Fate of Pulmonary Artery After Anatomic Correction of Simple Transposition of Great Arteries in Newborn Infants

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From April 1984 to April 1987, surgical anatomic correction was performed in 86 newborn infants, 2–23 days old (6.8±3.6 days, mean±SD) with simple transposition of the great arteries. In all patients, the pulmonary artery was reconstructed by end-to-end anastomosis according to the Lecompte maneuver, including eight patients with side-by-side position of the great arteries. Three different approaches were used. In the first 10 patients (group 1, six survivors), two separate patches of preserved tanned pericardium were used to reconstruct the pulmonary artery, whereas in the next 15 patients (group 2, 13 survivors), a single patch of the same material was used, and in the last 61 patients (group 3, 56 survivors), surgery was performed with a single patch of fresh autologous pericardium. Among the 75 survivors, 68 (including six in group 1, 12 in group 2, and 50 in group 3) were followed serially for at least 6 months (6–48 months, 26±9 months) with sequential noninvasive evaluations. At follow-up, all were asymptomatic with normal growth. Two patients with severe pulmonary artery stenosis (group 1) were successfully reoperated on. Four infants with moderate pulmonary artery stenosis have been followed medically and have had stable right ventricular pressures. The last 62 patients have normal or near-normal right ventricular pressures. The spatial relation of the great arteries did not affect the quality of the results. Group 1 had clearly the worst results. Although there were no statistically significant differences between the results in groups 2 and 3, the best results, judged clinically, were obtained with the last surgical approach (group 3). With this current technique, there have only been two moderate pulmonary artery stenoses, both localized at the pulmonary artery branches, and 46 of the 50 patients have right ventricular pressures less than 40 mm Hg. With this technique, the pulmonary arteries grow satisfactorily, and the right ventricular pressure does not increase. (Circulation 1988;78:870–876)

Anatomic correction (switch) of transposition of the great arteries is being performed more frequently with encouraging results in neonates.¹,² Potential problems with this procedure concern eventual coronary artery kinking with its effects on myocardial perfusion and possible stenosis or alteration in the growth of the great vessels that have been reshaped at surgery. We have reported our early and midterm results on the first 50 patients.³ In those results, we found that the uncommon and life-threatening problems were related to compromised coronary and myocardial perfusion producing early postoperative death (16% in the first 50 infants); the most common complication for survivors concerned the neopulmonary artery (PA) with a 15% incidence of stenosis. However, we could not draw conclusions regarding the PA growth because the follow-up was too short, the number of patients was too small, and the surgical technique of the PA reconstruction changed during the study. It is, indeed, the PA that requires the most extensive surgery during the switch operation. The mobilization of the distal PA with the Lecompte maneuver⁴ may distort the PA branches,⁵ and the reconstruction of the proximal PA that requires a patch to fill the gaps in the posterior wall of the original aorta due to the excision of the coronary arteries may cause main PA stenosis. In addition, the spatial relation of the great arteries may interfere with the PA repair, and there have been reports indicating poor results when the Lecompte maneuver had to be performed with great arteries in a
Patients and Methods

From April 1984 to April 1987, 86 newborn infants, 2–23 days old (6.8±3.6 days, mean ± SD) underwent an arterial switch operation for simple transposition of the great arteries. The spatial relation between the great arteries was more anteroposterior in 78 (d-transposition in 76 and l-transposition in two) and more side by side in eight patients (all in d-transposition). There are 75 survivors, all doing well with a follow-up of at least 6 months. Among these 75 survivors, we obtained a sequential prospective study in 68.

Techniques of Pulmonary Artery Reconstruction

The surgical approach to the reconstruction of the new PA has two aims: reconstitution of the sinuses of Valsalva that have been removed with the coronary ostia and bridging the defect between the aortic orifice and the pulmonary artery. In all patients, the Lecompte maneuver was applied (Figure 1) to avoid the use of prosthetic or modified biological conduits used by others.6,7 This was always possible, even in the eight patients with a side-by-side disposition of the great arteries. The reconstruction of the proximal PA was performed with three consecutive techniques that led us to separate the 68 follow-up survivors into three groups:

1) From March to September 1984, in the first 10 patients, we closed the defects made in the aortic wall with two small patches of tanned pericardium (Figure 2A). In this subset (group 1), there were six survivors; all six had a complete follow-up study.

