The Taussig-Bing anomaly: long-term results†

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Abstract

OBJECTIVES: The arterial switch operation (ASO) is the method of choice for the Taussig-Bing heart. The aim of the study was to analyse the long-term outcome of correction of the Taussig-Bing heart.

METHODS: Between 1986 and 2011, 44 infants, including 18 newborns, underwent an ASO. The staged and the primary approach were used in 9 and 35 patients, respectively. Aortic arch (AA) obstruction (n = 26) and right ventricle outflow tract obstruction (n = 34) were common. The mean age at corrective surgery was 112.9 days; the mean weight was 4.17 kg.

RESULTS: There were 1 early and 4 late deaths. Overall survival was 88% at 15 years, with a mean follow-up of 9.2 years. Freedom from reoperation was 67% at 15 years of follow-up. Eight and 6 patients required right and left ventricular outflow tract surgery, respectively, including resection of the right ventricular outflow tract obstruction (n = 8), a transanular patch (n = 6), aortic valve reconstruction (n = 3), aortic valve replacement (n = 2) and AA reoperation (n = 4). Freedom from aortic regurgitation >mild or aortic valve replacement/reconstruction was 76% at 15 years of follow-up. Freedom from any event was 56% at 15 years of follow-up. All patients are in sinus rhythm, and biventricular function is well-preserved in 95% of patients. All patients are doing well; 86% of them are without medication.

CONCLUSIONS: Corrective surgery offers excellent survival benefits and encouraging long-term functional outcomes, regardless of the coronary anatomy and associated lesions. Normal biventricular function is preserved in the vast majority of patients, and >3/4 of patients are without cardiac medication. Nevertheless, TBH associated with a complex anatomy continues to be a risk factor for long-term morbidity, and redos and reinterventions are equally common on both outflow tracts. Progressive neoaortic regurgitation and neoaortic root dilatation might be a problem in the future; therefore, close lifelong surveillance of patients is necessary.

Keywords: Taussig-Bing anomaly • Transposition of the great arteries • Arterial switch operation • Left ventricular outflow tract obstruction • Right ventricular outflow tract obstruction

INTRODUCTION

The Taussig-Bing heart (TBH) represents a complex subset of double outlet right ventricle. The aorta originates entirely from the right ventricle (RV), the pulmonary artery (PA) arises from above the non-restrictive ventricular septal defect (VSD) and there is no pulmonary-mitral fibrous continuity [1]. It is frequently associated with subaortic right ventricular outflow tract obstruction (RVOTO) and aortic arch obstruction (AAO).

The arterial switch operation (ASO) is the method of choice for the TBH, preferably performed early in life, using a primary one-stage approach. Despite improved early results, the incidence of RVOTO/LVOTO and neoaortic regurgitation (AR) are notable and reinterventions and reoperations are required [2–5].

INTRODUCTION

The aim of the study was to analyse the long-term outcome of correction of the TBH.

PATIENTS AND METHODS

Method

A retrospective chart review was undertaken to identify all patients with TBH in whom the single-stage or two-stage correction had been performed.

Patients

Between May 1986 and December 2011, 44 consecutive patients with TBH underwent an ASO at our institution. Previous publications included the data of 34 of our patients [2, 6, 7]. There were
27 male patients (61%). The mean age at the time of surgery was 112 ± 240 days, with a mean weight of 4.17 ± 2 kg. Associated lesions were present in 91% of all 44 patients. RVOTO (n = 34; 77%) and AAO (n = 26; 59%) were common. The most prevalent coronary pattern was 1LAD, Cx-2R (17 of 44 patients; 39%), followed by 1R-2LAD, Cx (9/44; 20%), 2R, LAD, Cx (8/44; 18%), 1LAD-2R, Cx (6/44; 14%) and 1R, LAD-2Cx (4/44; 9%). There were two intramural coronaries. Table 1 details the patients’ characteristics.

