Complete Repair of Transposition of the Great Arteries with Pulmonary Stenosis

A Review and Report of a Case Corrected by Using a New Surgical Technique

By G. C. Rastelli, M.D., Robert B. Wallace, M.D., and Patrick A. Ongley, M.D.

SUMMARY

Complete surgical correction of transposition of the great arteries associated with subvalvular pulmonary stenosis carries a high mortality rate. A new surgical technique that achieves redirection of the ventricular outflows and relieves pulmonary stenosis by bypassing it, was successfully used to repair complete transposition of the great arteries associated with ventricular septal defect (VSD) and valvular and subvalvular pulmonary stenosis in a 14-year-old patient. The repair consists of (1) division of the pulmonary artery, the cardiac end of which is oversewn, (2) repair of the VSD with a patch in such a way as to connect the left ventricle with the aorta, and (3) reconstruction of the pulmonary artery with an aortic homograft, including the aortic valve, which is anastomosed between the distal end of the pulmonary artery and the right ventricle.

A review of the cases in which the current techniques were used indicates that the location and nature of the obstruction in the left ventricular outflow tract defies successful repair in most instances. Localized ridges and diffuse hypoplastic outflow tracts are recognized causes of obstruction, but anomalies of the mitral valve commonly contribute to or are the primary cause of subvalvular obstruction.

Additional Indexing Words:
Dextro-transposition Complete transposition Aortic homograft

COMPLETE REPAIR of dextro-transposition1 (complete transposition) of the great arteries associated with severe pulmonary stenosis carries a high surgical mortality rate. A review of the literature has revealed no series in which complete repair has been done in such cases. Many patients with this defect have undergone palliative operations2 (subclavian artery-pulmonary artery or caval-pulmonary artery anastomoses) although the patients were of an age suitable for complete correction. Hemodynamically, most of these patients would be ideal candidates for complete repair because their pulmonary vascular bed is “protected” from obstructive changes that develop in absence of pulmonary stenosis and make transposition and VSD inoperable by late infancy or early childhood3 in most patients. Some of the surgical problems related to the anatomy have been pointed out by Shaher and associates.4 Clinical5,6 and angiocardiographic7 clues to diagnosis have been presented. The difficulties in differentiating this defect from tetralogy of Fallot are recognized, and angiocardiography is the most reliable differential diagnostic test. Barcia and associates8 reported that 47 of 60 patients with dextro-transposition studied by use of angiocardiography had VSD; and of these 47, 17 (36%) had valvular or subvalvular pulmonary stenosis. Of 36 patients with pulmonary stenosis in a combined

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Table 1

Patients Operated on for Dextro-Transposition and Valvular Pulmonary Stenosis

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex and age (yr)</th>
<th>Preoperative pressure (mm Hg)</th>
<th>Surgical repair</th>
<th>Pressure after repair (mm Hg)</th>
<th>Outcome</th>
<th>Pathological findings or follow-up</th>
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<td>Right</td>
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<td>Left</td>
</tr>
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<td>F 12</td>
<td>26/12</td>
<td>118/4-13</td>
<td>114/10</td>
<td>Senning</td>
<td>—</td>
</tr>
<tr>
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<td>50</td>
<td>75</td>
<td>75†</td>
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<td>—</td>
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<tr>
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<td>23/12</td>
<td>130/0-3</td>
<td>132/0-5</td>
<td>Senning</td>
<td>—</td>
</tr>
<tr>
<td>4 (1964)</td>
<td>M 5</td>
<td>31/12</td>
<td>—</td>
<td>80/0</td>
<td>Mustard</td>
<td>—</td>
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<tr>
<td>5 (1964)</td>
<td>M 4</td>
<td>25/14</td>
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<td>89/3-8</td>
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<td>—</td>
</tr>
<tr>
<td>7 (1966)</td>
<td>M 4</td>
<td>43/28</td>
<td>—</td>
<td>85/1-12</td>
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<td>98/2-7</td>
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<td>9 (1967)</td>
<td>F 9</td>
<td>25/20</td>
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<td>Mustard</td>
<td>18</td>
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<tr>
<td>10 (1968)</td>
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<td>55/25</td>
<td>101/9</td>
<td>107/3</td>
<td>Mustard</td>
<td>—</td>
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</table>

*= no data available.
†Data obtained at surgery.
series of 114 with dextro- or levo-transposition reported in the same paper, 14 (39%) had stenosis at the pulmonary valve, 10 (28%) had stenosis in the subvalvular region, and 12 (33%) had stenosis in both valvular and subvalvular regions.

