A New Technic for Complete Correction of Transposition of the Great Vessels

An Experimental Study with a Preliminary Clinical Report

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COMPLETE TRANSPOSITION of the great vessels is a common congenital cardiovascular anomaly. In this disease the aorta with its coronary arteries originates from the right ventricle, and the pulmonary artery arises from the left ventricle. Consequently, the aorta carries venous blood; the pulmonary artery, oxygenated blood.

Surgical correction of this malformation has been beset with many difficulties and remains a challenge to the cardiac surgeon. A logical method for correcting this anomaly is retransposing the aorta and pulmonary artery. A major technical difficulty in this approach has been transfer of the coronary arteries. Obviously, if they are left in place after transplating the major vessels, the myocardium will continue to be supplied with venous blood.

Experimental Work

From a study of autopsy heart specimens with transposition of the great vessels, the following observations were made: The right and left coronary arteries arise from the posterior sinuses of the aorta (fig. 1, nos. 3 and 4), approximately 5 to 7 mm. above the bottom of the aortic sinuses. The proximal portion of the coronary artery segments is not embedded in the myocardium as is usual in the normal heart. At their origin the coronary arteries are separated from the myocardium by a small triangular space filled with fat and areolar tissue, which facilitates their dissection.

These observations led us to develop a technic for switching the pulmonary artery and the aorta with its associated coronary arteries. This technic depends on the basic maneuver of creating an isolated aortic segment containing the ostia of both coronary arteries. This segment is then turned over and sutured proximally to the left ventricular outlet (fig. 2, nos. 6 to 8) and distally to the remaining aorta (fig. 2, no. 9).

The procedure was performed on 19 dogs with use of extracorporeal circulation. Obviously, no survivals would be expected in normal dogs when the switch-over is performed. Nevertheless, when the dogs were sustained by extracorporeal circulation at the termination of the operation their heart beats continued strong and rhythmic. The myocardium invariably remained oxygenated and pink, and the coronary circulation appeared functionally intact. Figure 3 is a roentgenogram of a dog's heart upon which the procedure was performed. The contrast material (Micropaque in gelatin solution) was injected into the aorta, demonstrating the complete filling of the coronary arteries.

The arterial switch-over was performed post mortem and in situ on several patients that died of transposition of the great vessels. Then two clinical trials were carried out.

Technic

To decrease the chances of confusion, we are giving technical details of this procedure as applied to the human heart with transposition of the great vessels. Figures 1 and 2 depict the operation as it was performed in our two clinical trials. The procedure as originally developed on dogs is slightly different from the one used in patients that is described below.

1. The chest is entered via a longitudinal sternum-splitting incision.

2. The thymus is excised and the pericardium is opened widely.

3. Umbilical tapes are placed around the superior and inferior venae cavae and the femoral artery in preparation for circulatory bypass.

4. The ascending aorta is dissected distally to the level of the ligamentum or ductus arteriosus. Proximally, the root of the aorta is freed from the fat and thin epicardium covering it.
No. 1. External anatomy of a heart with transposition of the great vessels. No. 2. The dissection of the aorta, pulmonary artery, and left coronary artery is completed. The scissors are clearing the triangular space between the proximal portion of the right coronary artery, aorta, and myocardium. No. 3. The aortic clump is in place and the first incision is being completed. The interrupted line demonstrates the site of the second incision. No. 4. The second incision is being completed. No. 5. The aortic segment containing both coronary ostia is shown. The segment is pulled to the right to demonstrate the outflow stumps and valves of the left and right ventricles.
No. 6. Beginning of the first anastomosis between the isolated aortic segment and the outflow stump of the left ventricle. No. 7. Lateral view of the heart and great vessels demonstrating how the aortic segment is turned over to fit the outflow stump of the left ventricle. No. 8. The first anastomosis being completed. No. 9. The first anastomosis completed and the second anastomosis started. Notice that the aortic segment is completely turned upside down. No. 10. The continuity of the systemic circuit is completed. The aortic clamp is removed. The pulmonary artery is pulled over and sutured to the outflow stump of the right ventricle. No. 11. The switch-over is completed.
This second incision completes the creation of an isolated aortic segment, containing the ostia of both coronary arteries. The average length of this segment is about 8 mm.

11. The pulmonary artery is transected about 1 mm, distal to the valve commissures (fig. 1, no. 5).

Care should be taken not to injure the valves when the pulmonary artery is transected. These valves ultimately become systemic. The length of the proximal pulmonary artery stump (outflow stump of the left ventricle) is of considerable importance. Too long a stump favors the production of regurgitation; too short a stump may injure the valve leaflets.

