Midterm follow-up of arterial switch operation for transposition of the great arteries with intact ventricular septum and left-ventricular outflow tract obstruction

Shahzad G. Raja a,⁎, Martin Kostolny a, Nilesh Oswal b, Ahmed Afifi a, Branko Mimica a, Ian D. Sullivan b, Marc R. de Leval a, Victor T. Tsang a

aDepartment of Paediatric Cardiothoracic Surgery, Great Ormond Street Hospital, London, WC1N 3JH, UK
bDepartment of Paediatric Cardiology, Great Ormond Street Hospital, London, WC1N 3JH, UK

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Abstract

Objective: We report the mid-term follow-up of patients, who underwent arterial switch operation (ASO) for transposition of the great arteries (TGA) with intact ventricular septum and left-ventricular outflow tract obstruction (LVOTO) over a 10-year period from 2000 to 2009.

Methods: Thirteen TGA patients (3.9% of our ASO cohort) with intact ventricular septum and LVOTO underwent ASO. LVOTO was defined as pulmonary valve z-score ≤−2.0 (n = 3) or peak LVOT gradient ≥40 mmHg with (n = 7) or without (n = 3) anatomic subvalvar stenosis on echocardiography. Median age and weight were 14 days (range, 7—130 days) and 3.2 kg (range, 2.1—4.6 kg). The LVOT abnormalities included fibromuscular narrowing (n = 5) and atrioventricular valve-related findings (n = 5). LVOT clearance was achieved by resection of accessory mitral tissue (n = 2) only.

Results: Follow-up was 100% complete. There were no early or late deaths. Freedom from re-operation for neo-aortic valve regurgitation and/or LVOTO was 100% at a median follow-up of 38 months (range, 6—115 months). All patients had functional status appropriate for their age. Three patients had mild aortic regurgitation. The median Doppler estimated LVOT systolic gradient was 12 mmHg (range, 0—18 mmHg) for the entire cohort at the latest follow-up.

Conclusions: Mid-term outcomes of ASO for a highly selected group of patients with pulmonary valve annulus z-score ≤−2.0, resectable organic LVOTO, and dynamic peak LVOT gradient ≥40 mmHg remain satisfactory, with a need for long-term follow-up.

⁎⁎ Corresponding author. Tel.: +44 2074059200; fax: +44 2078138379.
E-mail address: drrajashahzad@hotmail.com (S.G. Raja).

Keywords: Transposition of the great arteries; Arterial switch operation; Left-ventricular outflow tract obstruction; Intact ventricular septum

1. Introduction

Left-ventricular outflow tract obstruction (LVOTO) in the presence of concordant ventriculo-arterial connections produces an impediment to the flow of blood between the left ventricle and the aorta. When the ventriculo-arterial connections are discordant, (transposition of great arteries, TGA), it is the channel between the left ventricle and the pulmonary trunk which is impeded, but by the same individual obstructive lesions [1]. Although anatomic LVOTO is more commonly seen in the presence of a ventricular septal defect (VSD), it is an infrequent association of TGA with intact ventricular septum (IVS) [2—4]. Thus, the restriction to flow can be localised at valvar or subvalvar levels. Valvar restriction is quite rare, and the valve can be dysplastic, bicuspid, and stenotic. The subvalvar lesions can be a fibrous shelf or fibromuscular tunnel, tissue tags, and anomalous attachment of the atrioventricular valvar tension apparatus [1].

Obstruction to the LVOT is a complex entity in the setting of discordant ventriculo-arterial connections, the treatment of which has evolved markedly in the past couple of decades. Biventricular reconstruction, especially in the presence of IVS, can still represent a great surgical challenge. In recent years, arterial switch operation (ASO) has become the most favoured surgical option [2,3], but its feasibility needs to be thoroughly assessed after evaluation of the coronary arterial patterns, along with the anatomic characteristics of the obstructed LVOT. Special attention should be given to the size of the ventriculo-arterial junction, the nature of the obstruction, and its potential respectability [1]. Presence of LVOTO was considered to be a contraindication for ASO in the past [5,6]. However, in recent years, good outcomes have been reported following ASO for this morphologic entity [2,3]. When considering ASO in the setting of TGA/IVS/
LVOTO, the decision to proceed is influenced not only by the anatomy, but also by the physiology of the LVOT flow dynamics. In the context of TGA with high total pulmonary blood flow, ASO results in reduced LV stroke volume and relief of right-ventricular pressure load. This may be associated with resolution of the previous dynamic LVOTO because of septal shifting, without the need for direct surgical procedures on the outflow tract.