The total experience of the Mayo Clinic with repair of transposition with pulmonary stenosis comprises 23 patients operated on from 1960 through June 1968. These patients were divided into two groups according to the location of the stenosis: valvular or subvalvular (tables 1 and 2). All but one patient had an associated VSD. Before 1964, the Senning operation\(^9\) was carried out, and during and since that year the Mustard repair\(^10\) was performed.

Valvular Pulmonary Stenosis

Ten patients whose ages ranged from 10 months to 12 years (median, 4 years) were operated on. The pertinent data are summarized in table 1. There were three hospital deaths, a mortality rate of 30%. Preoperative pressure measurements showed that the stenosis was severe in five patients (cases 1, 3, 4, 5, and 9 of table 1), and mild or moderate in the rest. The pulmonary valve was approached usually through a longitudinal incision in the pulmonary artery, and in a few patients, its dilatation through the VSD was attempted. In each case, the VSD was closed either through a right ventriculotomy or through the tricuspid valve. At the end of the repair, all surviving patients but one (case 9) had left ventricular pressure (pulmonary ventricle) that was less than right ventricular pressure (systemic ventricle). Nonetheless, in most patients, there was residual stenosis across the pulmonary valve. In the three patients who were operated on early in the experience and who died, the cause of death was low cardiac output. Pathological examination of the hearts revealed that the stenosis had been relieved satisfactorily in two patients (cases 2 and 3) whereas in the third patient (case 1) an 8-mm opening was present in the pulmonary valve (fig. 1).

All the patients who survived the operation were well 5 months to 4 years after surgery.

Subvalvular Pulmonary Stenosis

Thirteen patients whose ages ranged from 3 months to 12 years (median, 7 years) were operated on. Pertinent data are summarized in table 2. There was no VSD in one patient (case 12, table 2). In the other patients, the VSD was usually large and was located in the vicinity of the cusps of the pulmonary valve in all but two patients (cases 1 and 3, table 2), each of whom had a hypoplastic left ventricular outflow tract. In these two patients, the VSD was about 2 cm from the pulmonary cusps, and there was no continuity between these cusps and the anterior leaflet of the mitral valve.\(^4\) In each case the VSD was closed.

The stenosis was severe in all patients, as shown by the preoperative measurement of the gradients between the left ventricle and the pulmonary artery. Although in eight patients there was associated pulmonary valve stenosis, the subvalvular stenosis in each of these patients was the major cause of obstruction to the left ventricular outflow.

**Figure 1**

Opened left ventricle and pulmonary artery of a patient (case 1, table 1) operated on for complete transposition of the great arteries, VSD, and pulmonary valvular stenosis. Left ventricular outflow tract is wide. VSD, which was repaired with a patch (asterisk), is located immediately beneath pulmonary valve, which is bicuspid.
Patients Operated on For Dextro-Transposition and Subvalvular Pulmonary Stenosis

<table>
<thead>
<tr>
<th>Case no. (yr)</th>
<th>Sex and age (yr)</th>
<th>Preoperative pressure (mm Hg)</th>
<th>Surgical repair</th>
</tr>
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</tr>
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<td>6 (1966)</td>
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<td>20/14</td>
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<td>11 (1968)</td>
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<td>31/16</td>
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<td>13 (1968)</td>
<td>M 10</td>
<td>19/16</td>
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</tbody>
</table>

Preoperative pressure (mm Hg): Pulmonary trunk, Left ventricle, Right ventricle.

*Data obtained at surgery.

There were eight hospital deaths, a mortality rate of 61%. The cause of death was related to low cardiac output. One patient (case 1) operated on with the Senning technique had, in addition, obstruction of the superior vena cava after the repair. In two patients (cases 2 and 11), complete atrioventricular block developed. Residual pulmonary stenosis was considered in most patients to be the major factor causing low cardiac output. Presence of residual stenosis is well documented by the pressure measurements after the repair, showing that a significant residual gradient across the pulmonary valve existed in those patients in whom it was measured (cases 4, 6, and 10), whereas in the other patients, the pressure in the left ventricle (pulmonary ventricle) was equal to (or higher than) that in the right ventricle (systemic ventricle).

