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Right Ventricle Failure and Outcome of Simple and Complex Arterial Switch Operations in Neonates

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Aim. To analyze the causes and role of right ventricle failure in the morbidity and mortality after arterial switch operation for transposition of the great arteries in neonates.

Method. Between January 1999 and December 2001, 62 neonates underwent arterial switch operation. The simple transposition group was comprised of 39 patients with transposition of the great arteries and intact ventricular septum. The complex transposition group included 23 patients with large ventricular septal defects, accompanied with left ventricle outflow tract obstruction in 6 cases and dextrocardia in 1 case. Arterial switch operation was performed on elective basis in all but 3 patients who underwent emergency operation.

Results. Patients with complex heart defects had significantly lower body weight ($p = 0.008$) than patients with simple trasposition of great arteries. The usual coronary artery pattern (ie, the left anterior descending artery and circumflex artery arising from the right aortic sinus; the right coronary artery arising from the left aortic sinus) was found in 74% of the neonates in the simple transposition group and 65% of the neonates in the complex transposition group. Age, weight, coronary artery anatomy, cardiopulmonary bypass, duration of aortic cross-clamp, bleeding, and the need for delayed chest closure did not influence the outcome of surgery. Low cardiac output after surgery was more common in the complex transposition group ($p = 0.0001$), although it was not a predictor of fatal outcome. Preoperative hypoxia coupled with acidosis (odds ratio (OR), 5.70; 95% confidence intervals (CI), 4.45-7.44), and emergency operations (OR, 3.62; 95% CI, 2.22-5.59) were strong predictors of unfavourable outcome. We lost 4 patients out of 62 (6.5%) because of right ventricle failure caused by persistent pulmonary hypertension. Right ventricle failure on the second postoperative day, e.g., sustained increased central venous pressure > 15 mm Hg ($p < 0.001$) and high velocity tricuspid regurgitation > 4 m/s ($p = 0.002$), indicated bad prognosis.

Conclusion. Difficult coronary anatomy was not a risk factor for morbidity and mortality after arterial switch operation. Poor preoperative health condition, hypoxia (despite effective balloon atrioseptostomy), and acidosis contributed to persistent pulmonary hypertension. Operation on the emergency basis and tricuspid valve insufficiency with right ventricle failure were strong predictors of unfavorable outcome.

Key words: *anastomosis, surgical; cardiac surgical procedures; heart defects, congenital; infant, newborn; surgical procedures, operative; transposition of great arteries; treatment outcome*

Most cardiac surgery centers have low neonatal mortality and morbidity rates after a simple arterial switch operation for the treatment of transpositon of great arteries (1,2). Mortality and morbidity remain higher in cases with coexisting ventricular septal defect, left ventricle outflow tract obstruction, and coronary anomalies (3). Patients with transposition of the great arteries constitute a well-defined group regarding the age of presentation, morphology, and perioperative management. The arterial switch operation has become as a standard, primary operation and a method of choice in the treatment of neonates with transposition of great arteries (1). It offers anatomic correction and has very good late results (4). After arterial switch operation, most patients are asymptomatic and can have a normal life. Formerly applied method, the atrial switch procedure (Senning or Mustard procedure), was followed by significant late morbidity and mortality due to late arrhythmia, right ventricle failure, and venous channel obstruction (5,6), whereas the arterial switch operation for transposition of the great arteries with intact ventricular septum can currently be performed early in life with a low mortality risk ($< 5\%$) (1,5). However, mortality and morbidity afer arterial switch operation remain higher in cases with a coexisting ventricular septal defect, left ventricle outflow tract obstruction, or coronary anomalies (6). We analyzed perioperative morphologic and physiologic predictors associated with right ventricle failure, which may affect the outcome of the

treatment of neonates with complex transposition of the great arteries.