12. With the coronary arteries used as the axis, the aortic segment is turned over to fit the left ventricular outflow stump (fig. 2, no. 7). An anastomosis is then performed between the aortic segment and the left ventricular stump with continuous 5-0 silk suture (fig. 2, nos. 6, 7, and 8).  

13. The second anastomosis is then performed between the aortic segment and the distal aorta (fig. 2, no. 9), so as to restore continuity of the systemic circuit with the left ventricle.

14. The aortic clamp is then removed, allowing the coronary arteries to be perfused and the heart to resume contractions. The duration of anoxic arrest is limited to an average of 30 to 35 minutes.

Because of heparinization, bleeding from the anastomotic site is a major problem. Metastatic placement of the sutures helps to prevent excessive bleeding. Gelfoam moistened with topical bovine thrombin placed over the suture line aids in stopping bleeding from needle holes. When bleeding is excessive, thrombin is washed out and becomes ineffective. We found it advantageous to cross-clamp the aorta for 2 to 3 minutes so that clotting may occur.

15. The pulmonary artery is anastomosed to the outflow stump of the right ventricle (fig. 2, no. 10), to complete the switch-over (fig. 2, no. 11).

16. Circulatory bypass is stopped. 

Due to the anatomic differences between the dog's heart with normal outflow tracts and the human heart with transposition of the great vessels, the procedure described above is slightly different from the one originally developed on dogs. The coronary arteries of the normal dog's heart are embedded in the myocardium. They are more difficult to dissect than in the human heart with transposition of the great vessels. Furthermore, the left coronary artery is covered by the pulmonary artery. We found it advantageous in dogs to perform as much as possible of the dissection and then to start bypass and transect the pulmonary artery. This maneuver facilitates the exposure and the completion of the dissection of the left coronary artery.
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Regurgitation was a frequent occurrence after completion of the switch-over in dogs. A single suture placed as shown in figure 4 helps to control any valvular insufficiency. Regurgitation was not encountered in the clinical trials.

Case Reports

Two patients with transposition of the great vessels were operated upon at The Children's Memorial Hospital in Chicago with use of this procedure.

Case 1

J. S. was a 7-year-old white boy with severe cyanosis since birth and severe clubbing of the digits. Clinical diagnosis of transposition of the great vessels was made soon after birth. Six months prior to surgery there was progressive clinical deterioration with decreasing exercise tolerance. Cardiac catheterization then confirmed the diagnosis of transposition. A bidirectional shunt at the atrial level and an intact ventricular septum were found. The pressure in the right ventricle was 108/8 mm. Hg, in the aorta 108/80 mm. Hg, in the left ventricle 37/8 mm. Hg, and in the pulmonary artery 37/15 mm. Hg. The arterial saturation was 69 per cent. The estimated pulmonary blood flow was 7.4 L./M.²/minute and the systemic blood flow 5.9 L./M.²/minute. The electrocardiogram demonstrated right ventricular and right atrial hypertrophy. The chest roentgenogram showed cardiac enlargement and increased pulmonary vascularity. A cineangiogram corroborated the diagnosis of transposition of the great vessels.

After adequate digitalization, the operative procedure described was performed on June 29, 1960. After the continuity of the aorta with the left ventricle was established, the aortic clamp was removed and the heart resumed its beats spontaneously. The heart contractions were strong and rhythmic, and the myocardium was uniformly pink. The electrocardiogram showed no evidence of coronary insufficiency at that time. The pulmonary artery anastomosis was then performed, and the interatrial septal defect was closed with a continuous suture through a right atriotomy.

The extracorporeal circulation was then stopped. The heart continued normal contractions for a few minutes, then started to slow down and the left ventricle became markedly distended. Cardiopulmonary bypass was resumed, and the heart rate increased and the cardiac contractions again became vigorous. There was no evidence of aortic valve regurgitation. Stopping and resuming bypass several times reproduced the same sequence of events; good cardiac contractions resulted on bypass, and weakening of the contractions with left ventricular dilatation occurred when off bypass.

The sutured interatrial defect was reopened with the hope that it would decompress the left side of the heart, but to no avail. The patient died of acute left ventricular dilatation and failure. Postmortem examination showed a thin-walled and dilated left ventricle. The right ventricular wall was markedly hypertrophied, three times the thickness of the left ventricular wall. The coronary arteries and ostia were patent to probing.

Comment

In retrospect, this patient may not have been a suitable candidate for this procedure. The age and size of this patient and his general satisfactory condition coupled with the fact that he had an intact ventricular septum were strong factors in our choice. Although the left ventricular pressure was low, it was anticipated that the left ventricular myocardium could support the pressure postoperatively, since it had been carrying a considerably increased flow load. We now think that for this type of procedure, the left ventricular pressure should approximate or equal the right ventricular pressure.