2) From October 1984 to March 1985, in the next 15 patients, the aortic root was reconstructed by a

FIGURE 1. Diagram of Lecompte maneuver. Panel A: Initial situation with both arteries transected and coronary ostia excised as in Group 1 (see Figure 2A). Aorta lies anterior and right to the pulmonary artery. Panel B: Final situation. Pulmonary artery has been passed in front of the aorta.

FIGURE 2. Diagram of techniques of coronary artery transfer. Panel A: Coronary arteries are anastomosed after removal of buttons from the aortic wall. Holes in the new pulmonary artery are filled with two separate patches (group 1). Panel B: Valsalva sinuses, destroyed by the excision of the coronary arteries, have been reconstructed with a large single “pantaloon” patch (groups 2 and 3).
single, large patch of tanned pericardium with a notched inferior border tailored to fit the posterior commissure (Figure 2B). In this group (group 2), there were 13 survivors; 12 of these had a complete follow-up study.

3) From March 1985 to April 1987, in the last 61 patients, the same technique of a single patch was used, but the material was changed to fresh autologous pericardium collected at the beginning of the operation. In this group (group 3), there were 56 survivors; 50 of these had a complete follow-up study.

Methods of Postoperative Follow-up

The 68 survivors in the study were followed serially in our outpatient clinic for at least 6 months, with a planned check-up at 1, 3, 6, 12, and 18 months postoperatively, and then once every year. Each examination included chest radiograph, 12-lead standard electrocardiogram, two-dimensional and M-mode echocardiography, and pulsed and continuous wave Doppler examination. The sizes of the PA anulus, the main PA, and the two branches were measured from a parasternal (Figure 3A) or a subxiphoid view (Figure 3B), and the larger size obtained was included in the results.

Right ventricular (RV) pressure was roughly estimated from the left ventricular (LV) end-systolic geometry on a transverse parasternal or subxiphoid view at the level of the tip of the mitral valve. The deformation of the LV by the effect of an increase in RV pressure on septal (and therefore LV) geometry was estimated by the ratio of the lateral to the anteroposterior LV diameters in systole, a method recently validated in our laboratory and one that we checked in this study by comparing estimated and measured right ventricular (RV) pressures in the patients that were catheterized. In five patients, tricuspid insufficiency was present on the Doppler examination. In these, the RV pressure was estimated from the velocity with the simplified Bernoulli formula and by assuming a right atrial pressure of 5 mm Hg (RV pressure = 4 V^2 + 5 mm Hg).

Right heart catheterization was obtained in 32 patients. It was performed systematically only for the first 26 patients, 12–18 months postoperatively. Subsequently, it was indicated only in case of a suspected complication (loud systolic murmur, persistent RV hypertrophy on the electrocardiogram, asymmetrical lung perfusion on chest radiography, and suspected RV pressure >50 mm Hg on echocardiographic or Doppler studies) or to assess the aorta or the coronary arteries. The six last patients were in these categories. In all patients, pressures were obtained in the main PA, and angiograms were performed in the RV or the PA. A complete noninvasive study was always performed just before catheterization to validate the noninvasive estimations. Pulmonary stenosis was considered to be significant when RV pressure was greater than 39 mm Hg, moderate between 50 and 70 mm Hg, and severe when greater than 70 mm Hg.

The evolution of the pressure gradient and the evaluation of the growth of the new PA were established by the sequential analysis of the anatomic and hemodynamic variables measured and estimated every 6 months by noninvasive techniques.

Statistical Analysis

Comparison between echographic estimations and hemodynamic measurements were made by a linear regression. Comparisons between the three groups were made by analysis of variance comparing values for patients at the same age and at follow-up.

Results

The length of follow-up of our patients in our laboratory is in Table 1. The catheterization hemodynamic and angiographic data are summarized in Table 2. The relation between catheterization and noninvasive data is shown in Figures 4 and 5. It can
be seen that LV end-systolic geometry correlated well to the RV:LV systolic pressure ratio (Figure 4) with $r=0.86$ and that two-dimensional echocardiographic assessment of the PA anulus size correlated well to angiographic measurement (Figure 5) with $r=0.82$.