Patients and Methods

Between January 1999 and December 2001, 62 consecutive neonates with the primary diagnosis of transposition of the great arteries underwent arterial switch operation at our institution. There were 40 boys and 22 girls. The median age of the neonates was 5 days (range, 2-26), and their median weight was 2.8 kg (range, 1.9-4.7 kg). Two patients with the transposition of great arteries and simultaneous coarctation of the aorta were excluded from study. Other exclusion criteria were unsuitable morphology for biventricular repair and provision of Rastelli repair (further two patients matching these criteria were unsuitable for arterial switch operation and underwent systemopulmonary shunts over the study period). The neonates were divided into two groups: simple transposition group and complex transposition group (Table 1). The simple transposition group consisted of 39 patients with transposition of the great arteries but intact ventricular septum; 3 patients with small muscular ventricular septal defects were also included in this group. In the complex transposition group we included 23 patients with large ventricular septal defects accompanied with left ventricle outflow tract obstruction in 6 patients, left pulmonary artery stenosis in one, and dextrocardia in another patient. The anatomic basis of the left ventricle outflow tract obstruction was accessory mitral valve tissue in 3 patients, bicuspid valvar stenosis in 2, and malaligned outlet septum in one patient. In the latter 3 cases, the pulmonary annulus diameter averaged only 80% of that of the aorta. Morphologic diagnosis, including coronary anatomy, was carefully assessed before the surgery by two-dimensional and Doppler echocardiography. No angiograms were performed to assess coronary anatomy.

Out of 62 patients, 40 underwent preoperative balloon atrial septostomy and all received prostaglandin-E_1 infusion. After an average of 5 days of stabilization, we performed elective repair by opening the chest through a midline sternotomy. We cannulated ascending aorta and both venae cavae and ran cardiopulmonary bypass at moderately hypothermic conditions (>28°C), using cold crystalloid cardioplegia. Coronary buttons were often used to enlarge the ascending aorta, especially in cases of aorta-to-pulmonary artery size mismatch (Fig. 1). Ventricular septal defects were closed with polytetrafluoroethylene patch and running polypropylene suture through the tricuspid valve in all patients but two. In those two cases, the ventricular septal defect was addressed through the pulmonary (neoaorta) orifice. In all patients, Lecompte-manoeuvre (translocation of the pulmonary arteries over the posterior aorta) was performed, followed by pulmonary artery reconstruction accomplished by a pantaloon-shaped autologous native pericardium patch. Relief of the left ventricle outflow tract obstruction was executed through the pulmonary (neoaorta) orifice. Atrial septal defects were closed with a running suture. Difficult repair, longer aortic cross-clamping time, and longer duration of cardiopulmonary bypass yielded reduced the possibility to leave the chest electively open. Neither left artial nor pulmonary artery pressure were monitored. Hemodynamic parameters were carefully assessed by intraoperative echocardiography and routine monitoring after surgery (arterial and central venous pressures, temperature, and urine output). Routine echocardiographic protocol comprised of a two-dimensional-Doppler study within 6 h of the arrival to the intensive care unit, then 24 and 48 h after surgery. Right ventricle and pulmonary artery pressures were estimated echocardiographically by measuring tricuspid and/or pulmonary regurgitation velocity, and plotted against the central venous pressure. Pulmonary hypertension was assumed when echocardiographically determined right sided pressures reached two-thirds of the systemic pressures and no obstruction could be observed in the right ventricle outfow tract or pulmonary brach arteries.

Table 1. Demographic and perioperative characteristics of 62 neonates undergoing arterial switch operation for simple and complex transposition of the great arteries

Characteristic	No. of patients with transposition of the great arteries		
	simple (n = 39)	complex (n = 23)	p
Age (days, mean ± SD)	11.33 ± 5.75	10.04 ± 6.48	0.434[a]
Weight (kg, mean ± SD)	3.36 ± 0.47	2.99 ± 0.52	0.008[a]
Sex (boys/girls)	26/13	13/10	0.155[b]
Coronary anatomy:			0.121[b]
Yacoub type A[c]	29	14	
Yacoub type D[d]	6	5	
Yacoub type E[e]	4	4	
Pulmonary artery/aorta ratio (mean ± SD)	0.92 ± 0.18	0.76 ± 0.18	0.002[a]
Preoperative hypoxia and metabolic acidosis (No.)	2	2	0.336[b]
Emergency operation (No.)	1	2	0.26[b]
Cardiopulmonary bypass (min, mean ± SD)	145.5 ± 37.9	157.3 ± 30.9	0.187[a]
Aortic crossclamp time (min, mean ± SD)	87.7 ± 21.1	91.5 ± 21.9	0.508[a]
Bleeding (No.)	3	3	0.263[b]
Chest left open (No.)	6	8	0.055[b]
Low cardiac output (No.)	2	5	0.001[b]
Mechanical ventilation (day, mean ± SD)	4.0 ± 1.7	5.1 ± 2.9	0.16[a]
Intensive care unit stay (day, mean ± SD)	6.4 ± 2.9	7.6 ± 4.3	0.286[a]
Mortality (No.)	1	3	0.123[b]

