THE SURGERY OF TRANSPOSITION OF THE GREAT VESSELS*

BY

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In transposition of the great vessels the aorta gets its blood from the right side of the heart into which the caval veins enter, and the pulmonary artery arises from the left side of the heart, getting its blood from the lungs. The normal crossed circulation has changed into two parallel circulations, and the patient can live only if there are shunts between the two circulations. The interior of the heart may also be grossly deformed, but fortunately in this respect most of these hearts are nearly normal, and it is these that will be discussed.

The diagnosis of transposition is suspected in babies who are blue from birth. Murmurs and ECG are not characteristic. X-ray pictures show the degree of filling of the pulmonary vessels. Angiocardiography shows the aorta arising from the right heart, and is at the same time important as showing the anatomy of the interior of the heart, especially the formation of the ventricular septum. Heart catheterization is extremely valuable if the pressure in the left ventricle is recorded.

According to Noonan, Nadas, Rudolph, and Harris (1960) patients with transposition are of four types.

**Group 1.** Patients in whom transposition is combined with a **ventricular septal defect** (a) with pulmonary stenosis (b) with pulmonic vascular obstruction, and (c) with large pulmonary blood flow.

(a) Ventricular septal defect and pulmonary stenosis. In the absence of an angiocardiogram a diagnosis of 'a severe case of Fallot’s tetralogy' will be made. This does not make much difference, as a shunt, according to Blalock or Potts, is indicated, because a total correction is difficult if not impossible, since the overlying left coronary artery does not allow the stenotic area of the pulmonary outflow to be enlarged. The results of a shunt, however, are gratifying, and here we find the first interesting fact

in the study of transposition. Compared to the relatively large number of 17 patients with a shunt who were operated on, we find only one with this anomaly in our anatomical collection of un-operated transpositions (Fig. 1). This means that the combination of transposition with pulmonary stenosis is favourable for long survival (Fig. 2).

(b) The patients with ventricular septal defect and high pulmonary resistance are quite a different group. In the cyanotic child the loud single second sound implies pulmonary hypertension. Radiographs show increased central lung fields. Angiocardiography confirms the diagnosis, and if one succeeds in taking the pressure in the left ventricle this will be high—as in the first group with pulmonary stenosis.

Until about a year ago we treated these patients with a partial transposition of the venous return according to Baffes, but lately we have changed our policy. The Baffes operation renders a patient less cyanotic and sometimes very much improved, but the high pulmonary resistance is not changed, and ultimately the patient dies from heart failure. Because the patients with transposition and pulmonary stenosis have done well (Fig. 2), we decided to 'band' the pulmonary artery, combining this—when the shunting from left to right is insufficient—with the creation of an atrial septal defect. Follow-up is too short to evaluate this approach, but we are impressed by the good results in 3 patients in whom banding plus atrial septal defect was carried out; the results in 7 patients where only banding was performed are far less gratifying. So we think the combined operation is better, and as the possibility of curing high pulmonary resistance is slight after the age of 3 years, the operation should be done in early childhood in the hope of preparing the children for a total correction in the future.

(c) This group comprises patients with a ventricular septal defect and an increased pulmonary

* A paper read at a meeting of the British Association of Paediatric Surgeons in Rotterdam, September 1964.
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129

48 anat. specimens; age at death
months years
1 3 6 12 2 3 4

28 no VSD
19 VSD
1 VSD+Pst

Fig. 1.—Age at death in 48 cases of transposition. Note the tendency for longer survival if a VSD is present.

Age at which symptoms became severe (alive) or insurmountable (dead)

\[ \text{\_\_\_\_\_\_} = \text{patient alive; age at clinical investigation (60 cases)} \]

\[ \text{\_\_\_\_\_\_} = \text{patient dead; (42 cases)} \]

months years
1 4 6 1 3 5 7 9 11 13 15 17

28 no VSD
47 VSD
27 VSD+Pst

Fig. 2.—Fate of 102 cases of transposition. Note (1) predominantly early death if there is no VSD, and (2) predominantly long survival if VSD and pulmonary stenosis (Pst) are present.

flow. This is rare and the diagnosis is difficult, but again the important point is the pressure in the left ventricle. If this pressure is low or slightly raised in a patient with increased lung fields on radiography, the presence of a (usually) small ventricular septal defect should be suspected. As regards treatment, this group presents the same possibilities as Group 4.

Group II. Patients in whom no ventricular septal defect is present: they survive by virtue of a patent ductus or an atrial septal defect, both of which are seldom adequate, and the children die very soon. Our anatomical collection (Fig. 1) shows 28 amongst 48, nearly all of them having died early. This is a pity as these are just the patients most suitable for a total correction. Thus if one finds a newborn baby, deeply cyanosed, in heart failure, and going rapidly downhill, think of this diagnosis: transposition with no (or small) ventricular septal defect. For the very ill patients in this group an emergency operation is needed to keep them alive, because the available shunts are small. Making a large atrial septal defect will help them to survive and give them a chance to be cured later in life.

