for the first 24 h. Pulmonary artery, left and right atrial pressures were monitored.

2.7. Data collection and statistical analysis

Preoperative and perioperative data were collected on retrospective review of patient records. Follow-up information was obtained between April and July 2000 by physician or patient contact and was obtained in all patients. Hospital mortality was defined as death within 30 days of operation or during the same hospital admission. SPSS for Windows version 8.0 (SPSS Inc., Chicago, IL) was used to perform statistical calculations. Data are expressed as median and either range or 95% confidence limits. Continuous variables were analyzed using independent $t$-test or Mann–Whitney $U$-test when data was not normally distributed. Data are expressed as median and either range or 95% confidence limits. Continuous variables were analyzed using independent $t$-test or Mann–Whitney $U$-test when data was not normally distributed. Pearson $\chi^2$ or Fisher’s exact test were used to determine differences when variables were expressed by dichotomous values. Potential risk factors associated with hospital mortality were analyzed using multiple Logistic regression performed as conditional forward stepwise procedure. Survival and freedom from RVOT reoperation were examined by the Kaplan–Meier product limit method and $P$ values for difference between distributions were obtained by the log-rank test. Potential risk factors associated with RVOT reoperation were analyzed by Cox proportional hazards regression using a forward conditional stepwise method.

3. Results

There were eight hospital deaths (13%; 95% confidence limits 4.6–21.4%) and causes are listed in Table 3. Four deaths were associated with severe truncal regurgitation (4/8 with this lesion), two were associated with coronary artery injury and one with aortic interruption.

When hospital death was stratified according to the type of RVOT reconstruction, direct RV–PA anastomosis with monocusp was associated with a higher mortality (Table 2).

Univariate analysis of the twenty variables, including method of RVOT reconstruction, examined as potential risk factors for hospital death (see Appendix A) revealed five significant variables (Table 4). Multivariate analysis by stepwise logistic regression identified three independent significant risk factors: small patient weight (continuous variable), greater than moderate truncal valve regurgitation and presence of coronary artery anomalies. The results of multiple regression analysis should be interpreted with caution because of the small population size and small number of dependant variable events (eight deaths). However the method of RVOT reconstruction consistently failed to enter the model despite the higher hospital death rate in infants who underwent RVOT by direct RV–PA anastomosis, specifically with the monocusp subgroup.

3.1. Follow-up

The mean duration of follow up was 50 ± 41 months, range 1.3 months to 11.8 years.

3.2. Late deaths

There have been eight deaths in the 53 patients

Table 3

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Time interval (days)</th>
<th>Age at repair (days)</th>
<th>Weight at repair (kg)</th>
<th>RVOT construction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Massive pulmonary haemorrhage following CPB</td>
<td>On table</td>
<td>2</td>
<td>1.7</td>
<td>Mono</td>
</tr>
<tr>
<td>Could not be weaned from CPB during reoperation for truncal root replacement</td>
<td>On table</td>
<td>55</td>
<td>2.4</td>
<td>Mono</td>
</tr>
<tr>
<td>Pulmonary hypertensive crisis</td>
<td>3 h</td>
<td>25</td>
<td>2.7</td>
<td>Mono</td>
</tr>
<tr>
<td>CP arrest</td>
<td>9 h</td>
<td>18</td>
<td>1.8</td>
<td>Mono</td>
</tr>
<tr>
<td>CP arrest following closure of post-op open sternum</td>
<td>1 day</td>
<td>25</td>
<td>2.4</td>
<td>Homo</td>
</tr>
<tr>
<td>LV failure following repair with truncal root replacement</td>
<td>2 days</td>
<td>13</td>
<td>3.4</td>
<td>Hetero</td>
</tr>
<tr>
<td>Myocardial infarction, LCA injury</td>
<td>8 days</td>
<td>99</td>
<td>3.3</td>
<td>Homo</td>
</tr>
<tr>
<td>RV failure, previous PAB, large RCA branch transacted, systemic RV pressure, died at reop</td>
<td>26 days</td>
<td>132</td>
<td>3.0</td>
<td>Mono</td>
</tr>
</tbody>
</table>