### Treatment management

In the past, 9 patients (20%) had undergone two-stage correction with banding of the main pulmonary trunk (PAB) and/or reconstruction of the aortic arch (AA) followed by correction. Primary one-stage surgery was performed in the remaining 35 patients (80%), including 16 newborns (36%). A detailed description of our technique of ASO and reconstruction of the AA has been described elsewhere [2, 6–8]. The approach to closure of VSD has been changed. Currently, the typical subpulmonary VSD is closed with a patch, working through the tricuspid valve, while the prominent conal septum is transected to release the subaortic obstruction of the RVOT and to get a better approach to the anterior-lateral rim of the VSD. Rarely, a tranventricular approach is used and the trans-pulmonary approach was completely abandoned.

### Functional outcome and follow-up

The patients underwent clinical assessment, an echocardiography (ECHO), and in selected cases angiography and magnetic resonance imaging were also performed. Continuous-wave Doppler analysis was utilized to measure the maximum velocities across the outflow tracts. RVOTO/LVOTO/coarctation of the aorta (CoA) was graded as mild (<30 mmHg), moderate (<60 mmHg) and severe (>60 mmHg). Colour-flow Doppler imaging was used to analyse the degree of AR by assessing the proximal regurgitation jet width and retrograde flow in the descending aorta. AR was graded as none to trivial, mild, moderate or severe. The diameter of the aortic root was measured at the level of the sinus of Valsalva. Dilatation of the aortic root was evaluated with an aortic root z-score using the Boston data-based formula [9]. Early death was defined as death in the hospital or within 30 days after the operation.

The follow-up data were complete in all of the surviving patients. Between February and May 2012, data were collected on morbidity, including neurological outcome, ECHO, ECG, New York Heart Association (NYHA) classification and actual medication, by using a questionnaire, as well as by direct contact with the referring cardiologists. For 4 patients, the data were >3 years old.

### Statistical analysis

Kaplan–Meier curves (GraphPad Prism version 2.0 10855; San Diego, CA, USA) were calculated for actuarial survival, freedom from reintervention and reoperation, freedom from post-operative RVOTO or aortic valve reoperation. Means were given with standard deviation and percentages with a 95% confidence interval (CI). The log-rank test was used to compare variables; a P-value of <0.05 was statistically significant.

### RESULTS

#### Survival

Considering both early and late events, the probability of survival from the time of the corrective operation was 88.3% (95% CI: 74.1–94.9%) at 15 years of follow-up (Fig. 1). There were 1 early and 4 late deaths. Early death occurred in a patient with an intramural course and stenosis of the right coronary artery, RVOTO with hypoplastic AA and multiple VSDs. Four days after correction, relocation of both coronaries was required due to persistent coronary ischaemia. The patient died on the 10th postoperative day on extracorporeal membrane oxygenation due to multiorgan failure.

Another patient, who suffered from a residual gradient in the AA and underwent reoperation on the 9th postoperative day, died on the 49th postoperative day due to chronic renal failure. Two other patients perished suddenly, most likely due to arrhythmias and/or ischaemia. The first patient had a monocoronary ostium (2R, L), RVOTO and CoA. The patient died 66

![Figure 1: Kaplan–Meier estimate of survival for 44 patients with TBA after ASO. Vertical bars represent standard errors.](image-url)
months after the operation, despite the fact that the ECHO at the last check-up showed good biventricular function, moderate AR and no outflow tract obstruction. The other patient died 46 months after surgery. He had undergone two-stage repair. Postoperatively, he suffered from atrial arrhythmias, which required medical treatment. The ECHO at the last follow-up showed moderate RVOTO, moderate MR and AR with mild LV dilatation. There was 1 non-cardiac-related death in a patient who died of sepsicaemia 8 months after surgery. The follow-up interval was 109.9 ± 75.3 months.

There was no statistical significance in the survival rates of patients who underwent corrective surgery in different eras (P = 0.26).

Reoperation and reintervention

Freedom from reoperation was 67.3% (95% CI: 43.0–81.2%) at 15 years of follow-up (Fig. 2). The mean interval free from reoperation after primary correction was 51.2 ± 78.1 months. Associated lesions were no predictor for reoperation on LVOT (P = 0.45) or RVOT (P = 0.49). Of the 44 patients, 13 underwent 17 reoperations and 2 were reoperated three times. The type and distribution of reoperation are given in Table 2.

**LVOT surgery.** Six patients required eight reoperations on the LVOT/AA, including reconstruction of the aortic valve (3 patients), a Konno operation (1 patient), mechanical aortic valve replacement (1 patient) and reconstruction of the AA (4 patients). Freedom from LVOTO-reoperation was 78.0% (95% CI: 70.9–85.0%) after 15 years of follow-up (Fig. 3).

