The neoaortic root in children with transposition of the great arteries after an arterial switch operation

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Abstract

OBJECTIVES: Neoaortic root changes in children with transposition of the great arteries (TGA) are reportedly risk factors for the development of neoaortic regurgitation (NeoAR). The aims of this study were to assess the neoaortic root diameter and relative proportion in children with TGA after surgical correction and to identify possible correlations with the development of neoarotic insufficiency.

METHODS: Of the 611 children who had the arterial switch operation performed in the Cardiology Department of the Polish Mother Memorial Hospital, 172 consecutive patients were qualified for this study. The inclusion criteria were: anatomical correction performed during the neonatal period, more than 10 years of postoperative observation and at least two full echocardiographic examinations.

RESULTS: NeoAR increased during postoperative follow-up and at the end of the observation period, 76% of the patients had NeoAR (27%-trace, 42%-mild, 7%-moderate and 0.6%-severe). Among the analysed risk factors for NeoAR development, the significant ones were arterial valve discrepancy (OR = 2.05; 95% CI: 1.04–4.02; P = 0.031) and the non-facing commissures (OR = 4.05; 95% CI: 1.34–11.9; P = 0.01). The neoaortic root diameter was not statistically significantly correlated with the presence of NeoAR or with the heart defects associated with transposition. The neoaortic root was initially, on average, 37% (z-score = 2.09) bigger than the aortic root in healthy children. This disproportion increased during the follow-up evaluations to 57% (z-score = 2.09).

CONCLUSIONS: The neoaortic root in children after the arterial switch procedure develops differently from that in healthy children, but this is not evidently related to NeoAR development or associated heart defects.

Keywords: Neoaortic valve • Aortic insufficiency • Root dilatation

INTRODUCTION

During the arterial switch procedure to correct transposition of the great arteries (TGA) in neonates, a neoaortic root is created by transplantating the coronary arteries to the native pulmonary sinus, and connecting the native pulmonary valve and its sinus to the ascending aorta [1]. Neoaortic root changes in children with TGA are described as risk factors for neoaortic regurgitation (NeoAR) in this population [2, 3]. Many studies support the view that enlargement of the neoaortic root and pathological dilatation can cause poor coaptation of the leaflets of the valve, leading to the development of NeoAR [4–6]. Numerous papers also report that changes in the particular root diameter or alteration in the relative proportion of these dimensions can lead to changes in root geometry and ultimately, neoaortic valve regurgitation [5, 6]. The complicated shape of the suture lines, plus the transplanted coronary arteries and even the Lecompte manoeuvre itself, can alter the geometry of the neoaortic root and thus cause NeoAR to develop.

The aims of this study were to assess the neoaortic root diameters and relative proportion in children with TGA after surgical correction and to identify possible correlations with the development of NeoAR.

PATIENTS AND METHODS

Study group

Between the years 1992 and 2011, the Cardiosurgery Department of the Polish Mother’s Memorial Hospital carried out an arterial switch operation (ASO) in 611 patients with TGA. The overall mortality was 6.8%, but with time, the mortality fell to 0.8% over the last 4 years. We selected 172 consecutive survivors from the patient group who met the following inclusion criteria: ASO performed in the neonatal period because of TGA; over 10 years of postoperative observation, with at least two postoperative echocardiographic examinations performed in our Cardiology Department, with the most recent performed more than 10 years after surgery. Patients with a two-step surgical procedure
(pulmonary artery banding prior to switch) and those with late switch procedure (more than 30 days after birth) were excluded.

The patients were divided into three groups on the basis of the congenital heart defect(s) associated with TGA:

(i) 'simple' (isolated) TGA—109 patients;
(ii) TGA associated with ventricular septal defect (VSD)—51 patients;
(iii) TGA associated with aortic arch anomalies—12 patients.

In each group, patients with and without any signs of neoaortic incompetence at the end of the follow-up were analysed separately.

The surgical technique employed has been described previously [7]. All of the procedures were performed by one surgeon (J.J.M.). The diagnosis process was consistent and the patients received similar pre- and postoperative care.