[a]Two-tailed paired Student's t-test.
[b]Fisher's exact test.
[c]Yacoub type A – the left anterior descending and circumflex arteries arising from the right aortic sinus and the right coronary artery arising from the left aortic sinus.
[d]Yacoub type D – the left anterior descending coronary artery arising from the right aortic sinus, and the right coronary artery and circumflex coronary artery arising from the left aortic sinus.
[e]Yacoub type E – the left anterior descending coronary artery and right coronary artery arising from the right aortic sinus, and circumflex coronary artery (=double loop) arising from the left aortic sinus.

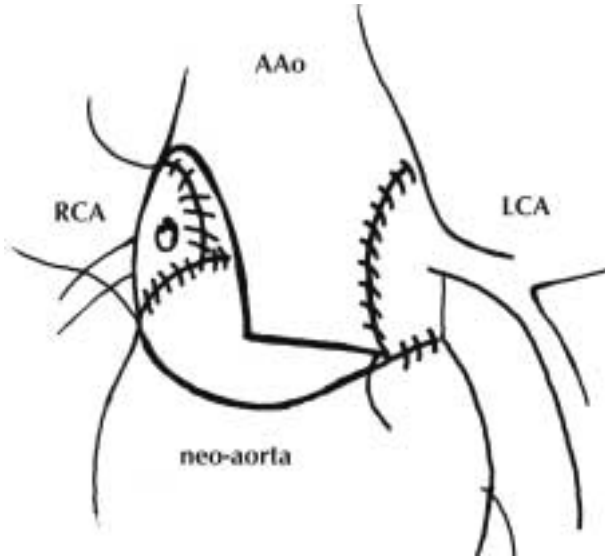

Figure 1. Method of coronary transfer in size-mismatched neoaorta trunk and ascending aorta (AAo). By being implanted above the neoaorta suture line, the coronary buttons contribute to the enlargement of the smaller ascending aorta. LCA – left coronary artery; RCA – right coronary artery.

Statistics

A two-tailed, paired Student's t-test was used for the comparison of numerical variables. P-value <0.05 was considered statistically significant. Categorical data were analysed by Fisher's exact test and significance limit was indicated where appropriate. For statistical analysis, we used Microsoft Excel 2000 and SPSS 7.5 for Windows (SPSS Inc., Chicago, IL, USA).

Results

Simple and complex transposition groups did not differ in age and sex (Table 1). The complex group