Autopsy on all eight patients who died revealed that in each the relief of the subvalvular stenosis had been inadequate. The obstruction was located in the left ventricular outflow tract, but the type and nature of the obstruction varied. Five patients (cases 2, 7, 8, 11, and 12) had localized obstruction; the ventricular septum bulged into the cavity of the left ventricle. Three of these five patients (cases 2, 11, and 12), in addition, had a fibromuscular ridge located just beneath the cusps of the pulmonary valve (fig. 2). In two patients, these cusps were fused to the subvalvular ridge at the ventricular aspect. One patient (case 7) also had a parachute-like mitral valve with two orifices and an anterior leaflet with a large aneurysm-like formation that bulged and partially obstructed the left ventricular outflow tract (fig. 3). One patient (case 8), in addition, had valvular tissue.
that extended from the mitral valve to the right ventricle where it attached through the VSD (fig. 4). This patient, as well as two others (cases 1 and 9) (fig. 5), had some redundant fibrous tissue in the left ventricular outflow tract that contributed to its obstruction. Two patients (cases 1 and 3) had diffuse stenosis due to a hypoplastic left ventricular outflow tract and a narrow pulmonary ring (fig. 6). One patient (case 9) had severe abnormalities of the mitral valve, which were responsible for the left ventricular obstruction. This patient had a fibrous attachment of the anterior leaflet of the mitral valve to the anterior aspect of the left ventricular outflow tract (fig. 7 A). In addition, anomalous chordae tendineae from the anterior and posterior leaflets crossed the VSD and were attached to a papillary muscle into the right ventricle (fig. 7 B).

Every patient who survived surgery had a difficult postoperative period, and most of them required assisted ventilation and digitalization. Seven months to 4 years after operation, they were all much improved although they all had evidence of residual stenosis.

From the analysis of the operated patients, the following observations can be made. (1) A high mortality rate is associated with complete repair of transposition of the great vessels and subvalvular pulmonary stenosis. (2) The primary reason for this high rate is incomplete relief of the subvalvular stenosis; residual pulmonary stenosis is still present in the surviving patients. (3) Although the mortality rate after repair of transposition associated with either atrial septal defect or VSD, with or without pulmonary valve stenosis, has improved after the introduction of the operation described by Mustard, no such improvement has occurred in cases associated with subvalvular pulmonary stenosis.

<table>
<thead>
<tr>
<th>Pressure after repair (mm Hg)</th>
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<td>65</td>
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</table>
(4) The location of the obstruction in the left ventricular outflow tract makes a satisfactory surgical exposure impossible, owing to the posterior position of the left ventricle. This prevents adequate relief of the stenosis. It is difficult even to evaluate the degree of subpulmonary obstruction at surgery if the evaluation is made through the pulmonary valve. A more effective assessment of subvalvular obstruction can be made by finger palpation of this region through the VSD.

(5) Even if exposure were satisfactory, the nature of the obstruction would often defy surgical repair in those cases associated with a hypoplastic left ventricular outflow tract, a small pulmonary valve ring, and obstructive anomalies of the mitral valve. Patch enlargement of a hypoplastic left ventricular outflow tract or of a narrow pulmonary ring would be required, but this is technically impossible in most cases because the circumflex coronary artery is located in front of the left ventricular outflow tract.4 Interference with an obstructing mitral valve would result in insufficiency of this valve. (6) Resection of subvalvular obstruction can cause complete atrioventricular block, as it probably did in two of the 13 reported cases.

Thus, it is apparent that a better surgical technique is needed for repair in cases of transposition associated with subvalvular pulmonary stenosis. Because this stenosis seems to defy appropriate treatment, the surgical technique should avoid a direct approach to the left ventricular outflow obstruction by circumventing it.

The theoretical basis and a demonstration of a surgical procedure that would achieve complete repair by redirection of the ventricular outflows while bypassing the pulmonary stenosis have been given in another paper.12 On July 26, 1968, these principles were applied in the complete repair of one patient with transposition, pulmonary stenosis, and VSD.

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Figure 4

Opened left ventricle and pulmonary artery of a patient with dextrocardia and situs inversus viscerum (case 8, table 2) operated on for transposition, VSD, and subvalvular pulmonary stenosis. There is bulging of ventricular septum toward cavity of left ventricle. Redundant fibrous tissue resembling valvular tissue extends from mitral valve (asterisk) to right ventricle, where it enters through the VSD. In addition, there is redundant endocardial tissue in the form of a band or bottomless cusp (probe) contributing to obstruction of left ventricular outflow tract.