* CP, cardiopulmonary; CPB, cardiopulmonary bypass; LV, left ventricular; L (R) CA left (right) coronary artery; PAB, pulmonary artery band; reop, reoperation.
discharged from hospital ranging from 2 to 22 months after the operation. Four patients died of pneumonia or septic episodes 2, 2, 6 and 22 months following surgery; only one patient had DiGeorge syndrome. Two patients died as a result of right ventricular outflow tract obstruction. One patient in whom RVOT reconstruction was performed by direct RV-PA anastomosis with pericardial patch developed acute RVOT obstruction and died 4 months after operation. Post-mortem examination confirmed a discrete stenosis at the level of the RV-PA anastomosis due to the formation of excessive granulation tissue. Another patient, who underwent RVOT reconstruction with a size 11 pulmonary allograft, died of conduit obstruction 5 months after surgery. This patient had been under medical review in another country. One patient, who had undergone initial repair with a monocusp RVOT reconstruction at 4 months of age, developed severe right ventricular failure in association with bilateral branch pulmonary artery stenosis and established pulmonary hypertension in the left lung. She was transferred for pulmonary artery repair and placement of RV to PA conduit but sustained a sudden cardiovascular collapse following admission. Finally one patient, previously well, had sudden death of unknown cause, 8 months after operation.

Survival for all patients was 92 ± 4% at 1 month, 79 ± 5% at 6 months and 73 ± 6% at 2 years and beyond (Fig. 1). There was no additional mortality after the second post-operative year. There was no significant difference in survival between the two methods of RVOT reconstruction (Fig. 2).

### 3.3. Re-intervention

Surgical revision of the RVOT including conduit replacement has been performed in 11 of the 53 hospital survivors at a median interval of 17 months (range 6–53 months) after initial repair. Seven patients (70%) with xenograft conduits have undergone re-operation at an interval of 7–48 months and three patients (12%) with allograft conduits have been replaced at an interval 6 to 28 months. One patient (4%) with direct RV-PA anastomosis has undergone re-operation for stenotic RVOT and RVOT patch aneurysm. Freedom from re-operation for all hospital survivors was 69 ± 8% at 54 months and beyond. Freedom from re-operation, stratified for method of RVOT reconstruction is shown in Fig. 3. Direct RV-PA anastomosis was associated with an improved freedom from re-operation (With conduit 58 ± 10%, direct anastomosis 89 ± 10% P = 0.035). No patient died at re-operation.

Percutaneous balloon dilation of pulmonary arteries was performed in seven patients (four direct RV-PA anastomosis, three conduit) with a median interval of 20 months (13–34 months) after initial repair. Freedom from all re-inter-

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**Table 4**

Factors found to be associated with increased operative mortality by univariate and multivariate logistic regression analysis

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Univariate P value</th>
<th>Multivariate P value</th>
<th>Odds ratio (95% confidence limits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight at operation</td>
<td>0.016</td>
<td>0.023</td>
<td>0.05 (0.005–0.67)</td>
</tr>
<tr>
<td>Presence of coronary abnormalities</td>
<td>0.021</td>
<td>0.022</td>
<td>0.037 (0.002–0.61)</td>
</tr>
<tr>
<td>Truncal regurgitation &gt; moderate</td>
<td>0.0023</td>
<td>0.018</td>
<td>0.031 (0.002–0.55)</td>
</tr>
<tr>
<td>Use of monocusp patch</td>
<td>0.015</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Truncal valve procedure</td>
<td>0.0043</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Fig. 1. Kaplan–Meier estimate of survival following complete truncus repair. Error bars represent the 70% confidence interval.