In 2 patients who underwent reconstruction of the aortic valve, intimate contact of the VSD-patch caused immobilization of the leaflets and prolapse, with subsequent progressive aortic regurgitation. In another patient, secondary damage of the leaflets with prolapse and root dilatation due to turbulence of flow in the LVOT was seen. Reconstruction of the valve included shaving of the leaflets, asymmetric reduction plasty of the aortic valve annulus using sub-commissural stitches and triangular resection plasty of the prolapsed leaflet. Percardial extension patches were used in 2 patients. There was one failure 17 months after reconstruction of the aortic valve in a patient who had undergone multiple redos (3x) on the RVOT and also on the LVOT. This patient finally underwent a Konno operation with a mechanical valve, RV-PA conduit placement and patch plasty of the right pulmonary artery (RPA).

Another patient underwent mechanical valve replacement for severe AR 111 months after the operation. Freedom from aortic valve surgery was 83.4% (95% CI: 60.9–93.6%) after 15 years of follow-up, respectively.

Re-reconstruction of the AA was required in 4 cases; 1 of these patients underwent two-stage repair. Initially, all 4 patients presented with a hypoplastic AA, CoA or interruption of the AA. The rate of redo of primary arch repair was 14% (4/29).

**RVOT surgery.** Of 34 patients who presented with RVOTO at the time of surgery, 5 (15%) patients underwent reoperation for re-RVOTO. Three additional patients (9%) were reoperated on both the RVOT and LVOT. Freedom from RVOTO reoperation was 80.3% (95% CI: 59.6–91.1%) after 15 years of follow-up (Fig. 4). Two patients underwent redo for RVOTO three times.

Resection of the subvalvular RVOTO was the most common procedure, which was carried out in 8 cases. Other reoperations on the RVOT included a transanular patch (6 patients), RV-PA conduit (3 patients), reconstruction of the pulmonary valve (2 patients), patch plasty of the main pulmonary trunk (2 patients) or peripheral pulmonary arteries (2 patients). Two patients with a transanular patch needed re-redo for re-RVOTO.

**Reinterventions.** Freedom from any event was 56.4% (95% CI: 33.4–72.2%) at 15 years of follow-up (Fig. 5).

Eight patients underwent percutaneous interventional procedures: dilation or stenting of a re-CoA was carried out in 6 cases and dilation of a re-RVOTO/stenosis of the main or peripheral pulmonary arteries in 5. The majority of reinterventions were carried out on the 2 patients who had multiple redos.

Functional outcomes

**Left ventricular outflow tract.** At the last follow-up, 7 patients (7/39; 18%) showed mild acceleration of flow in the LVOT. Two patients had mild/moderate aortic valve stenosis. Mild re-CoA

<table>
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<th>Table 2: Characteristics of reoperations</th>
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<td><strong>Surgery on the RVOTO</strong></td>
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<td>Transanular Patch</td>
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<td>Patch plasty of the PA, LPA, RPA</td>
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<td><strong>Surgery on the LVOTO</strong></td>
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<td>Konno operation</td>
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<td>Others</td>
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LPA: left pulmonary artery; LVOTO: left ventricular outflow tract obstruction; PA: pulmonary trunk; RPA: right pulmonary artery; RVOTO: subaortic right ventricular outflow tract obstruction.
was seen in 5 (13%) patients. All 5 patients underwent primary AA surgery, 2 were reoperated for recurrent CoA, and 2 others underwent ballooning of the AA.

At the last follow-up, of 37 patients with a native aortic valve, 16 (43%) had none/trivial regurgitation of the neoaortic valve. AR was mild in 19 (51%), moderate in 3 (8%) and severe in 1 patient (3%). Two patients had a mechanical aortic valve.

Freedom from aortic regurgitation > moderate or aortic valve replacement/or reconstruction was 76.1% (95% CI: 63.5–88.6%) after 15 years of follow-up.