There is a paucity of publications reporting mid- and long-term outcomes after ASO for TGA/IVS/LVOTO [2,3,7]. The purpose of this study was to review our experience with this subset of patients with particular reference to mid-term outcomes.

2. Methods

2.1. Patients

Thirteen patients with TGA and IVS, who underwent an ASO between January 2000 and December 2009, were studied (Table 1 and Fig. 1). This continuous series without omissions formed 3.9% of our ASO cohort of 332 patients. Preoperative and postoperative echocardiograms, catheterisation studies, operative notes, and follow-up clinic files were evaluated. From the preoperative echocardiogram, the aetiology of the LVOTO, pulmonary valve z-score, peak instantaneous LVOT gradient, and associated cardiac anomalies were noted. The aetiology of the LVOT on preoperative echocardiogram was assessed by examining the anatomic structure of the LVOT and the flow acceleration pattern by colour Doppler. Valvar obstruction was defined as flow acceleration at this level and valvar annular z-score ≤ -2.0. Obstruction was classified as subvalvar fibromuscular narrowing if there was flow acceleration and anatomic narrowing in the subvalvular region and a peak LVOT Doppler-derived gradient of ≥ 40 mmHg, as assessed by the simplified Bernoulli equation. LVOTO attributable to abnormal or accessory atrioventricular valvar tissue or attachments, or systolic anterior motion of the mitral valve (SAM) without abnormal septal attachment, was classified as atrioventricular valvar-related findings (Fig. 2(a)–(d)).

2.2. Surgical technique

Surgery was performed according to previously described standard operative technique [8]. Transthoracic echocardiography was performed in all patients before and after surgical correction. On transthoracic echocardiography, pulmonary

![Fig. 1. Indications of arterial switch operation for transposition of great arteries (TGA) Excluding Taussig Bing anomaly and ccTGA IAA = interrupted aortic arch; IVS = intact ventricular septum; LVOTO = left ventricular outflow tract obstruction; VSD = ventricular septal defect.](image1)

![Fig. 2. Aetiology of left ventricular outflow tract obstruction in patients with transposition of the great arteries and intact ventricular septum.](image2)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Preoperative</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, days, median (IQR)</td>
<td>14 (7–130)</td>
<td>10 (6–115)</td>
</tr>
<tr>
<td>Weight, kg, median (IQR)</td>
<td>3.2 (2.1–4.6)</td>
<td>3.2 (2.1–4.6)</td>
</tr>
<tr>
<td>Female</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>LVOT gradient, mean ± SD</td>
<td>45.6 ± 14.5</td>
<td>11.5 ± 6.5</td>
</tr>
<tr>
<td>LVOT gradient, median (IQR)</td>
<td>60 (24–90)</td>
<td>12 (0–18)</td>
</tr>
</tbody>
</table>

LVOTO aetiology

- Valvar
- PV z-score, median (range) -2.06 (-2.0–2.13)
- PV z-score, mean ± SD -0.78 ± 0.96
- Thickened leaflet 3
- Bicuspid 1
- Tricuspid 1
- Quadricuspid 1

- Subvalvar
  - Fibromuscular narrowing 5

AV valve-related

- Accessory septal attachment 2
- Dynamic (SAM) 3

- Follow-up duration, median (IQR) 38 (6–115) months
- LVOT gradient, mean ± SD 12 (0–18) mmHg
- LVOT gradient, median (IQR) 0.23 ± 0.17
- PV z-score, mean ± SD -0.78 ± 0.96

