Anatomical correction of transposition of great arteries with persistent ductus arteriosus

**One year after operation**

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**SUMMARY** The postoperative data are described of a boy who had a ‘switch’ operation for transposition of the great arteries with persistent ductus arteriosus more than a year before. The child is living a normal active life. Electrocardiography, echocardiography, and angiocardiography show persistent abnormality of structure and function of the right ventricle but no evidence of reduced coronary blood flow, coartation at the suture lines of the great arteries, or aortic regurgitation. Reference is made to an identical case operated upon 2½ years ago.

During the past 2 years there have been several reports of the early results of the ‘switch’ operation in transposition of the great arteries (Jatene et al., 1976; Ross et al., 1976; Yacoub et al., 1976; Kreutzer et al., 1977) but detailed follow-up information beyond the early postoperative period is not available. We describe the clinical, angiocardiographic, and serial echocardiographic data obtained over the first postoperative year from a child whose transposition of the great arteries was corrected by this procedure.

**Case report and surgical procedure**

A baby boy was admitted to the Wilhelmina University Children's Hospital in Utrecht at the age of 2 months with cyanosis, dyspnoea, feeding difficulties, and other signs of right and left heart failure. After the diagnosis of transposition of the great arteries with persistent ductus arteriosus was established at cardiac catheterisation a balloon atrioseptostomy was performed. The heart failure was treated medically. The child made satisfactory progress until the age of 5 months when cardiac failure returned with hypoxia and metabolic acidosis. Cardiac catheterisation was repeated (Table). The ductus was still widely patent. M-mode echocardiography disclosed a well-developed and strongly contracting left ventricle. It was decided to switch the great arteries and close the ductus. The operation was carried out using cardiopulmonary bypass with reduced flow and a body temperature of 20°C on 10 May 1977. The ductus was closed at the start of the operation. The pulmonary trunk was situated directly behind the aorta and had a slightly larger diameter. The coronary arteries arose normally from the aorta, they were dissected down to 5 mm from their origin, removed together with a small button

<table>
<thead>
<tr>
<th>Oxygen saturation (%)</th>
<th>Pressures (mmHg)</th>
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<tr>
<td>RA</td>
<td>RV</td>
</tr>
<tr>
<td>Before 71</td>
<td>72</td>
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<tr>
<td>After 78</td>
<td>76</td>
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RA = right atrium; RV = right ventricle; PA = pulmonary artery; LA = left atrium; LV = left ventricle; Ao = aorta.
of aortic wall, and implanted into the side of the pulmonary trunk at sites previously marked with 6/0 prolene sutures. The aorta was divided as far proximally as possible at a level just above the right coronary artery ostium and the pulmonary trunk as far distally as possible just proximal to the bifurcation. The distal part of the aorta could thus easily be sutured to the pulmonary artery root, and a Medox prosthesis (internal diameter 16 mm) used to join the aortic root and the bifurcation of the pulmonary trunk. The holes in the aortic root left by the removal of the coronary arteries were also closed with the prosthesis. Rewarming was carried out during the anastomotic procedure and the atrial septal defect was closed. At the end of bypass the heart was in sinus rhythm. The child made an uneventful recovery and was discharged after 3 weeks. Five months after operation cardiac catheterisation was repeated (Table). Angiocardiography (Fig. 1) showed that the right ventricle was still distinctly trabeculated. The pulmonary conduit was slightly dilated. The region of the suture lines in the aorta and the pulmonary artery showed no narrowing. The left ventricle had a normal appearance and the aortic valve was competent. There seemed to be good filling of the coronary arteries. More than a year later the child was taking no drugs and was thriving with his body weight and height at the 50th centile level. The electrocardiogram showed sinus rhythm, left ventricular preponderance, and no evidence of myocardial ischaemia. Twenty-four-hour ambulatory electrocardiographic tape monitoring showed no evidence of arrhythmias. There were still signs compatible with right ventricular hypertrophy but this had become less. Chest radiography showed mild to moderate cardiac enlargement and persistent plethora. On the M-mode echocardiogram the right ventricle remained enlarged but was clearly smaller than the well-developed left ventricle which was contracting well (Fig. 2).
Transposition of great arteries

Discussion

The ‘switch’ operation is an anatomical correction of transposition of the great arteries and may avoid some of the late complications of the Mustard repair, for example the development of obstruction to systemic and pulmonary venous return (Stark et al., 1974; Venables et al., 1974; Gutgesell and McNamara, 1975). Arrhythmias are also likely to be less of a problem than with the Mustard procedure (El-Said et al., 1972; Tynan, 1978). Certainly routine postoperative electrocardiograms and 24-hour ambulatory electrocardiographic tape monitoring of our patient, 14 months after operation, showed normal sinus rhythm and no arrhythmias. In contrast, too, the Mustard repair has no effect on the usual atrophy of the left ventricle in transposition of the great arteries (Silverman et al., 1978), whereas in our patient postoperative angiocardio-grams and echocardiograms showed normal left ventricular dimensions and contraction.

Other late problems of the Mustard repair, however, may be less avoidable. As the structure of the right ventricle is abnormal in transposition of the great arteries (Becú and Gallo, 1976), persistent abnormalities of its function should be expected, but since after the switch operation it becomes a low pressure ventricle, these abnormalities may at least occur less frequently than after the Mustard repair (Graham et al., 1975). In our patient the postoperative right ventricular angiogram continued to show an abnormal structure. Whether or not a progressive rise in pulmonary vascular resistance, sometimes observed after the Mustard repair (Rosengart et al., 1975), will also occur after the ‘switch’ operation cannot be predicted. The risk is clear if pulmonary hypertension is present preoperatively, particularly if the operation is not undertaken at an early age. In this respect it is interesting that at re-catheterisation after the ‘switch’ operation the pressure in the main pulmonary artery has been found to be either normal or much lower than pre-operatively (Ross et al., 1976; Kreutzer et al., 1977; Abe et al., 1978); in our patient it had fallen from two-thirds to one half of the systemic pressure.

Concern has been expressed about the likelihood of continued patency of the coronary arteries and the growth of the suture lines (Ross et al., 1976). Postoperative angiography in our patient was reassuring on these points and though the period between operation and cardiac catheterisation was relatively short it was a period of rapid growth for this child. The electrocardiogram showed no evidence suggestive of myocardial ischaemia.

Normally, in the presence of an intact interventricular septum the Mustard repair is still preferable to the ‘switch’ operation. Only if left ventricular pressure is close to systemic level can a ‘switch’ procedure be considered. In our case it was raised but only to two-thirds of the systemic pressure and many would have chosen the Mustard repair with division of the ductus, but because angiocardiography and echocardiography had shown a normally sized, well contractile left ventricle with a well-developed wall we opted for the switch operation. So far, the choice seems to have been justified.

One of our first patients in whom a ‘switch’ operation was performed also had an intact ventricular septum and a persistent ductus arteriosus (Ross et al., 1976)—a 20-month-old girl. Unfortunately, as
she lives abroad full follow-up information is not available but it appears that 2½ years after operation this girl is also living a normal active life without complications (J. Somerville, 1978, personal communication).

References


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Addendum

Since submission of this paper, the boy is still thriving. Right ventricular dimension has been further reduced. Whereas the LV/RV rate was 1:4:1 on the M-mode scan one year after the ‘switch’ (Fig. 2) it was 2:7:1 at the end of the second postoperative year. Recent electrocardiograms show less right ventricular activity with normal left ventricular preponderance.
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