Fig. 2. Kaplan–Meier estimate of survival following truncus repair stratified according to RVOT reconstruction. Error bars represent the 70% confidence limits. Survival was 77 ± 7% for conduit repair 67 ± 10% for the direct RV-PA anastomosis repair at 2 years and beyond (P = 0.25).
vention, including both reoperation and percutaneous balloon dilation was 56 ± 8% at 54 months for all patients. Fig. 4 displays freedom from re-intervention stratified for method of outflow tract reconstruction. There was no significant difference in freedom from re-intervention between the two methods of RVOT reconstruction. (With conduit 58 ± 10%, and by direct RV–PA anastomosis 68 ± 10%, at 4 years and beyond (P = 0.034).

Univariate analysis of potential risk factors for re-intervention (see Appendix A) revealed use of extra-conduit (P, 0.011) and use of xenograft conduit (P < 0.001) to be significant risk factors. Multivariate analysis using a Cox regression model identified only use of a xenograft conduit to be a statistically significant risk factor for early re-intervention (P < 0.001).

4. Discussion

The surgical management of truncus arteriosus has evolved over the past decade to include primary correction in the neonatal period. Although good results have been achieved, certain challenges still remain: the management of complex anatomy, including truncal valve regurgitation, and the optimum method of establishing right ventricular to pulmonary artery continuity. This retrospective study analyses the outcome of 61 consecutive patients who underwent surgical correction of truncus arteriosus at a single institution by one surgeon. This has provided a relatively large number of patients and a degree of homogeneity in the general surgical technique, which varied only in the method of establishing right ventricular to pulmonary artery continuity. This has allowed us to examine this specific contribution of RVOT reconstruction to the outcome of these patients in terms of hospital mortality, survival and re-intervention.

Establishing right ventricular to pulmonary artery continuity in the neonatal period presents certain unique challenges. Ideally such a reconstruction would have optimum haemodynamics, including low resistance to right ventricular ejection and minimal pulmonary valvular regurgitation. Following repair of truncus arteriosus the presence of a competent pulmonary valve may be particularly important due to the risks of pulmonary hypertension [12]. Due to rapid growth in the young infant a right ventricular outflow reconstruction that ‘grew’ with the patient would provide a distinct advantage. This would potentially delay the time to conduit, or RVOT revision and ultimately reduce the total number of surgical re-interventions required. Good tissue handling properties of the conduit are also important which will reduce injury to the delicate neonatal tissues, decreasing the risk of post-operative bleeding and stenotic anastomotic suture lines. Finally the RVOT reconstruction method should not be restricted by lack of availability. Delaying the procedure until a suitable conduit becomes available increases the risk of pulmonary hypertensive disease and is not be an option for infants who present with severe congestive heart failure.

Current opinion suggests that reconstructing the RVOT with a valved allograft conduit is probably the method of choice [2,3,12]. The allograft provides good haemodynamics, including a competent valve and low resistance to ejection. The tissue handling characteristics are excellent, [13] which enables tailoring and anastomotic suturing and represents a major advantage over Dacron porcine conduits in the neonate. However, the use of this conduit may be limited by lack of availability, particularly in the smaller sizes appropriate for neonatal reconstruction. In some countries difficulties in collecting small allografts and the tight regulations imposed on biological substitutes almost precludes their regular use. In this study the allograft conduit (ideally a pulmonary allograft), when available, was the method of choice of RVOT reconstruction. The
method was associated with good hospital and intermediate survival. In the follow-up there were a total of four allograft failures (14%, three reoperations and one death due to conduit obstruction), which was significantly lower than xenograft conduit failures (7/10, 70%, \( P = 0.001 \) Fisher’s exact test). Several studies have identified risk factors for allograft failure, which include: young recipient age [14,15], small conduit size [15,16], pulmonary hypertension [17] and use of an aortic rather than pulmonary allograft [18,19]. Determining the relationship between these known risk factors and allograft failure in the present study is of limited value, due to the small population size and small number of events (four allograft conduit failures). Nevertheless it appeared that allograft failure was associated with smaller conduit size (10 versus 14 mm, \( P = 0.007 \)), younger age at operation (18 versus 71 days, \( P = \text{n.s.} \)) and lower operative weight (2.9 versus 3.9 kg, \( P = \text{n.s.} \)).