Data collection

We reviewed the patients’ medical records retrospectively to establish the clinical and echocardiographic data before surgery, during the perioperative period and postoperatively. All of the available demographic and clinical data were assessed as potential risk factors associated with the development of NeoAR. Patients with missing data or who had undergone their last echocardiographic evaluation more than 2 years previously were invited to attend a clinical examination. Simultaneously, echocardiographic examinations were performed on patients who had routine control follow-up visits in the Cardiology Department.

All of the echocardiographic measurements of the respective diameters of the aortic annulus (AA), aortic sinus (AS) and sinotubular junction (STJ), as well as the NeoAR grade evaluations, were collected separately for each postoperative year of observation in the three groups (Fig. 1). As these root diameters are time, weight and height dependent in children, the measurement in the three groups (Fig. 1). As these root diameters are time, weight and height dependent in children, the normalized indexes we used to investigate the correlations between neoaortic root diameters and NeoAR were as follows:

(i) AS to AA ratio;
(ii) AS to body surface area ratio;
(iii) STJ to AA ratio;
(iv) STJ to body surface area ratio.

The last index was used to correlate the development of the neoaortic root in children after the arterial switch procedure in comparison with the healthy population:

We also calculated z-score values for the AA for the data from the beginning and the end of follow-up (z-score $AA = \frac{AA - AAn}{SDn}$, where $AAn$—average normal value of AA, $SDn$—standard deviation of the AA diameter in healthy population).

These indexes are independent of the child’s growth and development and allow direct comparison of the root proportion and diameter between the groups with and without NeoAR at the end of follow-up. For each patient, the average values and the differences from baseline to postoperative follow-up of the AS and STJ to AA ratio were calculated and included in the risk factor analysis.

Echocardiography

For a proper evaluation of the NeoAR and neoaortic root parameters as well as the changes that occurred during follow-up, we included in the analysis only those echocardiographic examinations performed in our Cardiology Department, which were overseen by a senior paediatric cardiologist (J.A.M.). However, all of the patients had yearly routine follow-up visits in the ambulatory clinic with echocardiographic evaluation. In the case that NeoAR occurred, its grade increased or there were other complications, children were admitted to the ward for clinical and echocardiographic evaluation. In this manner, it was possible to precisely establish the time at which NeoAR occurred, along with simultaneously checking the diameter of the aortic root.

We retrospectively analysed 1195 echocardiographic examinations performed in the Cardiology Department between the years 1992 and 2008. Seventy-eight percent of them analysed all of the parameters, the neoaortic root included. Additionally, we performed 78 full echocardiographic examinations in the patients who had their last echocardiogram prior to 10 years of follow-up or more than 2 years before 2008.

Neoaortic insufficiency was evaluated on a five-point scale: Grade 0 = none; Grade 0.5 = trivial; Grade 1 = mild; Grade 2 = moderate; Grade 3 = severe. This scale was based on semiquantitative evaluation of aortic regurgitation in paediatric cardiology [8, 9]. The neoaortic root was measured at the level of the AA (in diastole), the AS (at the widest diameter) and the STJ (the hyperechogenic zone resulting from the suture line between the native pulmonary valve and the ascending aorta) (Fig. 1).

Statistical analysis

Statistical analyses were performed using Statistica 9.0 software. Quantitative data are presented as the mean and standard deviation. Qualitative data are presented as percentages. NeoAR occurrence was shown using Kaplan–Meier survival curves, and the threshold for the data used was the occurrence of mild regurgitation or higher. Differences between groups were assessed by univariate analysis ($\chi^2$ test), or the Kruskall–Wallis test, and time-dependent differences were analysed by the $\chi^2$ test and log-rang test. We also calculated the risk of NeoAR occurrence at different times after surgery. All risk factors were assessed by univariate logistic regression. Multivariate logistic regression was constructed using potential risk factors found to have a $P$-value (values) $<0.2$ on the univariate analysis. The optimal regression
model was created using a stepwise strategy, and was assessed with the Hosmer–Lemeshow test.

We used logistic regression and Cox regression to analyse the derived indexes as independent risk factors for NeoAR (a mild regurgitation or higher was used as an end point in both analyses). For each patient, we calculated the mean value of the indexes normalized to the AA as well as the maximal value of the difference between the initial value and the value at the end of the follow-up. To check differences between the average ratios of the values for each year in those with and without neoaortic insufficiency at the end of follow-up, patients were matched in terms of the duration of follow-up and were assessed by analysis of variance (ANOVA). The average value and the difference during the follow-up of AS/AA and STJ/AA for each patient were also included into the multivariate analysis.