Global results show that only two patients had severe stenosis (RV pressure $>70$ mm Hg), and four had moderate stenosis (RV pressure between 50 and 70 mm Hg). The two patients with severe stenosis had RV systolic pressures of 80 and 120 mm Hg, respectively, mainly due to a retraction of the two tanned pericardial patches (group 1) involving not only the main PA but also the valve and the outflow tract (Figure 6). Both were successfully reoperated on 12 and 18 months after the switch with a transanular patch as was used for correction of tetralogy of Fallot. They are doing well 28 and 36 months after surgery with mild PA stenosis and regurgitation. The four patients with moderate PA stenosis had a normal RV in size and function. They are being followed medically (6–26 months postoperatively) with an apparently stable degree of stenosis. One patient, from group 1, had isolated valvar pulmonary stenosis that is likely to require percutaneous balloon valvuloplasty; one belongs to group 2, with supravalvar stenosis; and two are from group 3, with isolated PA branch stenosis, including one with hypoplastic branches as shown in Figure 7. The remaining 62 patients (91%) had normal or near-normal RV pressures, and 46 of the 50 patients of group 3 had measured or estimated RV pressures below 40 mm Hg. The poorest results were found in group 1 with significantly higher pressures and a high incidence (two of six patients) of severe PA stenoses. There were no significant differences in RV pressures between groups 2 and 3.

The location of the stenosis seems different among the groups. In groups 1 and 2 (tanned pericardium), the stenosis was predominantly supravalvar, eventually subvalvar (two patients) or valvar (three patients) as well but never with involvement of the PA branches. Conversely, in group 3, the only two patients with significant stenoses had stenoses at the branch level with no significant main PA narrowing nor valvar nor subvalvar involvement.

There was no difference in RV pressures between the eight patients with a side-by-side relation of the great arteries ($43 \pm 12$ mm Hg) and the 58 patients with an anteroposterior relation ($46 \pm 13$ mm Hg).

A pulmonary regurgitation was found on Doppler studies in six patients who belonged to group 1 (1 patient), group 2 (1 patient), and group 3 (4 patients). None had volume-loaded RV.

Finally, in the patients operated on according to the current technique (group 3), the RV systolic pressure did not increase over time (Figure 8), and the growth of the PA anulus was normal (Figure 9).

**Discussion**

Only a few reports mention the incidence of PA stenosis after switch operation, but they usually concern a small number of patients and a short follow-up, and sequential studies for observing evolution and growth are missing. In addition, some reports do not specifically deal with neonatal

**Table 1. Length of Follow-up**

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients (n)</th>
<th>Range</th>
<th>Mean ± SD</th>
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<tbody>
<tr>
<td>1</td>
<td>6</td>
<td>37–43</td>
<td>41</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>26–33</td>
<td>29</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>6–29</td>
<td>18</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>6–43</td>
<td>26 ± 9</td>
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</tbody>
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**Table 2. Hemodynamic and Anatomic Data**

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients (n)</th>
<th>After surgery (mo)</th>
<th>RV pressure (mm Hg)</th>
<th>Pressure (mm Hg)</th>
<th>Dimension (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>MPA</td>
<td>RPA</td>
<td>LPA</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>14 ± 3</td>
<td>76 ± 30*</td>
<td>24 ± 4</td>
<td>18 ± 3</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>13 ± 2</td>
<td>45 ± 10</td>
<td>23 ± 3</td>
<td>17 ± 2</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
<td>13 ± 2</td>
<td>38 ± 08</td>
<td>33 ± 8*</td>
<td>15 ± 3</td>
</tr>
</tbody>
</table>

All values are mean ± SD.

RV, right ventricle; MPA, main pulmonary artery; RPA, right pulmonary artery; LPA, left pulmonary artery; Ao, aorta; PA, pulmonary artery.

*p<0.05.
repair and combine the surgical results in simple and complex transposition of the great arteries.

Our purpose, therefore, was to evaluate the incidence and the location of postoperative PA stenosis and to study the growth of the "new" PA after neonatal switch on a large number of patients now followed for several years. We also tried to assess the effects of the surgical technique and of the spatial relation between the great arteries on the results. To obtain sequential data and to avoid unwarranted repeated cardiac catheterizations, we had to use noninvasive techniques to evaluate RV pressures and morphological anomalies of the RV outflow tract and the pulmonary arterial tree.