An aortic root diameter >3Z was found in 6 patients (15%), with a mean diameter of 33.8 ± 5.9 mm. There was severe aortic regurgitation in only 1 patient; the remaining patients had no or mild AR. No predictors of aortic regurgitation or root dilatation (age at time of surgery, AAO, staged approach, PAB, need for Lecompte manoeuvre or access of the VSD through the neoaortic root) were identified. In particular, the use of the trap-door technique was no statistically significant risk factor for aortic root dilatation (P = 0.61).

Echocardiography showed good left ventricular function in all patients and a mildly impaired function in 1. The dimensions of the LV were within a normal range in all patients but 2, (37/39, 95%) who showed a mildly enlarged LV. One patient had a mild form of myocardial hypertrophy after replacement of a mechanical aortic valve.

Right ventricular outflow tract. Mild, moderate and severe RVOTO was present in 11 (28%), 6 (15%) and 1 of 39 cases (3%), respectively. Nevertheless, systolic pressure in the RV ≥ ½ of systemic pressure was seen in only 3 patients (3/39, 8%).

Fourteen patients (78%) underwent resection of the RVOTO during primary repair. Postoperative mild or moderate RVOTO was a risk factor for RVOTO redo (P = 0.01).

Neo-pulmonary valve regurgitation was mild in 9 patients (23%) and moderate in 7 (18%). Progressive regurgitation was associated with transanular patch enlargement of the RVOT. Only 1 patient, who underwent reoperation with a RV-PA conduit, showed mildly impaired RV function; 4 others had a mildly dilated RV.

Tricuspid and mitral valve. Seven patients (18%) showed mild mitral valve regurgitation (MR); mild tricuspid valve regurgitation (TR) was present in 18 (46%) and moderate TR in 2 patients (5%).

Residual VSDs. Restrictive residual VSDs were seen in 3 patients (8%).

Arrhythmias

At the time of the last follow-up all patients were in sinus rhythm (SR), but there were 2 who showed a stable atrial rhythm (2/39, 5%). The patient with postoperative complete atroventricular block was also in SR. No arrhythmia of a higher degree was reported and none of the patients was on antiarrhythmic agents.

Clinical outcomes

Growth was adequate in all cases and all patients are doing well. Seventy-nine percent of the patients presented with NYHA I, and 8 patients with NYHA II (21%). Eighty-five percent of the patients were not taking any cardiac medication. Six patients (15%) were on cardiac medication: all of them received angiotensin-converting-enzyme inhibitor; 1 of them was being treated with β-blockers, and in addition, with calcium channel
DISCUSSION

ASO with VSD closure and corrections of associated lesions is the method of choice for the repair of a TBH. Single-stage repair has shown excellent early and mid-term outcomes [3, 4, 6, 7] and has also become the preferable management choice at our centre. During the last 15 years, the vast majority of our patients underwent single-stage repair and >1/3 of them underwent correction within the first month of life. Two-stage repair might still be considered in a complex anatomy; prematurity or preoperative complications make single-stage repair too risky [3, 4, 10].

The complexity of corrective surgery is determined by morphological features of TBH (internal cardiac morphology, relationship of the great arteries, anatomy of coronaries etc) and by associated lesions (subaortic obstruction, coarctation and/or AAO, tricuspid valve attachments, multiple VSDs etc.). Nevertheless, associated cardiac anomalies such as AAO and an unusual coronary pattern were not identified as risk factors for early or late mortality [4]. Our study confirmed these findings and showed reasonable survival benefits despite the high incidence of associated lesions. Low early mortality (2.2%) and an 88% survival rate at 15 years of follow-up compare favourably with recently published series [3–5].

On the other hand, a complicated early postoperative course is common and long-term morbidity is apparent. Reoperations and reinterventions are reported to be more common in TBH with side-by-side arteries, subaortic obstruction and obstruction of AA [4].

Our study demonstrated similar results. Obviously >70% incidence of primary subaortic obstruction and concomitant AA problems seen in our patients is high; nevertheless the 67% freedom from reoperation for both outflow tracts at 15 years is suboptimal. During the study period, which spans over more than three decades, the management strategy and the technique of reconstruction of both outflow tracts have evolved [2, 4, 5]. The current management strategy of primary early correction focuses on the aggressive resection of the conal septum in every patient, avoiding the Lecompte manoeuvre whenever possible particularly with side-by-side position of the great arteries, and shortening and proper sizing of the reconstructed ascending aorta and arch to improve the geometry of both outflows. In fact, there was only one reoperation for outflow tract in patients operated on since 2000. More data and the follow-up are needed to prove the positive impact of this approach on event-free survival.