- Aortic regurgitation
  - Nil 10
  - Mild 3
  - LVOT reintervention 0

* IQR = interquartile range; SD = standard deviation; LVOT = left ventricular outflow tract obstruction; PV = pulmonary (neo-aortic) valve; AV = atrioventricular valve; SAM = systolic anterior motion.
* p = 0.0001 compared to preoperative gradient.
* p = 0.0018 compared to preoperative z-score.
valve diameter, leaflet pliability and thickness, and the
anatomic basis of the subvalvar obstruction when present,
were assessed. The pulmonary valve diameter was compared
with the predicted diameter of normal aortic valve and the z-
score was calculated [9]. An ASO consisted of pulmonary
artery mobilisation up to lobar branches, ligament/ductus
division, and marking sutures on the pulmonary trunk at the
proposed site of coronary transfer. After cardioplegic arrest
was achieved, the aorta was transected distally and the
pulmonary artery proximally. Clearance of the L VOT was then
undertaken through the pulmonary (neo-aortic) valve when
necessary. The pulmonary valve was inspected, and if finally
deemed usable as the future aortic valve, coronary button
excision and transfer was undertaken followed by the
Lecompte manoeuvre and reconstruction of the ascending
aorta. The aortic clamp was removed after deairing the left
side of the heart. Pulmonary artery continuity was re-
established.

2.3. Follow-up

All patients had a predischarge transthoracic echocardiogram. Subsequently, they were followed up in the outpatient clinic at periodic intervals. At each visit, a detailed clinical and echocardiographic evaluation was performed.

2.4. Data collection and statistical analysis

The data are reported as mean ± standard deviation or median (range). Comparison of pre- and post-ASO Doppler-derived L VOT gradient and z-score was performed using a paired t-test. Statistical analysis was performed with Statistical Package for Social Sciences (SPSS) 11.0 for Windows (SPSS Inc. Chicago, IL, USA). Values of p less than 0.05 were considered statistically significant.

Our institutional ethics committee approved this study, and informed consent was waived due to its retrospective nature. The study was registered with the Research and Development Department, Institute of Child Health, University College London.

3. Results

3.1. Patient characteristics

The study group comprised of three patients with pulmonary valve z-score ≤ −2.0 and 10 patients with peak L VOT Doppler gradient >40 mmHg. The group with L VOT gradient ≥40 mmHg included patients with (n = 7) or without (n = 3) anatomic subvalvar stenosis on echocardiography. Median age and weight were 14 days (range, 7–130 days) and 3.2 kg (range, 2.1–4.6 kg). The L VOT abnormalities included accessory mitral valve septal attachment (n = 2), systolic anterior motion of the septal mitral leaflet (n = 3), and subvalvar fibromuscular narrowing (n = 5) (Table 1).

3.2. Position of great arteries and coronary anatomy

The aorta was right anterior to the pulmonary artery in 11 patients, side by side in one, and directly anteroposterior in

one. The coronary anatomy was classified according to the
Leiden classification. 1LCx-2R was found in 10 patients, 1L-
2CxR was observed in two patients, and 1RL-2Cx in one
patient.

3.3. Early outcomes

Resection of accessory mitral tissue was undertaken in two
patients only to achieve LVOT clearance. No intervention was
required on the LVOT for the remaining eight patients with
subvalvar LVOTO or the three pulmonary (neo-aortic) valves
with z score ≤ −2.0. There were no early deaths. One patient
required extracorporeal membrane oxygenation following
relief of cardiac tamponade. Delayed chest closure was
performed in the same patient. The median duration of
invasive ventilation was 4 days (range, 2–11 days). Median
length of intensive care unit stay was 6 days (range, 4–
13 days) with 11 days (range, 7–19 days) as median duration
of hospital stay.

3.4. Recent clinical status

Follow-up was 100% complete. At median follow-up of 38
months (range, 6–115 months), there were no late deaths.
All patients were asymptomatic.

3.5. Progression of LVOT gradient and neo-aortic valve regurgitation

Table 1 shows the mean and median peak LVOT gradients
and number of patients with aortic regurgitation (AR) at
recent follow-up. There was a statistically significant
reduction in peak Doppler-derived LVOT gradient at midterm
follow-up (p = 0.0001) (Table 1 and Fig. 3). In two of the
patients, who had mild AR at discharge, the regurgitation
has not progressed after a follow-up of 4 and 9 years,
respectively. One patient with dysplastic quadricuspid
pulmonary valve developed mild AR, which was recognised
6 weeks after the operation and has remained static after a
follow-up of 7 years. Freedom from re-operation for

![Fig. 3. Effect of arterial switch operation on Doppler estimated left ventricular outflow tract pressure gradients. (Valvar left ventricular outflow tract obstruction (LVOTO) = Patients 1, 2 and 11; LVOTO due to accessory mitral tissue = Patients 7 and 8; LVOTO due to systolic anterior motion = 9, 10 and 12; LVOTO due to fibromuscular narrowing = Patients 3–6 and 13).](image-url)
neo-aortic valve regurgitation and/or LVOTO was 100% at last follow-up.