had significantly lower body weight than the simple transposition group (p = 0.008). The usual coronary artery pattern was Yacoub type A, ie, the left anterior descending and circumflex arteries arising from the right aortic sinus (ie, aortic sinus facing corresponding pulmonary sinus, positioned to the right of non-facing aortic sinus), and the right coronary artery arising from the left aortic sinus (7). Unusual coronary artery patterns, ie, other than Yacoub type A, were Yacoub types D and E. Yacoub type D included the left anterior descending coronary artery arising from the right aortic sinus, and the right coronary artery and circumflex coronary artery arising from the left aortic sinus. In Yacoub type E the left anterior descending coronary artery and right coronary artery arose from the right aortic sinus, and circumflex coronary artery (=double loop) arose from the left aortic sinus. Unusual coronary artery patterns were observed in 25.6% of the neonates in the simple transposition group and 34.8% of the neonates in the complex transposition group. In two patients with ventricular septal defect and left ventricle outflow tract obstruction, a single coronary system (all branches – left anterior descending, circumflex, and right coronary artery – stemming from a single orifice) arose from the left aortic sinus, with the left coronary artery coursing between the two great arteries. Preoperative echocardiography was accurate in predicting coronary anatomy in 57 out of 62 (91.9%) neonates. The relationship of the great arteries was antero-posterior in 32 out of 39 (82.1%) neonates in the simple transposition group and in 5 out of 23 (21.7%) in the complex transposition group (chi-square = 21.78, $p < 0.001$). Usually, antero-posterior relationship between the great vessels allows more straightforward surgery, whereas side-by-side great vessels may be more difficult to switch back (3). Side-by-side relationship of the great vessels was associated with a high incidence of unusual coronary patterns (Yacoub types D and E). Both side-by-side great vessles and pulmonary artery-to-aorta size mismatch were more frequent in the complex transposition group (p = 0.002). The simple and complex transposition groups differed in aortic cross-clamping time, cardiopulmonary bypass duration, and postoperative parameters, but not in low cardiac output. Surviving and deceased patients did not differ in age, body weight, and coronary artery anatomy (Table 2). Longer cardiopulmonary bypass and aortic cross-clamping time, occurence of bleeding or the need for delayed chest closure did not differ between the survivors and the deceased, either. Low cardiac output after surgery was more common in the complex transposition group (p = 0.001); it occured in three neonates as a solitary but reversible problem. In four patients, low cardiac output associated with pre-existing pulmonary hypertension and/or right ventricle failure was a mode of death. Preoperative hypoxia (arterial $pO_2 < 3.8$ kPa) and metabolic acidosis (pH < 7.25, base excess > 8 mmol/L) affected four patients, of whom three underwent surgery on an emergency basis. Preoperative hypoxia associated with acidosis (OR = 5.70, 95% CI = 4.45-7.44), and emergency operations (OR = 3.62, 95% CI = 2.22-5.59) were strong predictors of unfavourable outcome. We lost one patient in the simple transposition group due to multiorgan failure induced by preoperative hypoxia and acidosis. This patient had undergone surgery on an emergency basis and presented with intractable (ie, resistant to nitric oxyde) pulmonary hypertension and concomitant right ventricle failure. No other pulmonary hypertensive episodes were observed in the simple transposition group. Three out of 23 neonates in the complex transposition group were lost. In these 3 neonates, intractable right ventricle failure was a primary cause of low cardiac output. One of them was an emergency case with severe hypoxia and acidosis despite effective balloon atrioseptostomy. The cause of right ventricle failure after surgery was pulmonary hypertension in two patients, and gross tricuspic valve regurgitation caused by restricted leaflet motion by the ventricular septal defect patch in one patient. In the complex transposition group, five more patients had episodes of increased pulmonary pressure and pulmonary hypertension, which could all be reversed with nitric oxyde and/or pulmonary vasodilators. The analysis of postoperative hemodynamic parameters in the survivors and the deceased showed that persistent right ventricle failure, e.g. sustained elevated central venous pressure (> 15 mm Hg, $p < 0.001$) and high velocity tricuspid regurgitation (> 4 m/s, p = 0.002), on the second postoperative day indicated unfavorable outcome (Table 3).

Echocardiography in 57 of 62 neonates revealed a temporary, albeit universal, decrease in mean left ventricle linear ejection fraction from 0.35 ± 0.11 to 0.29 ± 0.08 on the first postoperative day (difference between survivors and non-survivors did not reach

Table 2. Demographic and perioperative characteristics of surviving and deceased neonates undergoing arterial switch operation for transposition of the great arteries

Characteristic	No. of patients undergoing arterial switch operation: survivors (n = 58)	deceased (n = 4)	p
Age (day, mean ± SD)	10.83 ± 6.19	11.25 ± 2.50	0.786[a]
Weight (kg, mean ± SD)	3.22 ± 0.53	3.21 ± 0.36	0.933[a]
Sex (boys/girls)	39/19	1/3	0.10[b]
Coronary anatomy:			0.259[b]
Yacoub type A[c]	42	2	
Yacoub type D[d]	10	1	
Yacoub type E[e]	6	1	
Pulmonary artery/aorta ratio (mean ± SD)	0.87 ± 0.20	0.75 ± 0.13	0.168[a]
Preoperative hypoxia and metabolic acidosis	1	3	< 0.001[b]
Emergency operation	1	2	0.0001[b]
Cardiopulmonary bypass (min, mean ± SD)	148.2 ± 35.9	173.5 ± 23.5	0.111[a]
Aortic cross-clamp time (min, mean ± SD)	89.6 ± 22.0	97.5 ± 9.8	0.216[a]
Bleeding (No.)	6	0	0.658[b]
Chest left open (No.)	12	2	0.184[b]
Low cardiac output (No.)	3	4	< 0.001[b]