Summary data from the indexes are presented as time-dependent graphs with Spearman’s correlation test (R—Spearman correlation coefficient). A P-value of <0.05 was considered statistically significant.

RESULTS

Neoaortic regurgitation

One hundred and seventy-two patients were included in this analysis. The average time of follow-up was 13.5 years (SD 2.4). Table 1 shows the demographic and clinical data. NeoAR was a common finding. Trivial insufficiency of the native pulmonary valve was observed in 9 patients (6%), and 38 (22%) had signs of NeoAR at the first postoperative examination. In the first year of observation, the number of patients with neoaortic insufficiency fell to 17 (10%). However, in subsequent years the prevalence of regurgitation increased steadily to finally reach 85 (49%). These data exclude cases of trivial insufficiency, which comprised a further 27% of the patients (Fig. 2). Despite the good linear correlation between NeoAR and time (R = 0.98), it can be seen in Fig. 2 that a logarithmic function better describes the development of NeoAR in the postoperative period (better), as the biggest increase in new cases of NeoAR occurred in the first years after surgery and then gradually decreased during the period of postoperative observation. At the end of follow-up, NeoAR was mild in the majority of cases (85%), moderate in 15% and severe in 1%. Only 28% of patients exhibited no change in neoaortic competence over the whole follow-up period. In 27%, insufficiency increased by 0.5 grade, in 39% by one grade, in 1% by 1.5 grade and in 5% by two grades. The mean increase in the grade of neoaortic insufficiency was 0.64 and there were no statistically significant differences between the groups (P = 0.58, Kruskall–Wallis test).

The differences in NeoAR occurrence between the groups were not statistically significant on univariate analysis (χ² test; P = 0.83). Similarly, there were no statistically significant differences on the time-related analysis (Kaplan–Meier, P = 0.98 χ², log-rank test; Fig. 2). The highest risk of neoaortic insufficiency was between the first and sixth postoperative year. The risk then decreased (Fig. 3). Of the factors we analysed, the only significant risk factors on univariate analysis were non-facing commissures and pulmonary and aortic valve (AoV) discrepancies (Table 2). These were also confirmed as independent risk factors on the multivariate logistic regression analysis (pulmonary and AoV discrepancies: OR = 2.10; 95% CI: 1.04–4.02; P = 0.031; non-facing commissures: OR = 4.05; 95% CI: 1.34–11.9; P = 0.01), as well as on the Cox regression time-related analysis (pulmonary and AoV discrepancies: β = 0.51; 95% CI: 0.02–1.01; RR = 1.67; P = 0.04; non-facing commissures: β = 0.61; 95% CI: 0.01–1.21; RR-1.84; P = 0.046).

Neoaortic root development

We found no significant differences in the average values of the calculated indexes for each year between the analysed groups or between children with and without postoperative neoaortic insufficiency development (Figs 4 and 5) (ANOVA test; AS to AA ratio: F = 1.19, P = 0.31; STJ to AA ratio: F = 1.29, P = 0.28; AS to body surface area ratio: F = 0.11, P = 0.09; STJ to body surface area ratio: F = 0.06, P = 0.99). This lack of significance led us to analyse the data derived from the whole study group together, and to correlate this data with the time of observation. The last index used was the ratio of the AA to the average AAn, which