Figure 5

Opened left ventricle and pulmonary artery of a patient (case 1, table 2) operated on for transposition, VSD, and subvalvular pulmonary stenosis. VSD (arrow) was repaired by direct suture. Redundant endocardial tissue (asterisk) beneath pulmonary valve partially obstructs left ventricular outflow tract.

Figure 6

Opened left ventricle and pulmonary artery of a patient (case 3, table 2) operated on for complete transposition of the great arteries, VSD, and valvular and subvalvular pulmonary stenosis. Pulmonary valve was bicuspid. Underneath the valve is a long, hypoplastic, obstructing left ventricular outflow tract (arrow points to sharp ridge separating hypoplastic left ventricular outflow tract and sinus of left ventricle). Mitral valve is not in continuity with cusps of pulmonary valve, and VSD (asterisk) is located at a distance from pulmonary artery valve.

Report of Case

A 14½-year-old boy was first seen in 1968 at the Mayo Clinic to which he was referred for surgical correction of complete transposition of the great arteries, VSD, and pulmonary stenosis. A heart murmur had been present from birth, and cyanosis was noted at 6 months of age. Squatting commenced at 2 years of age, and one episode of anoxic loss of consciousness occurred at 7 years of age. At 8 years, an exploratory median sternotomy was performed and the diagnosis of transposition and pulmonary stenosis was made. Ten days later, a right subclavian artery-pulmonary artery anastomosis was created. When he was 8½ years, left subclavian artery-pulmonary artery anastomosis was performed, and since that
time, no squatting or anoxic spells had occurred. The patient usually became dizzy after climbing one flight of stairs but could walk about 10 blocks slowly.

Physical examination revealed a well-developed boy, 5 feet 4 inches tall and weighing 118 lbs. There was a moderate degree of generalized cyanosis, with clubbing of the fingers and toes. Hemoglobin value was 20 g/100 ml of blood and the hematocrit value was 63%. The heart was quiet. The first sound was normal in intensity, and there was a loud single second sound. A harsh holosystolic, grade III/VI, murmur was heard on the lower left sternal border, with a grade III/VI continuous murmur over the upper left sternal border. Blood pressure was 130/70 mm Hg, and heart rate was 90 beats per minute.

On the chest roentgenogram, the heart was mildly enlarged (cardiothoracic index, 0.55), the aortic arch was on the left side, and the pulmonary vascularity appeared increased. The electrocardiogram showed a wandering atrial pacemaker with normal QRS duration. The mean QRS axis in the frontal plane was \(+130^\circ\), and the frontal plane QRS vector loop was clockwise. There was evidence of right ventricular hypertrophy but not of left ventricular hypertrophy.

Data gathered during cardiac catheterization performed in February 1968 are summarized in table 3.

A biplane angiocardiogram taken after injection of contrast medium into the right ventricle demonstrated that the aorta arose directly from the right ventricle and was positioned anteriorly (fig. 8) and slightly to the left (fig. 9) with respect to the pulmonary artery. A left subclavian artery entering the left pulmonary artery was
visualized, and there was no evidence of patency of the right Blalock anastomosis. A left ventricular cineangiocardiogram (transseptal catheterization) taken after the injection of contrast medium into the left ventricle showed that the left ventricle emptied into an enlarged pulmonary artery whose valve was dome-shaped and exhibited a systolic jet. The subvalvular area could not be clearly visualized, and, therefore, subvalvular stenosis could not be assessed.

Surgical Repair

Operation was performed on July 26, 1968. A secondary median sternal incision was made, and the heart was freed from extensive pericardial adhesions. The left subclavian artery was dissected free near its origin and later ligated after institution of cardiopulmonary bypass. The aorta was anterior and slightly to the left of the pulmonary artery. The pulmonary artery was considerably smaller than the aorta. The right and left ventricles were enlarged and hypertrophied. The peak systolic pressure in the two ventricles was equal, being 100 mm Hg. The mean pressure in the pulmonary artery was 12 mm Hg.