[a]Two-tailed paired Student's t-test.
[b]Fisher's exact test.
[c]Yacoub type A – the left anterior descending and circumflex arteries arising from the right aortic sinus and the right coronary artery arising from the left aortic sinus.
[d]Yacoub type D – the left anterior descending coronary artery arising from the right aortic sinus, and the right coronary artery and circumflex coronary artery arising from the left aortic sinus;
[e]Yacoub type E – the left anterior descending coronary artery and right coronary artery arising from the right aortic sinus, and circumflex coronary artery (=double loop) arising from the left aortic sinus.

Table 3. Comparison of postoperative hemodynamic parameters (mean ± SD) of surving and deceased neonates after arterial switch operation for transposition of the great arteries

Parameter	Survivors (n = 58)	Deceased (n = 4)	p[a]
Blood pressure on the day of operation (mm Hg)	49.03 ± 6.25	42.25 ± 6.08	0.108
Blood pressure on the 1st postoperative day (mm Hg)	49.83 ± 5.84	43.25 ± 5.74	0.121
Blood pressure on the 2nd postoperative day (mm Hg)	54.48 ± 4.54	37.33 ± 4.62	0.028
Central venous pressure on the day of operation (mm Hg)	7.28 ± 2.83	12.50 ± 5.75	0.004
Central venous pressure on the 1st postoperative day (mm Hg)	7.2 ± 2.6	15.0 ± 2.0	0.002
Central venous pressure on the 2nd postoperative day (mm Hg)	6.1 ± 2.0	16.0 ± 4.5	< 0.001
Tricuspid regurgitation velocity on the day of operation (m/s)	2.7 ± 0.6	4.1 ± 0.6	0.029
Tricuspid regurgitation velocity on the 1st postoperative day (m/s)	3.1 ± 0.5	4.0 ± 0.4	0.010
Tricuspid regurgitation velocity on the 2nd postoperative day (m/s)	3.07 ± 0.45	4.40 ± 0.21	0.002
Left ventricle linear ejection fraction on the day of operation	0.38 ± 0.06	0.33 ± 0.05	0.135
Left ventricle linear ejection fraction on the 1st postoperative day	0.32 ± 0.06	0.28 ± 0.04	0.080
Left ventricle linear ejection fraction on the 2nd postoperative day	0.37 ± 0.07	0.32 ± 0.03	0.063

[a]Two-tailed, paired Student's t-test.

statistical significance). Major complications or residual defects were observed only in two neonates from the simple transposition group, who had to undergo early reoperation for left atrium obstruction relief and left pulmonary arterioplasty within 4 and 7 days, respectively. Both neonates fully recovered afterwards. Relief of left ventricle outflow tract obstruction was completely achieved even in cases of smaller neoaorta annulus diameter. Postoperative two-dimensional and Doppler echocardiography revealed a maximum of 2.5 m/s velocity across the left venticle outflow tract-neoaorta junction. Formal closure of the chest was accomplished on the first postoperative day in 10 out of 12 cases. There was only one case of temporary atrioventricular block, which occured in a neonate with complex transposition in whom the ventricular septal defect was closed through the pulmonary (neoaorta) valve.

The follow-up period lasted 5-35 months. All hospital survivors were asymptomatic and majority was not administered any medication. There were no late deaths or reoperations.