Case 2

M. B. was a 3½-month-old white girl, cyanotic since birth. The diagnosis of transposition of the great vessels was considered clinically, and it was then confirmed by cineangiogram and cardiac catheterization. A left-to-right shunt at the atrial level, a patent ductus arteriosus, and an intact interventricular septum were found. The left ventricular pressure was 52/0 to 5 mm. Hg, the right ventricular pressure 62/5 mm. Hg, the aortic pressure 62/38 mm. Hg. The arterial saturation was
67 per cent. Chest roentgenogram showed 2+ cardiac enlargement and increased pulmonary vascularity.

The operation was performed on August 17, 1960, and the procedure as described above was done. Since this patient was an infant, we thought that we should interrupt the suture line at several points in the anastomosis to prevent any interference with the circumferential growth of the major vessels. At the completion of the second anastomosis, the aortic clamp was removed and the heart resumed its beats spontaneously. Excessive bleeding from the anastomosis compelled us to reclamp the aorta and try to control the bleeding. We also elected to perform the pulmonary artery anastomosis at this time while we had a dry field. The aortic clamp was removed and again the heart resumed its beats. Bleeding from the anastomotic sites continued to be excessive and could not be controlled. Although Gelfoam soaked in topical bovine thrombin had previously been very effective in controlling bleeding, it did not have any clotting effect in this case.

A vicious circle resulted from the bleeding. The pump had to be transfused rapidly with large amounts of cold blood. This dropped the temperature of the infant to 29 C. Since we were using a radiant heating unit against the oxygenator, warming of the blood was not sufficiently effective.

Circulatory bypass was stopped, and protamine sulfate was given intravenously but it did not control the bleeding. The heart rate was slow from the effect of cooling. The left ventricle showed no sign of dilatation. The bleeding continued, the aorta became flabby, and the patient died from acute blood loss. Postmortem examination showed the right and left ventricles to be of almost equal size. The coronary ostia and arteries were patent to probing. Figure 5 shows a view of the left ventricle with the outflow tract in the postmortem specimen. Two probes indicate the coronary ostia.

Comment

In this case, bleeding appeared to be the cause of failure.

Discussion

This work demonstrates that switching the pulmonary artery and aorta with both coronary arteries is technically feasible and appears to be practical in correcting transposition of the great vessels. The basic features that make it possible to transfer both coronary arteries with the aorta are the intact circular aortic segment and the "turning over" maneuver. The intact aortic segment acts in reality as a graft lengthening the coronary arteries. This lengthening obviates the difficulty associated with the individual transfer of the small coronary arteries. Since the coronary ostia in transposition of the great vessels are usually on the side of the aorta adjacent to the pulmonary artery, the only way to transfer this segment of the aorta containing both coronary arteries is by turning the segment over to fit the outflow stump of the left ventricle. This maneuver does not interfere with coronary circulation. Adequacy of the coronary circulation was demonstrated by the uniform presence of good heart action in the dog experiments and the roentgenogram of the postoperative injection study of a dog's heart (fig. 3). The experience with the clinical trials further demonstrates sufficiency of the coronary circulation as evidenced by good filling of the coronary arteries, the uniformly oxygenated myocardium, the strong myocardial contractions, and the absence of coronary insufficiency pattern in the electrocardiogram during the bypass.

Our experience with the first case demonstrates the importance of patient selection. This first patient died because the left ventricle was unable to handle the sudden load imposed on it. A left ventricle adequate to cope with a work load comparable to that pre-

Figure 5

Postmortem specimen of case 2 showing a view of the left ventricle in continuity with the aortic segment and aorta. The two aortic suture lines are seen. Two probes point to the coronary ostia.
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sent by the systemic circuit seems to be a prerequisite to the success of this operation. Young infants with equally developed right and left ventricles and older children with associated ventricular septal defects and high left ventricular pressure would probably be the best candidates for the arterial switching procedures.

A high percentage of children with transposition of the great vessels succumb in the first few months of life. Many of them are without associated ventricular septal defects. We think that this operation may provide the best opportunity for salvage of such infants.

The question of repairing associated defects at the time of arterial correction remains to be answered. Patent ductus arteriosus must be closed and preferably divided and sutured to permit complete mobilization of the pulmonary artery. We do not plan now to close the ventricular septal defects in subsequent cases.

Summary

A procedure for complete anatomic correction of transposition of the great vessels is described. The technic is based on the concept of isolating an aortic segment containing both coronary ostia and turning the segment over to fit the outflow stump of the left ventricle. Experimental work and two clinical trials are presented and discussed.

We believe that this operation provides an anatomic and physiologic correction of the great vessels and coronary arteries in transposition. In spite of two clinical failures, we think this operation is worthy of further trials.

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