Correction of Congenital Cardiac Defects

Utilizing the Pump Oxygenator*

THE INTERIOR of the human heart, the last anatomic frontier confronting the surgeon, is no longer an area inaccessible to surgical correction of congenital or acquired defects. General hypothermia was first used by Lewis in 1952, to successfully close an atrial septal defect. Gibbon* pioneered the application of a mechanical heart and lung apparatus to cardiac surgery and in 1953-54, he successfully closed an atrial septal defect in an eighteen-year-old girl utilizing total cardio-respiratory bypass.

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reservoir of arterialized blood† and in 15 patients by a biologic (dog lung) oxygenator.§ In 1955 the University of Minnesota group introduced a simple disposable bubble oxygenator# which provided adequate flow and gaseous exchange. This safe and effective pump oxygenator system has since been utilized in various centers throughout the world. Because of previous experience with this type pump oxygenator, we elected to use this apparatus in our initial clinical cases. The following seven patients represent our initial clinical experience at the Children's Memorial Hospital with correction of congenital cardiac defects utilizing total cardio-pulmonary bypass.

Case Presentations

Patient Number One: A.J.A. (Wilburton, Oklahoma).

Referring Physician: C. K. Holland, Jr., M.D. (McAlester, Oklahoma).

This twelve-year-old boy was noted to have a heart murmur in the spring of 1957. There was a history of exertional dyspnea

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and decreasing exercise tolerance for the past year. Physical examination revealed a somewhat underdeveloped boy with no evidence of cyanosis. There was a grade three ejection systolic murmur at the upper left sternal border and a fixed, split second sound. There was also a grade three mid-diastolic rumbling murmur maximal at the lower left sternal border. X-ray examination revealed marked increase in cardiac transverse diameter, mainly due to right ventricular enlargement. The main pulmonary artery segment was enlarged and the pulmonary vascular markings were accentuated. The electrocardiogram revealed right axis deviation, incomplete right bundle branch block, and probably right ventricular hypertrophy. In November 1958, he was admitted to Children's Memorial Hospital for cardiac catheterization. This study revealed a large left to right shunt through an atrial septal defect and anomalous pulmonary venous drainage from the right lung. Pulmonary blood flow was approximately four times the systemic blood flow and the right ventricular pressure was slightly elevated. On 1-21-59, the congenital defects were corrected under direct vision utilizing total extracorporeal circulation. The chest was entered through a bilateral anterior thoracotomy in the fourth interspace with a transverse sternotomy at that level. On opening the pericardium, the right atrium was seen to be approximately two and one-half times normal size and the pulmonary artery was much enlarged. After establishing total cardiac bypass the right atrium was opened and the defect in the atrial septum was found to be low and just posterior to the coronary sinus. There was a fair margin of atrial septal wall between the lowest aspect of the defect and the annulus of the atrio-ventricular valves. Both the right superior and right inferior pulmonary veins entered to the right of the atrial defect. The septal defect was closed with interrupted silk sutures and positioned in such a fashion as to divert the right pulmonary venous drainage into the left atrium. His immediate postoperative course was uncomplicated; however, three weeks following operation he developed "post-commisurotomy" syndrome characterized by fever, arthralgia, anorexia, and tachycardia. He responded to treatment with bedrest and salicylates and was discharged home on 3-21-59.

**Patient Number Two: M. F. (Bristow, Oklahoma).**

**Referring Physician: D. L. McAlister, M.D. (Bristow, Oklahoma).**

This five-year-old boy was first admitted to the Children's Memorial Hospital at the age of ten months because of a loud systolic murmur at the second interspace and frequent upper respiratory infections. Physical examination revealed a well-developed boy with malar flush and redness of the fingertips but no obvious cyanosis. There was a grade four stenotic systolic murmur maximal at the pulmonary area and a diminished pulmonary second sound. X-ray examination revealed slight cardiac enlargement with right ventricular contour. The pulmonary artery segment was prominent, and the pulmonary vasculature diminished. Electrocardiogram showed marked right ventricular hypertrophy and minimal right atrial hypertrophy. He was followed in our congenital heart clinic until 11-7-58, when he was admitted for cardiac catheterization which revealed pure pulmonary valvar stenosis of marked severity with a right ventricular systolic pressure of 160 mm Hg. On 2-4-59, corrective surgery was performed utilizing cardiac bypass. The heart was exposed through a midline sternotomy incision. The pulmonary artery showed typical post-stenotic dilatation. A harsh thrill was present both over the main pulmonary artery and over the outflow tract of the right ventricle. The pulmonary valve could be palpated through the soft pulmonary artery wall. After establishing total extracorporeal bypass, the main pulmonary artery was opened, revealing a valve orifice measuring 3 mm in diameter. One of the commissures was partially open but the other two were completely fused. Under direct vision, each of the three commissures was incised out to the valve ring. No infundibular obstruction was present. The incision in the main artery was closed with running arterial silk suture. After completion of the intracardiac procedure, the right ventricular pressure was normal. Hg.