More reoperations and interventions for recurrent RVOTO than for recurrent LVOTO have been reported [3, 4, 6, 11, 12], but Soszyn et al. [5] recently published a study with a greater number of events for LVOTO than RVOTO. In our cohort, we found a comparable amount of reoperations and interventions performed on both outflow tracts. Morphological variability of LVOT and RVOT might explain these discrepancies.

AR after ASO might develop over time [12–14]. Schwartz et al. [13] and Soszyn et al. [5] identified previous PAB or two-stage repair, VSD closure and older age as risk factors for the development of AR. In patients with TBH, the incidence of moderate and more AR is reported <10% [5, 13] with no need for aortic valve repair or replacement at a mean follow-up of 9 years. On the contrary, our results have shown increased risk of progressive AR in the second decade after the ASO. In our cohort, the incidence of moderate AR was 8%, however, an additional 5 patients required neoaortic valve intervention; therefore, the freedom from AR > moderate and/or aortic valve replacement/reconstruction was only 76% at 15 years. We were not able to identify any risk factors for the development of AR after surgery. Two-stage repair, an older age, the technique of VSD closure, and aortic root dilatation did not predict the development of AR, and in fact after repair, only 1 patient had moderate AR, and the remaining patients had no or mild AR at discharge. Later on, immobilization of the annulus and aortic cusp adjacent to the patch used for trans-pulmonary VSD closure was found in 2 patients, who had undergone reconstruction of the aortic valve due to progressive AR. In any case, 1 has to be aware of the progressive nature of AR during the follow-up.

Another issue is dilatation of the neoaortic root. After an ASO, a prior PAB and an older age were found to be risk factors for root dilatation [13]. McMahon et al. [15] found a TBH itself to be a risk factor for root dilatation, but there were no associations with an unusual coronary pattern, the technique of coronary transfer or associated lesions. Neoaortic root dilatation was identified to be a risk predictor of progressive AR [12, 15, 16]. In our cohort of patients, 5 of 6 patients with aortic root dilatation >3Z had none or mild AR, and only 1 patient has severe AR and is waiting for surgery. Our data did not confirm an impact of older age or two-stage repair on the development of root dilatation. Only 1 in 6 patients was post-PAB, and the patient’s age at the time of ASO was below the mean age of the whole patient population. The trap-door technique was no predictor of dilatation of the aortic root.

The incidence of recurrent AAO is ~10% in a larger series [3, 7, 10].

In our cohort, the rate of reoperation for recurrent CoA was 15%; not surprisingly, all patients who underwent reoperation on the LVOT had initially presented with a hypoplastic AA or CoA, or interruption of the arch. Aggressive resection of ductal tissue, the technique of end-to-side repair as well as shortening and proper sizing of reconstructed ascending aorta to improve geometry of the arch might decrease the incidence of recurrent AAO.

Despite the palliative nature of ‘correction’ of the TBH, the functional outcomes are encouraging. The majority of patients have normal biventricular function, leaving without any cardiac medications. Since 2000, when this management strategy was introduced, only one reoperation on the arch was needed.

CONCLUSION

Corrective surgery offers excellent survival benefits and encouraging long-term functional outcomes, regardless of the coronary...
anatomy and associated lesions. Normal biventricular function is preserved in the vast majority of patients, and > three-fourth of patients are without cardiac medication. Nevertheless, TBH associated with a complex anatomy continues to be a risk factor for long-term morbidity, and redo and reinterventions are equally common on both outflow tracts. Progressive neoaortic regurgitation and neoaortic root dilatation might be a problem in the future; therefore, close life-long surveillance of patients is necessary.

Conflict of interest: none declared.

REFERENCES


APPENDIX. CONFERENCE DISCUSSION

Dr C. Brizard (Melbourne, Australia): This paper is the third or the fourth from your unit on the topic in the last 20 years. I think it is very important that excellence in the unit is perpetuated throughout the generations of surgeons. Your paper demonstrates very good survival as shown by most of the literature, so I do not have any questions on that. But you have a relatively high reoperation rate, and my questions will be mostly technical.