3.6. Progression of pulmonary (neo-aortic) valve $z$-score

Median pulmonary (neo-aortic) valve $z$-score pre-ASO was −0.41 (range, −2.13 to 0.65), whereas the median $z$-score post-ASO was 0.2 (range, 0.04–0.72) (Table 2). There was a significant reduction in mean pulmonary (neo-aortic) valve $z$-score at the latest follow-up ($p = 0.0018$) (Table 1 and Fig. 4).

4. Discussion

This study reports favourable mid-term outcomes for patients with TGA/IVS/LVOTO, who underwent ASO. When ASO for TGA/IVS was introduced in the early to mid-1980s, surgical mortality was high. Abnormalities of the LVOT, whether at pulmonary valve or subvalvar level, were considered a contraindication to ASO. A Senning operation, albeit with persisting pulmonary outflow tract obstruction, was considered a better alternative. Sometimes, the Senning procedure was combined with an extracardiac conduit to connect the LV apex to the left pulmonary artery to decompress the LV [10].

As experience and confidence with ASO for TGA/IVS increased, so did the acceptance of marginal cases. This is illustrated by the present series, in which only a single Senning operation with insertion of an extracardiac conduit for TGA/IVS/LVOTO was performed in our unit over the course of the decade (2000–2009). It could be argued that an ASO might have been preferable to a Senning procedure even in this infant.

One of the concerns with performing ASO for patients with TGA/IVS/LVOTO is the development of neo-AR over time [3,7]. LVOTO has been shown to be a risk factor for development of significant regurgitation [11]. In our series, three patients developed neo-AR, which remains mild at mid-term follow-up. Two of these patients had resection of accessory mitral tissue through the pulmonary valve. The most logical explanation for development of neo-AR in these two patients may be surgical manipulation through the neo-aortic valve leading to microscopic damage of the neo-aortic leaflets during operation. The third patient had a dysplastic yet competent quadricuspid valve at the time of ASO.

At present, there is limited information available regarding the fate of the smallish dysplastic pulmonary valve after ASO [2,12,13]. In our series, three patients with pulmonary valve $z$-score $\leq -2.0$ at the latest follow-up have remained free from re-intervention, although one patient with quadricuspid valve has developed mild regurgitation.

One of three patients with pulmonary valve $z$-score $\leq -2.0$ has a bicuspid valve. The presence of a bicuspid aortic valve is associated with aortic root dilatation, annuloaortic ectasia, and aortic dissection [13,14]. It is more susceptible to fibrosis and calcification, leading to aortic stenosis and mixed aortic valve disease in otherwise normal hearts [15]. A bicuspid aortic valve is more prone to degenerative changes in its native position. Hence, there has been concern about the long-term performance of a bicuspid aortic valve used in the systemic circulation (arterial switch, Damus Kaye Stansel, and Norwood operations) [12].

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Table 2. Pre- and post-ASO pulmonary (neo-aortic) valve dimensions and $z$-scores.