**Patient One**

**Referees**

(A. Th.)

The present paper deals with two cases of congenital heart disease seen at Children's Memorial Hospital. The first patient was a five-year-old boy who was first admitted to the hospital at the age of ten months because of a loud systolic murmur at the second interspace and frequent upper respiratory infections. The physical examination revealed a well-developed boy with malar flush and redness of the fingertips but no obvious cyanosis. There was a grade four stenotic systolic murmur maximal at the pulmonary area and a diminished pulmonary second sound. The electrocardiogram showed marked right ventricular hypertrophy and minimal right atrial hypertrophy. He was followed in our congenital heart clinic until 11-7-58, when he was admitted for cardiac catheterization which revealed pure pulmonary valvar stenosis of marked severity with a right ventricular systolic pressure of 160 mm Hg. On 2-4-59, corrective surgery was performed utilizing cardiac bypass. The heart was exposed through a midline sternotomy incision. The pulmonary artery showed typical post-stenotic dilatation. A harsh thrill was present both over the main pulmonary artery and over the outflow tract of the right ventricle. The pulmonary valve could be palpated through the soft pulmonary artery wall. After establishing total extracorporeal bypass, the main pulmonary artery was opened, revealing a valve orifice measuring 3 mm in diameter. One of the commissures was partially open but the other two were completely fused. Under direct vision, each of the three commissures was incised out to the valve ring. No infundibular obstruction was present. The incision in the main artery was closed with running arterial silk suture. After completion of the intracardiac procedure, the right ventricular pressure was normal. Hg.
was measured and found to be only 50 mm. Hg. Postoperatively, the patient did exceptionally well and was discharged from the hospital on 2-26-59.

**Patient Number Three: R.F.T. (Fox, Oklahoma).**

Referring Physician: C. E. Baker, M.D. (Oklahoma City, Oklahoma).

This seven-year-old girl was admitted to Children’s Memorial Hospital on 2-17-59. A precordial murmur had been noted at the age of one and one-half years and her development had been poor with frequent upper respiratory infections and perioral cyanosis with crying. Physical examination revealed a small girl with no evidence of right to left shunting. There was a grade three stenotic murmur maximal at the pulmonary area and a diminished pulmonary second sound. X-ray examination revealed slight right ventricular enlargement and a prominent main pulmonary artery segment. The electrocardiogram showed right axis deviation and moderate right ventricular hypertrophy. Cardiac catheterization on 2-4-59, showed a right ventricular systolic pressure of 100 mm.Hg. and a mean pulmonary artery pressure of 10 mm.Hg. Angiograms showed pulmonary valvar stenosis with no evidence of a right to left shunt. Corrective surgery was performed on 2-18-59. The pulmonary artery was incised so that the pulmonary valve could be visualized directly. The stenosis was completely relieved by a direct incision of the fused cusps. Following this procedure, right ventricular pressure fell to 25 mm.Hg. The patient’s postoperative course was completely uneventful and she was discharged home on 3-12-59.

**Patient Number Four: R.G. (Ardmore, Oklahoma).**

Referring Physician: R. W. Murphy, M.D. (Ardmore, Oklahoma).

This 4½-year-old boy had partially compensated congestive heart failure and frequent bouts of severe pneumonitis. He was markedly underdeveloped and very limited in activity. Physical examination revealed a small boy with no evidence of cyanosis. Physical examination showed moderate hepatosplenomegaly, venous engorgement and tachypnea as evidence of congestive failure. He had a combined hyperdynamic cardiac impulse. There was a grade three systolic ejection murmur at the pulmonary area with a fixed, split second sound. There was also a grade three regurgitation systolic murmur at the apex which transmitted out into the axilla. X-ray examination revealed marked cardiac enlargement with a prominent pulmonary artery segment and pulmonary plethora. The electrocardiogram showed left axis deviation, incomplete right bundle branch block and combined ventricular hypertrophy. Cardiac catheterization on 2-17-59, revealed an atrial septal defect of the low or primum type and evidence of mitral regurgitation, probably due to a cleft mitral valve. On 2-25-59, surgical correction of his defects was carried out on a total cardiac bypass. The heart was exposed through a bilateral transternal thoracotomy. On opening the pericardium the right atrium was found to be approximately three times normal size. After total extracorporeal bypass was established a right atriotomy was made and a large volume of bright red blood appeared in the operative field. This bleeding was due to regurgitation from the left ventricle through the cleft in the mitral valve and into the right atrium via the atrial defect. To facilitate repair, cardiac arrest was accomplished by cross clamping the ascending aorta. Two defects were then visualized in the atrial septum. One defect was a classical ostium primum type and measured 2 cm. in diameter. The second defect was at the foramen ovale level and measured 1 cm. in diameter. The foramen ovale defect was closed with interrupted silk sutures. The cleft in the anterior leaflet of the mitral valve was exposed and repaired through the primum defect. The primum defect was then closed with interrupted silk sutures which were tied over a strip of compressed polyvinyl sponge. The ascending aortic clamp was then released and normal sinus rhythm returned. Blood samples obtained from the superior vena cava and from the right ventricle after correction of the defects showed no residual left to right shunting. This child’s postoperative...
course was uneventful until 3-6-59, when he developed a pneumothorax on the right side. He responded to drainage by chest suction and antibiotics and is now ready for discharge.