Total cardiopulmonary bypass was instituted for 110 minutes at flow rates of 1.5 to 2.0 L/min/m² at 30 C. The left ventricle was vented. The pulmonary valve was inspected through a transverse incision in the main pulmonary artery. The valve was mildly stenotic owing to commissural fusion, and there was severe diffuse subvalvular stenosis, which was best assessed after right ventriculotomy was performed and the left ventricle was explored through the VSD. Because adequate relief of the pulmonary stenosis was not possible, it was decided to attempt a new type of repair.

The pulmonary artery was transected, and the proximal end was oversewn (fig. 10 A). A sump sucker was placed in the right pulmonary artery. Because of the large pulmonary venous return, the circulation was interrupted for one period of 8 minutes after the esophageal temperature had reached 30 C. The VSD was repaired with a low porosity Teflon patch which was inserted in such a way as to form a tunnel from the left ventricle to the aorta (fig. 10 B and C).

The right ventriculotomy was then enlarged by excision of a portion of the right ventricular wall around it. The resulting opening measured about 3 by 4 cm. The thickness of the wall surrounding this opening was thinned. A homograft was then selected for reconstruction of the pulmonary artery (fig. 10 D). The homograft had been preserved at −70 C and sterilized by irradiation. Its diameter at the aortic ring was 2.5 cm. After thawing, the graft was trimmed, preserving the anterior leaflet of the mitral valve. The origins of the innominate artery, the left carotid artery, and the coronary arteries of the homograft were oversewn. The aortic arch of the graft was then divided between the left carotid and the left subclavian arteries and anastomosed.
Figure 10

Drawings illustrating steps in surgical repair. A, Left Blalock anastomosis is ligated (upper left). Pulmonary valve, inspected through pulmonary arteriotomy, shows moderate fusion of cusps (upper right). Main pulmonary artery is divided, and proximal end is oversewn. Broken line indicates ventriculotomy. A = aorta; PA = pulmonary artery; RV = right ventricle; IVC =
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end-to-end to the main pulmonary artery near the bifurcation. Proximally, the homograft was anastomosed to the right ventricle along the edges of the enlarged ventriculotomy, using the anterior mitral leaflet of the graft as a gusset in the apical aspect of the anastomosis (fig. 10 E).

During the repair, the aorta was clamped for 75 minutes. Nevertheless, adequate coronary perfusion was maintained throughout this period because of the huge pulmonary venous return and the heart kept beating for most of this period. At the end of the repair, the heart was defibrillated and sinus rhythm resumed. After bypass was discontinued and hemodynamics were stabilized, systolic pressures in mm Hg were: 90 to 100 in the left ventricle, 65 in the right ventricle, 45 in the homograft, and 30 in the right pulmonary artery. Simultaneous cuff pressure measured in one leg was 120/90 mm Hg. Copious oozing from raw surfaces was controlled after considerable time, and only after bleeding subsided was the sternum reaproximated with wire sutures. The subcutaneous tissue and skin were closed and two drainage catheters were left, one in the pericardial space and one in the anterior mediastinum.

The postoperative period was completely uneventful, and digitalis was not required at any time. Exercise tolerance improved significantly, and on the tenth postoperative day, the patient walked briskly up six flights of stairs without tiring. The patient was dismissed on the fourteenth postoperative day, at which time he was not receiving any medication. The blood pressure was 120/80 mm Hg, with a pulse rate of 66 beats per minute and a respiratory rate of 18 per minute. The liver was not enlarged, the heart was quiet, and there was a distinct pulmonary second sound, a grade III/VI systolic murmur over the upper left sternal border, and a grade II/VI short diastolic murmur with musical quality. An arterial blood sample taken on the fourteenth postoperative day while the patient was breathing room air gave the following results: pH, 7.4; Pco2, 44 mm Hg; Pao2, 54 mm Hg, oxygen saturation, 91%, and hemoglobin, 11.8 g/100 ml of blood.

The patient was asymptomatic and living a normal life 3 months after surgery. A chest roentgenogram taken at this time showed a cardio-thoracic index of 0.50 and no calcification of the homograft.