An alternative to the allograft is the freely available xenograft valve conduit and a variety of such conduits were used in this series, including porcine and lamb pulmonary valves, Dacron-housed porcine conduits, and bovine jugular vein conduits. Some studies have shown excellent results following RVOT reconstruction with porcine valved conduits with good patient survival and freedom from reoperation, comparable to allograft conduits [16,20]. Despite this, the experience appears less good in the smaller infants and those undergoing repair of truncus arteriosus, with higher rates of re-intervention compared to the cryopreserved allograft [5,21]. Conduit dysfunction can result from valve leaflet degeneration, sternal compression and intimal peel formation [20]. This is supported by the findings of the present study in which we identified a high incidence of conduit failure in the xenograft group. In addition, we identified the use of a xenograft conduit as the single independent risk factor for intervention by the Cox proportional model. These findings contrast with the data of Lacour-Gayet et al. [4] in which, in a similar study involving truncus arteriosus patients, freedom from re-intervention was superior in the Dacron porcine conduit group compared with the homograft group (77% versus 43% at 5 years, respectively). Furthermore the use of a homograft emerged as an incremental risk factor for early reoperation [4]. Ultimately in the absence of a truly prospective comparative study, the advantages of homografts over porcine conduits remain controversial.

In 1990 Barbero-Marcial described a method of establishing RV to PA continuity in type I and 2 A truncus arteriosus without the use of an extracardiac conduit [6]. In this technique the mobilized pulmonary artery confluence is anastomosed either directly to the right ventriculotomy or bridged with the left atrial appendage in type 2A anatomy. The reconstruction is completed anteriorly by placing a valved pericardial patch. This method has several potential advantages over an extracardiac conduit. Since the posterior wall is created with the patient’s tissues there is the potential for growth and this has been confirmed by MRI and angio-
may not be possible to directly attach the mobilized pulmonary arteries to the right ventricle and reconstruction of the posterior wall with the left atrial appendage may be used [6,7]. In such situations we have constructed the back wall with a strip of bovine pericardium, although this may impede anastomotic growth it avoids the risk of obstruction due to bulging of the left atrial appendage into the RVOT [7]. The principal advantage of the direct RV–PA anastomotic technique was the significantly lower incidence of reoperation compared with the conduit group. In total there have been two direct RV–PA anastomotic failures (one patch revision and one death due to RVOT obstruction). Although RVOT and conduit re-operation may be performed with a low mortality as demonstrated in the present series (no deaths in 11 re-operations), the effect of sub-optimal conduit haemodynamic prior to revision and additional periods of cardiopulmonary bypass and cardioplegic arrest are likely to have negative long-term sequel for right ventricular function and patient survival.

Hanley et al. [3] identified in the repair of truncus arteriosus, the presence of complex anatomy, defined by the presence of one or more of the following lesions: severe truncal valve regurgitation, major coronary abnormalities or interrupted arch, increased the operative risk. In this series the overall hospital mortality was 13% comparable to other published reports [2,3] and was strongly influenced by the presence of complex anatomy (simple versus complex anatomy 2.8 versus 28%, P < 0.006 by Fisher’s exact test). Both severe truncal incompetence and presence of major coronary abnormalities emerged as independent risk factors by step-wise logistic multivariate analysis. By contrast with others [3] we did not find an increased risk with the more complex anatomical subtypes of truncus arteriosus. Repair of interrupted arch was performed in seven patients with one death (14%) and non-confluent pulmonary arteries were repaired in four patients with no deaths. In the repair of type 3A TA we believe it is necessary to recruit both the right and left pulmonary circulation, even when the lung is supplied by MAPCAs. All such patients in this study are alive and symptom free at medium term follow-up. By contrast, the experience of incorporating a single lung has been associated with elevated right ventricular pressures and high mortality in the early follow-up period [3].