Discussion

Patients who underwent the arterial switch operation for complex transposition of the great arteries in this study did not die of causes related to the operation. Not even a very difficult coronary anatomy, such as origin of all coronary branches from a single orifice in the left aortic sinus and the course of the left anterior descending and circumflex coronary artery between the great vessels, presented a risk factor. Our common practice is to implant the right coronary artery above the neoaorta suture line (1,5). Where complex anatomy is concerned, it is advantageous to implant the left coronary button higher above the neoaortic suture line (Fig. 1). Implantation of the coronary buttons higher above the neoaorta has several potential advantages (1). As the rotational axis of the proximal coronary segment opens up (ie, its radius increases), kinking may be avoided and adequate lateralization of the coronaries readily achieved. Furthermore, the cranial part of the coronary button can be used as a patch to enlarge the ascending aorta and thus diminish the mismatch between the circumference of neoaorta and ascending aorta. Also, reimplantation of both coronaries above the sinotubular junction may preserve its integrity, thus reducing the risk of late aortic regurgitation (8). The classical techniques of coronary transfer during arterial switch operation (1,4,5) allow avoiding the dangers of coronary artery obstruction and kinking. With side-by-side orientation of the great vessels, a more generous patch of autologous pericardium is required to avoid the neopulmonary trunk squeezing the coronary ostia. The relief of left ventricle outflow tract obstruction was complete in all our patients.

Preoperative echocardiography often overestimates the degree of left ventricle outflow tract obstruction and/or pulmonary annulus hypoplasia (9,10). We strongly advocate an attempt to open up the valvular/subvalvar region and then proceed to arterial switch operation, even in cases with relatively hypoplastic pulmonary annulus (0.70-0.75 pulmonary artery-to-aorta diameter ratio). In our case of transposition of the great arteries with ventricular septal defect and left ventricle outflow tract obstruction (consisting of posterior malaligned outlet septum, bicuspid pulmonary valve amenable for valvotomy, and pulmonary artery-to-aorta diameters of 5 and 11 mm, respectively) the left ventricle outflow tract obstruction could be redeemed up to an adequate left ventricle outflow tract diameter. Since a straddling tricuspid valve was intraoperatively discovered, the arterial switch operation had to be abandoned and the patient was shifted to univentricular circulation (a Fontan-type of operation at a later period). Poor outcome in our patients was always somehow associated with the right ventricle failure. A number of causes may be offered as an explanation (5,11). Poor preoperative condition, hypoxia (despite effective balloon atrioseptostomy), and metabolic acidosis affected both ventricles, but the left ventricle seemed to have better recovery after surgery. Direct manipulation inside the right ventricle, inadvertent restriction of the tricuspid valve function by inappropriate placement of the ventricular septal defect patch, increased afterload due to increased pulmonary vascular resistance, and residual defects (pulmonary artery kinking or obstruction) contributed to the complexity of the problem. Echocardiographic findings showed a drop in the linear ejection fraction of the left ventricle 24-36 h after surgery, which could have been accompanied by a parallel deterioration in right ventricle function. It is generally acknowledged

that the temporarily reduced left ventricular function does not mean a bad prognosis (12,13). However, every ventricular failure lasting >24 h had to be diagnostically evaluated and treated. Sustained left ventricle failure is rare, and right ventricle failure entails unfavorable outcome. Tricuspid regurgitation of grade II/III, as a surrogate of the right ventricle failure, on the second postoperative day holds much worse prognosis than impaired left ventricle linear ejection fraction. Early administration of nitric oxyde and pulmonary vasodilators improves survival, even in cases where pulmonary vascular resistance is not increased. Nitric oxyde reversed low cardiac output in 3 out of 7 cases in our series. However, such patients may not qualify for surgery except on a high emergency basis since these changes remain reversible only for a short time. Alternatively, adequate preoperative stabilization with nitric oxyde can improve clinical condition and defer surgery until hypoxia and acidosis have been resolved. The fact that longer cardiopulmonary bypass and aortic cross-claming time were not associated with unfavorable outcome encourages us to believe that complex arterial switch may also prove a successful method in pediatric cardiac surgery.

In conclusion, arterial switch operation can be performed with low morbidity in both simple and complex transposition of the great arteries. We did not find unusual coronary anatomy, longer cardiopulmonary bypass and aortic cross-clamping time a possible risk factors. Patients with complex transposition of the great arteries die of unrelated causes to arterial switch operation (ie, unrelated to coronary transfer and arterial trunks' reconstruction). Risk factors for poor outcome are preoperative hypoxia and acidosis, which may require emergency operations and lead to persistent pulmonary hypertension. Tricuspid regurgitation with right ventricle failure is a strong predictor of unfavorable outcome. We believe that the preoperative administration of nitric oxíde in selected cases may contribute to preoperative stabilization and prevent postoperative right ventricle failure, thus improving the outcome.

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