You mention in the manuscript, that you have reoperation on the aortic valve for aortic regurgitation due to the proximity of the patch. I would like to ask you about your VSD closure technique in detail, and which patch material you use.

Dr Schwarz: A dacron patch is used to close the VSD. And I think it is very important to stay away from the aortic annulus in order to preserve the function of the aortic valve.

Dr Brizard: You have a large number of reoperations and reintervention on the aortic arch. I have found six dilation and stenting and redo arch in four out of 29 patients who have had arch surgery. So what technique of arch repair did you use? Has it evolved over time, and do you see a difference?

Dr Schwarz: For the arch repair, we use the patch technique, and this might play a role in the development of recurrent obstruction of the aortic arch.

Dr Brizard: You also have a large number of reoperations for right ventricular outflow tract obstruction despite an aggressive management at the time of primary repair. You have 15 patients out of 34 who had right ventricular outflow tract obstruction diagnosed before the repair. Fifteen had recurrent by the time of the primary repair, and despite that, you have eight reinterventions with right ventricular outflow tract obstruction resection. But you also have six transannular patches, two pulmonary valve reconstructions, and you have three RV-to-PA conduit. So do you think that right ventricular outflow tract obstruction might be a result of the hypertrophy in response to obstruction at the pulmonary artery level and the stretched French manoeuvre?

Dr Schwarz: Concerning the RVOTO management, I would like to first point out that we do only resect subvalvular RVOTO during primary repair if there is a substrate to be found. We had three conduits placed, LV-PA conduits, due to the coronary pattern because we had coronaries crossing the outflow tract. And I think, as we discussed in the paper published in 2007, recurrent RVOTO is a problem also in patients who did not undergo resection.

Dr M. Siepe (Freiburg, Germany): May I ask you if you changed your treatment strategy change over time? The experience you report covers quite a long period, and I imagine that with the evolving evidence, you may have changed your strategy - perhaps operating on the patients earlier? And did that have an influence on the outcome parameters you looked at?

Dr Schwarz: You mean generally the operation strategy?

Dr Siepe: I guess that you operate earlier, in younger patients, right now than you did in the beginning of your experience?

Dr Schwarz: Of course, yes.

Dr Siepe: And did this point in influence your results? Did you look at this subgroup?

Dr Schwarz: In this study, we did not particularly look at the subgroup, but, yes, we do operate earlier, and we have good results.

Dr D. Barron (Birmingham, UK): Can I just clarify about your strategy? You said your policy is a single-stage repair, but a fifth of your patients have had two-stage repair. Is that historical now?

Dr Barron: That is historical, yes.

Dr Schwarz: No, never.

Dr F. Lacour-Gayet (New York, NY, USA): One real concern of the arterial switch is the late dilation of the aortic root and the risk of aortic regurgitation. I have a couple of comments and questions in this regard.

I think that to prevent aortic regurgitation, the VSD should be closed in a way that does not impact with the aortic valve leaflets. Initially we had been closing the VSD through the pulmonary valve which is the large one, which is easier, but we ended up with a number of aortic regurgitations. And then we move to closing the VSD through the aortic valve which is smaller, but if you take out first the coronary buttons, you actually have very good access. So my first question is to ask you which way are you currently doing your VSD closure in Taussig-Bing?

Dr Schwarz: In our study, we had mostly transatrial access.

Dr Lacour-Gayet: Through the atrium?

Dr Schwarz: Yes.

Dr Lacour-Gayet: Oh, that is good. The second question is, one of the causes of aortic root dilation is the way we manage the coronary buttons.
Using a technique that is going to enlarge the already very large pulmonary annulus which will become the neo-aortic annulus is, of course, a cause of aortic dilation. So in this regard, I personally believe that the so-called trap-door technique is very likely to enlarge the pulmonary annulus. I know that people have very good results with this technique, but I wonder if this is not a cause of increased risk of aortic root dilation. And I would like to have your input on that point, although you said it did not seem to be significant in your experience.

Dr Schwarz: Yes. I think altogether we will be able to show better evaluation in a few more years because the cohort of patients who presented with these issues is too small. But we do downsizing of the neo-aortic root, and I will be happy to maybe evaluate this in the future.