Management of significant truncal valve regurgitation remains a challenge, resulting in 50% (four of eight patients) hospital mortality in patients who required a truncal valve procedure. In the present series, of the four patients who underwent truncal root replacement only one had this performed as a planned part of the original procedure, while three patients underwent root replacement in the early post-operative period. This underpins the difficulties associated with assessing truncal valve regurgitation, both pre and intra-operatively. Before repair the large runoff into the pulmonary bed reduces the diastolic pressure and echocardiographic examination can under-estimate the severity of truncal valve regurgitation. The surgical options for truncal valve insufficiency vary from simple re-suspension during truncal root closure [4], to various repair techniques [25] and truncal valve replacement [2]. We have favoured a conservative approach, with repair as the initial option and truncal root replacement with a cryopreserved aortic allograft for severe regurgitation, or re-operative valve procedures.

4.1. Conclusions

In this study hospital mortality following truncus arteriosus repair was increased by the presence of severe truncal valve regurgitation and major coronary abnormalities, but was not influenced by anatomical subtype. In establishing right ventricular to pulmonary artery continuity the allograft valved conduit is preferred to the xenograft because of the superior tissue handling properties and lower incidence of conduit failure. As an alternative to a conduit, the direct RV–PA anastomosis method has the potential for RVOT growth, and was associated with a low incidence of reoperation. This technique may be considered in the neonate or younger infant, where the risks of pulmonary hypertension are less, and when an appropriate allograft valve conduit is unavailable.

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References

Appendix A. Potential risk factors for hospital mortality and potential factors associated with RVOT re-intervention

Potential risk factors for hospital mortality.

Demographic variables
- Age
- Continuous
- Less than 30 days (y/n)
- Gender (m/f)
- Weight at operation
- Continuous
- Less than 3 kg (y/n)
- Previous palliative operation (y/n)
- Pre-operative ventilation support (y/n)

Morphological variables
- Truncus anatomical subtype other than type A1 or A2 (y/n)
- Truncal valve regurgitation
- Mild or greater (y/n)
- Moderate or greater (y/n)
- DiGeorge syndrome (y/n)
- Presence of coronary abnormality (y/n)

Operative variables
- Year of operation, before 1995 (y/n)
- Perfusion time
- Cross-clamp time
- Arrest time
- RVOT reconstruction
- With conduit (y/n)
- Use of monocups patch (y/n)
- Truncal valve procedure
- VSD enlargement (y/n)

Potential factors associated with RVOT re-intervention
- Age
- Continuous
- < 30 days
- Weight
- Continuous
- < 3 kg
- Anatomical subgroup
- Van Praagh type I/II versus III/IV
- RVOT reconstruction
- Conduit RVOT repair (y/n)
- Use of Homograft (y/n)
- Use of Heterograft (y/n)

Appendix B. Conference discussion

_Dr F. Lacour-Gayet_ (LePlessis Robinson, France): We had a similar experience in Paris, and some years ago we reported a similar group of patients looking at the various methods of reconstruction. One striking difference was in the result of the homograft. I don’t know if this has something to do with the method of preservation of the homograft, but we have observed early homograft failure, occurring quite early, within 3 months after implantation.

Now, the question I want to ask you is what particular technical detail do you suggest for the Barbero–Marcial technique? We have found it sometimes difficult to place this patch inside the pulmonary so as not to create stenosis. We have also had to reoperate a significant number of patients looking at the various methods of reconstruction. One striking difference was in the result of the homograft. I don’t know if this has something to do with the method of preservation of the homograft, but we have observed early homograft failure, occurring quite early, within 3 months after implantation.