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Sex</th>
<th>Age at operation (days)</th>
<th>BSA (m$^2$)</th>
<th>PV diameter $^*$ (mm)</th>
<th>$z$-Score</th>
<th>Age at follow-up</th>
<th>BSA (months)</th>
<th>PV diameter $^*$ (mm)</th>
<th>$z$-Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>14</td>
<td>0.17</td>
<td>5.2</td>
<td>−0.06</td>
<td>50</td>
<td>0.62</td>
<td>13.0</td>
<td>0.35</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>7</td>
<td>0.21</td>
<td>5.3</td>
<td>−2.0</td>
<td>84</td>
<td>1.05</td>
<td>16.2</td>
<td>0.2</td>
</tr>
<tr>
<td>3$^\dagger$</td>
<td>M</td>
<td>130</td>
<td>0.27</td>
<td>6.5</td>
<td>−1.33</td>
<td>6</td>
<td>0.35</td>
<td>9.8</td>
<td>0.2</td>
</tr>
<tr>
<td>4$^\dagger$</td>
<td>M</td>
<td>14</td>
<td>0.20</td>
<td>7.0</td>
<td>−0.41</td>
<td>7</td>
<td>0.37</td>
<td>10.0</td>
<td>0.13</td>
</tr>
<tr>
<td>5$^\dagger$</td>
<td>M</td>
<td>7</td>
<td>0.20</td>
<td>7.2</td>
<td>−0.1</td>
<td>6</td>
<td>0.23</td>
<td>8.0</td>
<td>0.26</td>
</tr>
<tr>
<td>6$^\dagger$</td>
<td>M</td>
<td>28</td>
<td>0.18</td>
<td>6.8</td>
<td>−0.11</td>
<td>38</td>
<td>0.66</td>
<td>13.2</td>
<td>0.08</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>11</td>
<td>0.18</td>
<td>7.0</td>
<td>0.2</td>
<td>28</td>
<td>0.56</td>
<td>12.4</td>
<td>0.22</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>13</td>
<td>0.24</td>
<td>8.8</td>
<td>0.65</td>
<td>27</td>
<td>0.51</td>
<td>11.5</td>
<td>0.01</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>13</td>
<td>0.24</td>
<td>8.8</td>
<td>0.65</td>
<td>27</td>
<td>0.51</td>
<td>11.5</td>
<td>0.01</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>12</td>
<td>0.16</td>
<td>6.2</td>
<td>−0.31</td>
<td>6</td>
<td>0.19</td>
<td>7.2</td>
<td>0.17</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>23</td>
<td>0.21</td>
<td>7.2</td>
<td>−0.19</td>
<td>115</td>
<td>0.84</td>
<td>14.3</td>
<td>0.11</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>7</td>
<td>0.21</td>
<td>5.2</td>
<td>−2.13</td>
<td>96</td>
<td>0.75</td>
<td>13.8</td>
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</tr>
<tr>
<td>13$^\dagger$</td>
<td>F</td>
<td>15</td>
<td>0.23</td>
<td>7.2</td>
<td>−1.04</td>
<td>97</td>
<td>0.71</td>
<td>13.3</td>
<td>0.2</td>
</tr>
</tbody>
</table>

$^*$ Neo-aortic valve. ASO = arterial switch operation; BSA = body surface area; F = female; LVOT = left ventricular outflow tract; M = male; PV = pulmonary (neo-aortic) valve. $^\dagger$ Pre-ASO LVOT dimensions for patients 3–6 and 13 with fibromucular narrowing (mm) ($z$-score) = 5.1 [−2.73], 5.2 [−2.18], 5.0 [−2.18], 4.9 [−2.17], 5.4 [−2.75]. $^\ddagger$ Post-ASO LVOT dimensions for patients 3–6 and 13 with fibromucular narrowing (mm) = 7.8 [−1.25], 7.2 [−1.84], 6.1 [−1.48], 9.9 [−1.89], 11.0 [−1.61].

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Fig. 4. Evolution of pulmonary (neo-aortic) valve $z$-score after arterial switch operation. (Valvar left ventricular outflow tract obstruction [LVOTO] = Patients 1, 2 and 11; LVOTO due to accessory mitral tissue = Patients 7 and 8; LVOTO due to systolic anterior motion = 9, 10 and 12; LVOTO due to fibromucular narrowing = Patients 3–6 and 13).
A bicuspid aortic valve is not generally considered a contraindication to the ASO; however, there are still limited data on its medium-term to long-term function in the neo-aortic position [13,14].

As acquired haemodynamic abnormalities in the setting of a dysplastic valve in an otherwise normal heart may develop, it is obviously necessary to continue to investigate these patients over a much longer term. However, as such, abnormalities do not usually become of major clinical importance; the presence of a dysplastic bicuspid pulmonary valve should not preclude ASO. On the other hand, a unicommissural pulmonary valve or a severely dysplastic pulmonary valve with z-score < −4.0 in isolation or in addition to multilevel obstruction would preclude ASO in our opinion and practice. Interestingly, we did not come across this subset of patients in our study population.