Comment

A patient with transposition, VSD, mild valvular and severe subvalvular pulmonary stenosis underwent successful repair by use of a technique that reestablishes normal ventricular arterial relationships through interventricular redirection of the left ventricular outflow and homograft reconstruction of the pulmonary artery. At the end of bypass, the arterial pressure was 20 mm Hg higher than the left ventricular pressure, indicating the absence of obstruction to left ventricular outflow. The patient tolerated the procedure well, and the hemodynamics were normal throughout the postoperative period. The systolic murmur present after the operation might be a result of the small gradients existing across the suture lines of the homograft. Mild incompetence of the aortic valve or of the homograft valve might have been the basis for the diastolic murmur. Mild arterial desaturation (91%), still present on the fourteenth postoperative day, is often observed after bypass and is probably related to differences in ventilation-blood flow ratios in the lung, venous admixture resulting from perfusion of non-ventilated and airless alveoli, and possibly mild impairment of the oxygen diffusion capacity. The pliability and plasticity of the homograft facilitates suturing. Use of the anterior mitral valve leaflet of the homograft for completion of proximal anastomosis allows optimal function of the graft's valve. The operation is not technically difficult, and direct relief of pulmonary stenosis is not required in order to achieve complete repair. The repair results in the anatomic right ventricle's supplying the pulmonary circulation and the ana-

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inferior vena cava; SVC = superior vena cava. B, Drawing illustrates inside of right ventricle and relationship of ventricular septal defect (VSD), aortic valve, and tricuspid valve (TV). C, Teflon tunnel is constructed to connect left ventricle and aorta. D, Homograft of aorta, including aortic valve and anterior leaflet of mitral valve, is sutured to distal end of main pulmonary artery. Portion of anterior wall of right ventricle is excised around ventriculotomy. E, Proximal anastomosis is carried out, using homograft's mitral anterior leaflet to complete apical aspect of anastomosis.

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Anatomic left ventricle’s supplying the systemic circulation.

The long-term fate of aortic homografts is unknown. Calcification of the homografts used to repair truncus arteriosus defects has occurred in each of the five patients operated on at the Mayo Clinic since September 1967. This has not prevented continued function of these homografts. To this date, no calcification has been observed in the region of the valve. Reports from other centers on the fate of aortic homografts that have been in place for as long as 17 years in the thoracic and abdominal aorta indicate that the segments of aorta and aortic valves have stood up well to the challenge of time. Some segments of aorta, especially if used fresh, have not calcified. In the others, calcification is present, but it has not caused difficulty.

Concluding Remarks

Successful repair in a case of complete transposition of the great arteries associated with VSD and valvular and subvalvular pulmonary stenosis was carried out in a 14½-year-old boy. A new surgical technique that achieved redirection of the ventricular outflows was employed.

The rationale for seeking an alternate technique to transposition of the venous return as described by Mustard is the high surgical mortality rate with which transposition with subvalvular pulmonary stenosis is associated when complete repair with that technique is carried out. A review of the cases in which this technique was used indicates that the location and nature of the obstruction in the left ventricular outflow tract defy successful repair in most cases.

The success of the surgical repair described herein is possible because the technique does not require direct approach to the subvalvular stenosis but bypasses it. Anatomic as well as physiological repair is achieved because the anatomic right ventricle is made to empty into the pulmonary circulation, while the anatomic left ventricle empties into the systemic circulation.

Acknowledgment

We appreciate the cooperation of Drs. J. W. Kirklin and D. C. McGoon in allowing us to report on patients on whom they operated. We also are grateful to Dr. Ephraim Glassman for allowing us to publish hemodynamic and angiocardiographic data in table 3 of the catheterization study that he performed.

References

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Paradox in Prophecy

... Unfortunately, it is impossible to predict new ideas—the ideas people are going to have in ten years' or in ten minutes' time—and we are caught in a logical paradox the moment we try to do so. For to predict an idea is to have an idea, and if we have an idea it can no longer be the subject of a prediction. ...

Most people feel more confident in denying that certain things will come to pass than in declaring that they can happen or surely will happen. Many a golden opportunity to remain silent has been squandered by anti-prophets who do not realize that the grounds for declaring something impossible or inconceivable may be undermined by new ideas that cannot be foreseen.—P. B. Medawar: The Art of the Soluble. London, Methuen & Co. Ltd., 1967, p. 99; also distributed by Barnes & Noble, Inc., New York.
Complete Repair of Transposition of the Great Arteries with Pulmonary Stenosis: A Review and Report of a Case Corrected by Using a New Surgical Technique
G. C. RASTELLI, ROBERT B. WALLACE and PATRICK A. ONGLEY

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