_Dr Danton_: Do you mean using the direct anastomosis, you find PA stenosis with that technique?

_Dr Lacour-Gayet_: Yes. We have had to reoperate a significant number of patients for pulmonary branch stenoses.

_Dr Danton_: I have read your paper in detail, it’s an excellent study but...
there are a number of major differences between it and our own results and conclusions. We have been careful in selecting the appropriate patient for the direct RV–PA anastomosis technique, and I think our good results, in terms of operative mortality and survival, reflect our considered approach to right ventricular outflow tract reconstruction. We favour the technique in the younger patients particularly neonates. The anatomical subtype is also important, and we have employed the technique in Van Praagh subtype 1A and 2A. When the pulmonary arteries are not in close proximity to the right ventricle or when they are non-confluent, as in subtype 3A, we have tended to avoid this technique. In such patients the extensive mobilisation required to directly anastomose the pulmonary arteries to the right ventricle may produce excessive tension and result in PA branch stenosis. Furthermore, if the truncal valve is significantly regurgitant, we would favour reconstructing the RVOT with a valved conduit and thereby avoiding potentially two incompetent outflow valves.

Finally compared with your patients, our patients in whom the direct anastomosis repair was performed were younger, with the majority being in the neonatal group. We believe that if the repair is performed late, with the development of pulmonary hypertension, the direct anastomosis technique may be poorly tolerated due to significant pulmonary regurgitation.

**Dr G. Ziemer (Tubingen, Germany):** I’m doing direct anastomosis also, but not in the Barbero–Marcial technique, I rather detach the pulmonary artery and connect it to the infundibulotomy. However, I do it much less frequently than you do.

One concern I have is at the point where you anastomose the dorsal part of the pulmonary artery, while on the other side you will have the patch for the VSD. So with a very short length, you will have some point where there is a circumference with no possible growth, in the back the VSD-patch, in the front the RVOT-patch. So do you do something about it? Do you suture your patch far down in the myocardium? I sometimes see at the end of the operation that there is the patch ending at the epicardium of the VSD, and then I put my outflow tract patch, either pericardium, or even Gore-Tex, too, and so there is this length of 2 or 3 mm where there will be no growth. I have not had any problem with this, but it occurred in neonatal pulmonary atresia, so I am concerned. Do you have any specific approach to this region in order to avoid these possible problems?

**Dr Danton:** I will offer my explanation, but I would defer to Mr Brawn on this. In a number of patients, I think four in this series, where things have not looked right in terms of creating a direct anastomosis, we have put in an interposition strip of pericardium, about 0.5 cm in width, that has allowed a short bridge. In the original paper Barbero–Marcial has used the appendage of the left atrium to achieve the same function, but there have been some reports that can herniate into the outflow tract and obstruct. Mr. Brawn?

**Dr W. Brawn:** I think you are right, there is a problem. You hope for viable tissue, but in practice it’s difficult to achieve. In the majority of instances the PA has been attached to the right ventricle or to the muscle margin rather than to the VSD patch itself, but I agree with you, there are some situations where the two abut together and you wonder whether it’s going to be of use or not. I think it is notable, though, that even in the neonate the actual connection that you make is quite large, so there is potential for there to be space for the child to grow quite a bit, but we’re not pretending that that won’t need replacing in the long-run. They just last longer than the very small homograft in the same situation.

**Dr Ziemer:** It is good to see that this technique works in a larger series also.

**Dr R.H. Anderson (London, UK):** I do not mean this as any criticism of what you say, but I think if you check up, you’ll find that the procedure was first done by the late Kenneth Reid in Edinburgh and was published several years before Barbero–Marcial, and I think Barbero–Marcial also acknowledged that in his paper.

**Dr Danton:** You are indeed correct. Dr. K. Reid published the direct RV–PA anastomosis method for correction of Truncus Arteriosus in 1985 in the *British Heart Journal*. We will acknowledge this in our manuscript.