For many years anatomists and cardiologists have been 'flirting' with transposition—according to the definition of flirting, paying a lot of attention without real intention of doing something about it. But lately surgeons like Albert, Kay, Merendino, Senning, and Mustard have developed methods of correcting transposition by switching the venous return. These operations have been tried many times, but only a few—I think about 15—successes are known. We are convinced this is because the success of the
operation depends upon the degree of pulmonary vascular resistance. If this is high, success is impossible; but results should be good in cases with normal or only moderately raised pressure in the left ventricle (the ventricle from which the pulmonary artery arises). We take pressure readings at the left side before or during operation, Mustard takes lung biopsies in dubious cases and if the pulmonary resistance is not raised, he proceeds with the correction.

Figs. 3-5 illustrate variations of the Senning operation. The atrial septum is resected and the switching of the veins is accomplished by the use of an interatrial prosthesis of a plastic material or pericardium (Mustard). I believe it is better for the total correction to be done after the preliminary creation of an atrial septal defect, but so far we have not done this.

**Results of Surgery**

The results of operation in 11 cases of transposition are set out in Table 1.

The shunt operations give good palliation in transpositions with pulmonary stenosis. The Hanlon-Blalock procedure is done in all transpositions without pulmonary stenosis, mostly as an emergency operation in extremely ill children, which gives rise to a high mortality. To do the operation under hyperbaric oxygen may perhaps be an advantage.

The essential part of this communication is to give our results for total correction by Senning's technique. A study of the few repaired cases that have been successfully operated on shows that they have one thing in common—there is no high pulmonary resistance and no pulmonary stenosis.

We have tried the Senning operation 7 times (Table 2) with a successful outcome in 2; 2 other patients lived for some time after correction: one died the day before she should have returned home, from a haemoptysis after pleural puncture, and the second after 3 months from brain damage due to excessive (more than 1 hour) total circulatory arrest at low temperature. (We no longer use circulatory arrest.) However, both these patients remained free of cyanosis between operation and death. Two died from mistakes in perfusion technique, which is difficult in these small infants, and in the last patient we missed an atresia of the isthmus of aorta and so, while on perfusion, no blood reached the brain.

**Summary and Conclusions**

In patients with transposition and pulmonary stenosis the Blalock and Potts procedures give good
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Table 1
RESULTS IN SIX DIFFERENT TYPES OF OPERATION IN 71 CASES OF TRANSPOSITION

<table>
<thead>
<tr>
<th>Procedure</th>
<th>No.</th>
<th>Operative Mortality</th>
<th>Late Mortality</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Senning</td>
<td>7</td>
<td>3</td>
<td>1 died after 1 mth.</td>
<td>2</td>
</tr>
<tr>
<td>Blalock-Hanlon</td>
<td>22</td>
<td>9</td>
<td>1 died after 3 mth.</td>
<td>11</td>
</tr>
<tr>
<td>Baffes</td>
<td>15</td>
<td>5</td>
<td>2 died after 1 mth.</td>
<td>10</td>
</tr>
<tr>
<td>Anastomosis</td>
<td>17</td>
<td>0</td>
<td>2 had too big a shunt: and died 3 and 6 mth. after operation</td>
<td>15</td>
</tr>
<tr>
<td>Banding PA</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Creation of ASD + banding PA</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>17</td>
<td>6</td>
<td>48</td>
</tr>
</tbody>
</table>

Table 2
RESULTS OF SENNING OPERATION FOR TOTAL CORRECTION IN 7 CASES OF TRANSPOSITION

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Pre-operative Pressures (mm. Hg)</th>
<th>Anatomy</th>
<th>Corrections</th>
<th>Post-operative Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>RV</td>
<td>LV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3 mth.</td>
<td>—</td>
<td>—</td>
<td>PFO, PDA, VSD atretic, aortic isthmus</td>
<td>Closure PDA and VSD, Senning operation</td>
</tr>
<tr>
<td>2</td>
<td>11 mth.</td>
<td>80/0</td>
<td>25/0</td>
<td>PFO</td>
<td>Senning operation</td>
</tr>
<tr>
<td>3</td>
<td>3½ mth.</td>
<td>70/0</td>
<td>50/0</td>
<td>PFO</td>
<td>Senning operation</td>
</tr>
<tr>
<td>4</td>
<td>8 mth.</td>
<td>140/5</td>
<td>42/0</td>
<td>PFO, PDA, coarctation</td>
<td>Senning operation, closure PDA, Senning operation</td>
</tr>
<tr>
<td>5</td>
<td>2½ yr.</td>
<td>70/0</td>
<td>32/0</td>
<td>PFO</td>
<td>Senning operation</td>
</tr>
<tr>
<td>6</td>
<td>16 yr.</td>
<td>100/0</td>
<td>27/0</td>
<td>PFO</td>
<td>Senning operation</td>
</tr>
<tr>
<td>7</td>
<td>5 yr.</td>
<td>100/5</td>
<td>45/0</td>
<td>PFO</td>
<td>Senning operation</td>
</tr>
</tbody>
</table>

PFO = patent foramen ovale; PDA = patent ductus arteriosus; VSD = ventricular septal defect.

Palliation, and will keep the patients alive until a way is found to deal with the coronary artery which crosses the stenotic pulmonary artery.

The creation of an atrial septal defect can be highly effective as an emergency operation and may prepare the patient for a total correction. The same is true for the treatment of high pulmonary resistance by ‘banding’, combined with the creation of an atrial septal defect.

The shunt, the banding, and the atrial septal defect prepare for the final answer—total correction. And here the Senning procedure (or its modifications) is proving to be valuable in carefully selected patients.

REFERENCE