Similarly, patients with fibromuscular narrowing and smallish LVOT have shown a significant reduction in the gradients during the follow-up period without the need for any further intervention. We refrain from addressing this surgical repair strategy should not just be based on the presence of a higher gradient. The gradient per se may not be a good indicator for decision making as, quite often, a dynamic component to the gradient exists that will usually resolve after the ASO when the right ventricle to left ventricle pressure ratio is normalised [16]. On the other hand, an LVOT z-score < −4.0 or presence of multilevel obstruction may necessitate resection of obstruction or resorting to another surgical repair strategy.

Consequently, decision making in favour of an ASO now seems relatively straightforward for this rare group of patients. The alternative of a compromised Senning procedure, in an era when most surgeons will have little experience of this procedure, or a single-ventricle strategy, which would require a neonatal Damus type procedure with systemic to pulmonary shunt, is not attractive. Surgical decision making may be more difficult in the larger group of infants with TGA/VSD/LVOTO, in whom the Rastelli type of approach may provide a satisfactory alternative. However, even in these patients, the emphasis has shifted in favour of an ASO approach over the past 20 years.

The major limitations of this study include its retrospective design, small sample size, and limited duration of follow-up.

In summary, our results in the mid-term support the notion that the ASO remains a good option for a highly selected group of patients with pulmonary valve annulus z-score ≤ −2.0 ≥ −4.0, resectable organic LVOTO and dynamic peak LVOT gradient ≥40 mmHg. However, it is necessary to continue to investigate these patients to verify the long-term safety and efficacy of ASO in the setting of TGA/IVS/LVOTO.

References


Appendix A. Conference discussion

Dr C. Brizard (Victoria, Australia): I have a very few brief technical questions. I think the essence of your paper relies on the difference between a dynamic obstruction and an organic obstruction. So could you define how you would categorize the two on inspection? How would you define on inspection a dynamic obstruction?

Dr Raja: I think that dynamic obstruction on direct inspection at the time of operation is a very subjective assessment. It is eyeballing of the left ventricular outflow tract. As I mentioned, in patients who have got fibromuscular narrowing, we use a Hegar dilator to assess whether there is an obstruction. Also for dynamic, I think we relied more on the echocardiographic assessment prior to surgery. Although I would admit that, yes, there is an element of septal hypertrophy occasionally, or septal bulge associated with this dynamic obstruction. But we did not undertake any intervention on patients who had an element of septal bulge or septal hypertrophy in the setting of dynamic left ventricular outflow tract obstruction.
And I think it is very difficult to categorize patients into pure dynamic and pure mechanical or fixed obstruction.

**Dr Brizard:** So would you say that the dynamic obstruction has a normal-looking appearance on inspection at the time of surgery?

**Dr Raja:** I would not go to the extent of saying it is normal-looking. I think what I would suggest here is that it is important to correlate the echocardiographic findings with your intraoperative findings. Because, as I said earlier, patients with dynamic obstruction do not have pristine left ventricular outflow tracts. Yes, there is an element of high pulmonary blood flow which would contribute to a left ventricular outflow tract obstruction, but a lot of these patients do have some element of septal bulge or septal hypertrophy. So distinction between the two can become a bit vague for this subgroup of patients.

**Dr Brizard:** Now, you have approached that quickly during your presentation. Upon inspection, if you had a finding that would preclude an arterial switch, what would be your bail-out procedure? You have quoted Senning plus LV-PA conduit. There are other options. Have you considered them?

**Dr Raja:** I would take this opportunity to highlight that this is a very highly selective group of patients.

**Dr Brizard:** Very much, yes.

**Dr Raja:** And I think that as an institution, we have a policy that we would accept a suboptimal arterial switch as a better option than any of the other bail-out procedures which, both in the short- as well as the long-term, do not have as good results as an arterial switch operation.

So I think we, in our experience, have not come across a situation where we have had to make that decision on table intraoperatively. Hence I would not be in a position to comment on what would be a bail-out strategy for patients who have got intraoperative findings which we were not aware of preoperatively.

**Dr Brizard:** In other words, a similar strategy to the one used for interrupted aortic arch with subaortic obstruction?

**Dr Raja:** I think that is a different subgroup of patients, and I think in that case we may have to tailor our strategy according to the substrate.