Doppler estimations of RV to PA pressure gradient may not be accurate because multiple stenoses from the outflow tract to the distal branches may occur in the same patient with different orientations of the flow. The only way to use Doppler accurately in this setting would be to evaluate the RV pressure through a tricuspid insufficiency blood velocity.
This situation is uncommon when RV pressure is low, and we were able to use this method in only five patients. In the remaining 61 patients, we estimated RV pressure by assessing the end-systolic deformation of the left ventricle caused by the RV through the ventricular septum. We realize that this is a rough estimation, but we, as well as others, have already obtained reasonably good results. In addition, as it can be seen from Figure 4, we checked its validity in this specific study in the 32 recatheterized patients with measured RV pressures. Furthermore, to avoid missing any significant stenosis, we elected to perform a control right heart catheterization as soon as the RV:LV pressure ratio was thought to be greater than 0.3, that is, when the lateral:anteroposterior LV dimensions ratio was found to be greater than 1.3. This occurred six times in group 3, and in each instance, RV pressure estimations were correct. We, therefore, believe that the way we assessed sequential RV pressures (Figure 8) did not underestimate the developing pulmonary stenosis.

Regarding the anatomic evaluation of the sizes of the PA components by echocardiography, we are confident of our estimations of the PA anulus (Figure 5), the main PA, and the right branch. We are more concerned about the possibility of missing a left PA branch stenosis that may not affect RV pressure because the right PA is normal. We, therefore, looked carefully for asymmetrical lung perfusion on chest x-ray and performed catheterization in any doubtful case.

Our results indicate that 1) the overall incidence of significant postswitch PA stenosis is low (<10%) and is even lower (<5%) when two separate patches are not used in the repair. These figures are remarkably lower than in previously reported series. They are very similar to those reported by Quagebeur and others, who also use the Lecompte maneuver and a single pericardial patch to reconstruct the PA when the vessels are not side by side.

2) The side-by-side disposition of the great arteries did not affect our surgical approach nor the quality of the repair. This contradicts the results reported by most investigators who avoid the Lecompte maneuver in this situation because of technical problems or bad results. They chose a different approach by inserting a conduit or by dividing the aorta very distally and the PA proximally to have enough tissue to reconstruct a new PA that lies left posterior to the aorta. In our hands, the Lecompte maneuver in a side-by-side disposition was as easy to perform as in others as long as the PA was dissected quite far in periphery and as long as a larger pericardial patch for the reconstruction of the vessel was provided.

3) The surgical approach to reconstruct the PA with a double patch (group 1) was clearly less satisfactory than the one with a single patch (groups 2 and 3) and, therefore, should be avoided. The differences between preserved tanned (group 2) and fresh autologous single pericardial patches (group 3) were not statistically significant. Nevertheless, the large number of excellent results (92%), the extreme stability of RV pressure, and the lower RV pressure during catheterization are in favor of a better outcome with the fresh autologous pericardium. It is also noteworthy that we never observed any serious main PA stenosis in this group, suggesting that there is no early severe retraction of the native fresh autologous patch.

4) The incidence of distal stenoses affecting the PA branches is very low (two of 66 patients) and did not appear to be due to the Lecompte maneuver. In one patient (Figure 7), the branches were already small preoperatively, and this may have been the reason for the stenosis; in the second patient, the deformity was more likely to be due to a retraction of the patch.

5) The growth of the PA is not impaired by the current surgical approach, and the RV pressure does not increase with time. This is a very reassuring finding concerning the long-term prognosis of this type of surgery.

In conclusion, we believe that the fate of the pulmonary artery in those children who have undergone an arterial switch operation for simple transposition of great arteries in the neonatal period is excellent when the current surgical approach is used. The postoperative status of the pulmonary artery must not constitute an argument in favor of atrial repair of the disease or for the use of a prosthetic conduit when arterial switch is performed.

References
2. Castaneda AR, Norwood WI, Jonas RA, Colan SD, Sanders SP, Lang P: Transposition of the great arteries and intact
8. Van Doesburg NH, Biereman FZ, Williams RG: Left ventricular geometry in infants with d-transposition of the great arteries and intact ventricular septum. *Circulation* 1983;38:733–739

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