| Table 1: Demographic and perioperative data in patients divided into the three analysed groups |
|---------------------------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
| Sex (♂-male, ♀-female)                     | TGA (N = 109)               | TGA + VSD (N = 51)          | TGA + AAA (N = 12)          | P-value                    |
| Follow-up (years)—average (SD)             | 13.6 (±2.5)                 | 13.4 (±2.1)                 | 12.9 (±2.06)                | 0.88                       |
| Age at operation (days)—average (SD)       | 6.3 (±2.6)                  | 15 (±13.1)                 | 17.7 (±15.1)                | <0.001                     |
|Weight at operation (g)—average (SD)        | 3350 (±560)                 | 3496 (±567)                 | 3066 (±427)                 | 0.1                       |
| Ao cross-clamping time (min)—average (SD)  | 69 (±10.6)                  | 86 (±17.4)                 | 98 (±10)                    | <0.001                     |
|Stay at ICU (days)—average (SD)             | 5.5 (±4.6)                  | 4.5 (±2.3)                 | 4.9 (±1.85)                 | 0.28                       |
|Non-facing commissures, n (%)               | 9 (8)                       | 12 (24)                    | 4 (33)                      | 0.06                       |
|PAV > AoV discrepancy, n (%)                | 40 (37)                     | 26 (51)                    | 12 (100)                    | <0.001                     |
|Coronary anomalies, n (%)                   | 35 (32)                     | 17 (33)                    | 6 (50)                      | 0.46                       |
|Perioperation complications, n (%)          | 29 (27)                     | 12 (24)                    | 3 (25)                      | 0.92                       |
|Complications at birth, n (%)               | 18 (17)                     | 7 (14)                     | 3 (25)                      | 0.63                       |

Values in bold indicate statistically significant differences and values in italics indicate the differences which are close to the statistical significance. VSD: ventricular septal defect; AAA: aortic arch anomalies; TGA: transposition of the great arteries; SD: standard deviation; PAV: pulmonary artery valve; AoV: aortic valve; ICU: intensive care unit.
Figure 2: Freedom from neoaortic insufficiency in the analysed groups against time after surgery. Data included mild insufficiency or greater (trace NeoAR was excluded). VSD: ventricular septal defect; AAA: aortic arch anomalies; TGA: transposition of the great arteries.

Figure 3: The risk density for NeoAR development with time after surgery. The data included mild insufficiency or greater (trace NeoAR was excluded).
Table 2: Univariate analysis of potential risk factors for NeoAR occurrence

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR (95% CI)</th>
<th>P-value</th>
<th>OR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAV/AoV discrepancy (PAV &gt; AoV)</td>
<td>2.51 (1.33–4.58)</td>
<td>0.004</td>
<td>2.05 (1.04–4.02)</td>
<td>0.031</td>
</tr>
<tr>
<td>Coronary anomalies</td>
<td>1.48 (0.75–2.92)</td>
<td>0.25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-facing commissures</td>
<td>5.04 (1.78–14.28)</td>
<td>0.002</td>
<td>4.05 (1.34–11.9)</td>
<td>0.01</td>
</tr>
<tr>
<td>Perinatal complication</td>
<td>1.15 (0.49–2.65)</td>
<td>0.74</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgery complications</td>
<td>0.86 (0.41–1.78)</td>
<td>0.68</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at operation (days)</td>
<td>1.02 (0.97–1.08)</td>
<td>0.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight at operation (kg)</td>
<td>0.99 (0.99–1.0)</td>
<td>0.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ao crossclamping time (min)</td>
<td>1.01 (0.99–1.04)</td>
<td>0.12</td>
<td>1.01 (0.32–11.3)</td>
<td>0.46</td>
</tr>
<tr>
<td>Stay at ICU (days)</td>
<td>1.03 (0.93–1.13)</td>
<td>0.57</td>
<td></td>
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<tr>
<td>Mean value: AS/AA</td>
<td>0.48 (0.03–8.77)</td>
<td>0.62</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximal difference: AS/AA</td>
<td>0.75 (0.12–4.73)</td>
<td>0.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean value: STJ/AA</td>
<td>0.04 (0.0006–2.13)</td>
<td>0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximal difference: STJ/AA</td>
<td>1.49 (0.17–13.52)</td>
<td>0.72</td>
<td>0.02 (0.007–1.19)</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Factors with P < 0.2 (bold) were included in the multivariate logistic regression model.

In our study, the number of patients with neoaortic insufficiency increased continuously after the first year of follow-up and ultimately reached 49%, and when trivial insufficiency was included, 76% of the patients. Immediately after surgery, it was observed in 22% of the cases, but in the first year of observation the neoaortic incompetence was ameliorated, so at the end of the first year of observation the incidence of NeoAR was 10%. This may be related to transient dilatation of neoaortic root after surgery, as reported in the literature [11]. The highest risk occurs between first and sixth year after the switch procedure, and after this time the incidence of new cases decreased and finally reached a plateau at the level of 2%. After 14 years of follow-up, no new cases of NeoAR were observed. Most of the patients had trivial or mild regurgitation without any implication for the general condition of the patients and did not require any intervention, such as reoperation or even pharmacotherapy. However, only 28% of our patients had no neoaortic function during the course of follow-up. Similar results were previously reported by Marino et al., who in a period of almost 9 years of postoperative monitoring, observed a continuous increase of neoaortic insufficiency that finally included 84% of the patients. In the majority of cases, there was only trivial or mild regurgitation without serious clinical implications. During follow-up, they described an increase of at least one grade in 46% of the study population [4].

There were no differences in the frequency of NeoAR observed in the three analysed groups, which indicates that the heart defects associated with TGA did not exert an impact on neoaortic function after the switch. From the risk factors analysed, only a disproportion between the pulmonary and aortic valves and non-facing commissures, respectively, were confirmed as significant risk factors for NeoAR. This study group was homogeneous in terms of surgical technique, surgeon, diagnostic and pre-, peri- and postoperative care, so these factors were not examined in our study. In fact, the differences between the frequency of neoaortic insufficiency that occur in different centres.
Figure 4: Changes of the average values of AS and STJ to AA ratios during follow-up. Data from Groups I–III in patients with and without significant NeoAR.

Figure 5: Changes of the average values of AS and STJ to body surface area ratios during follow-up. Data from Groups I–III in patients with and without significant NeoAR.
may be related to the surgical technique used, including coronary reimplantation [11]. In our department, all of the operations were performed under the guidance of a single surgeon using a modified trapdoor technique.

We also did not check the impact of two-stage correction or a bicuspid native pulmonary valve because of the small number of patients with it, although these factors were shown to be significant in other studies [14, 15]. Many other risk factors that have been described previously in the literature, coronary anomalies, age at the time of the operation or VSD associated with TGA [10, 12], were not significantly correlated with NeoAR in our study. The lack of significance in the case of VSD is interesting because it is frequently related with pulmonary artery valve (PAV)/AoV discrepancy (Table 1), which is reportedly a significant risk factor for NeoAR. In our series, all of the patients with closed VSD were included in the second group, but in the cases of larger, significant ventricular defects PAV/AoV was frequently present. It may be VSD is not a cause of NeoAR but its haemodynamic implications.

The yearly average value of the calculated indexes was also similar in all of the analysed groups, with no differences in patients with and without signs of NeoAR in the postoperative period. This was quite a surprising result, because root dilatation has been put forward in a number of reports as a possible explanation for the higher incidence of neo-aortic insufficiency in children with TGA after anatomical correction [4–6]. During follow-up, we observed AS growth and development proportional to the AA, while there was impaired growth of the STJ compared with the AA and sinus.

In healthy children, the growth of the AA, sinus and STJ is proportional and the indices of the AS to the AA and STJ to the AA are both constant and independent of the weight and body surface area [16]. The value for the AS to AA ratio in the healthy population was 1.37 (95% CI: 1.18–1.56), while in our study it was 1.28 (95% CI: 1.13–1.45). The decrease in the STJ to AA index during follow-up observed in our study population is not reportedly seen in healthy children. The value in this group was constant—1.11 (95% CI: 0.95–1.28), while in our group it decreased during 18 years of follow-up from 0.99 (95% CI: 0.87–1.15) to 0.89 (95% CI: 0.8–1.1) [16]. The poor growth of the STJ compared with the AS and annulus may be related to the weakened growth potential due to the placement of the suture line and connection of the aorta with the native pulmonary sinus during the anatomical correction of TGA.

Analysing out the study group all together, we discovered the root diameter on average to be 37% bigger than in the healthy population (average z-score = 1.58). This may in part be related to the norms used in calculation for AoV, because the neo-aortic valve is natively the pulmonary valve, which is usually ~20% bigger. The disproportionate increase that was observed over time and at the end of follow-up was such that the neo-aortic root was ~57% bigger than in the healthy population (average z-score = 2.09).

Quite different results were presented by Hutter et al. They reported that in a group of 144 patients, the z-score for neoAA was initially 1.5 and this score did not change during 8.5 years of follow-up [17]. Continuous dilatation of the neo-aortic root was reported by Schwartz et al. In a group of 335 patients, they observed an increasing z-score of the neo-aortic root from 2.6 to 3.6 after 5 years and 4.6 after 10 years of follow-up [18]. A report on the development of the neo-aortic root that is similar to our results was presented by Bové et al. [19]. They also did not find a correlation between the neo-aortic root diameter and NeoAR.

The coincidence of neo-aortic root dilatation with NeoAR without any direct correlation between the root diameter and NeoAR may suggest that the degree of dilatation is not a critical factor related to the impairment of neo-aortic valve function. Non-facing commissures and aortic and pulmonary valve discrepancies are factors strictly related to the root geometry, so other approaches, such as four-dimensional magnetic resonance imaging or non-standard diameter analysis with echocardiography may be needed to find the key-factor that is responsible for the occurrence of more frequent neo-aortic insufficiency presence in the postoperative period.

CONCLUSIONS

NeoAR is common after the switch procedure and increases in prevalence in the first years after surgery. The highest risk occurs between the first and sixth year after surgical correction. However, postsurgical analysis indicates that this complication is rather mild and carries no clinically significant haemodynamic implications.

Non-facing commissures and aortic and pulmonary valve discrepancies are significant risk factors for neo-aortic insufficiency. None of the root diameter data or derived indexes were correlated with the development neo-aortic insufficiency in this series.

The neo-aortic root develops proportionally (excluding worse growth potential of the STJ), however, it is initially bigger than in healthy children and this disproportion increases during follow-up from 137 to 157% of the normal value.

Conflict of interest: none declared.

REFERENCES


Editorial Comment

Re: The neoaortic root in children with transposition of the great arteries after an arterial switch operation

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Keywords: Neoaortic valve • Aortic insufficiency • Root dilatation

The first neonatal arterial switch procedure was performed by Norwood and Castaneda in January 1983 at Children’s Hospital Boston [1]. This was the first ‘elective’ neonatal procedure, and not surprisingly its introduction created a storm of controversy. Not only was it elective in the sense that alternative proven satisfactory operations were available, namely the atrial switch procedures and the two-stage arterial switch with preliminary pulmonary artery banding, but in addition, the rationale for its introduction was more theoretical than based on hard statistical data. The hypothesis at the time was that the right ventricle and tricuspid valve of the individual with D-transposition of the great arteries would not perform as well as systemic pressure in the long-term relative to a left ventricle. Those who vigorously opposed the new operation claimed that it could never be done with a comparable early mortality to the atrial switch procedures. When this prediction proved to be incorrect, opponents continued to suggest that there would be important long-term complications including anastomotic obstruction, particularly of the reconstructed pulmonary arteries and late coronary problems. Interestingly, little attention was paid at the time to the possibility that the ‘neoaortic valve’ might fail, despite the fact that even in the newborn, particularly those with a large ventricular septal defect (VSD), there is often considerable disparity in size between the original large pulmonary valve and the relatively smaller aortic valve.

The first report to examine the fate of the neoaortic valve was published by Hourihan et al. [2] from Children’s Hospital Boston. The authors reviewed serial echocardiograms on 50 patients after the arterial switch procedure, confirming that the pulmonary root (neoaortic root) and the pulmonary annulus (neoaortic annulus) were both larger in infants with D-transposition relative to the aortic root and annulus of control patients. The authors found that there was appropriate growth of the aortic anastomosis, but a surprise finding was that there was progressive dilatation of the neoaortic root, particularly in patients who had a history of pulmonary artery banding and those with neoaortic regurgitation. A subsequent study by Schwartz et al. [3] from Children’s Hospital Boston examining 335 patients after the arterial switch procedure for either D-transposition or double outlet right ventricle reported that although the aortic root was indeed dilated relative to control patients, there did not appear to be progressive dilatation at late follow-up. Furthermore, the incidence of greater than moderate aortic regurgitation was quite small, being 93% at 10 years, and the freedom from any surgery on the neoaortic valve or neoaortic root was 95% at 10 years. Risk factors for moderate aortic regurgitation or greater were a previous pulmonary artery band and older age at the time of the arterial switch procedure, which were both related to the presence of an associated VSD.