**Patient Number Five: V.W. (Midwest City, Oklahoma).**

**Referring Physician: V. M. Rutherford, M.D. (Midwest City, Oklahoma).**

This nine-year-old girl was noted to have a heart murmur and anemia on a preschool examination at the age of five years. She showed poor development and had exertional dyspnea and decreased exercise tolerance. Physical examination revealed a poorly developed girl with no cyanosis. There was a combined hyperdynamic cardiac impulse. There was a grade three systolic ejection murmur at the pulmonary area with a fixed, split second sound. There was also a grade three systolic regurgitant murmur at the apex radiating out into the axilla. X-ray examination revealed marked cardiac enlargement with a prominent main pulmonary artery segment and pulmonary plethora. The electrocardiogram showed left axis deviation, incomplete right bundle branch block and combined ventricular hypertrophy. Cardiac catheterization on 2-16-59 revealed a large left to right shunt through a low atrial septal defect. A diagnosis of an ostium primum type atrial septal defect with mitral regurgitation due to a cleft mitral valve was made. On 3-4-59, an open heart operation was performed. The heart was exposed through a bilateral transsternal thoracotomy incision. The heart was markedley enlarged and had a paradoxical beat due to systolic regurgitation of blood from the left ventricle across the atrial septal defect into the right atrium. The aorta was relatively small and the pulmonary artery quite large. Operative correction of the defects was carried out in a fashion similar to that of patient number four. Blood samples from the superior vena cava and right atrium after repair showed no residual left to right shunt. This patient has had an uneventful postoperative course and is now ready for discharge.

**Patient Number Six: P.S. (Oklahoma City, Oklahoma).**

**Referring Physician: C. F. Foster, Jr., M.D. (Oklahoma City, Oklahoma).**

This six-year-old girl was first admitted to Children's Memorial Hospital on 1-25-54, at the age of one year with anasarca, precordial murmur, and a hemoglobin of 4 mg.%. She responded to treatment consisting of blood transfusions, anticongestive measures, and antibiotics. A grade four systolic stenotic murmur at the upper left sternal border persisted, however, after correction of her anemia, and a diagnosis of congenital heart disease was made. She was then followed in our congenital heart clinic until 1959, when she was admitted for further cardiac studies. At this time physical examination revealed a well-developed little girl with a malar flush and red fingertips but no evidence of cyanosis. The cardiac murmur was unchanged from previous examinations. The pulmonary second sound was diminished. X-ray examination revealed slight cardiac enlargement with right ventricular contour. The main pulmonary artery segment was not remarkable and the pulmonary vasculature slightly diminished. There was a right aortic arch. On 3-3-59, cardiac catheterization and angiocardiography was performed. These studies revealed a severe infundibular and valvar pulmonary stenosis and a ventricular septal defect. The right ventricular systolic pressure was 110 mm. Hg. There was a very small right to left shunt at the ventricular level. The systemic arterial saturation was 90%. On 3-11-59, open correction of her defects was carried out utilizing the pump oxygenator. The heart was exposed through a mid-sternotomy incision and upon opening the pericardial cavity the aorta was seen to be somewhat anterior and larger than normal. The main pulmonary artery was small, measuring approximately 8 mm. in diameter. The left and right pulmonary arteries, however, were of normal size. After establishing cardiac bypass a right ventricular cardiotomy was made in the outflow tract of the right ventricle. To facilitate correction, cardiac arrest was performed by cross clamping the ascending aorta. The ventricular septal defect was closed with numerous interrupted arterial silk sutures which were tied over a compressed polyvinyl sponge. After closure
of the ventricular defect the clamp on the ascending aorta was released. No left to right shunting at the ventricular defect was noted. Cardiac rhythmicity returned to normal spontaneously. The obstructing infundibular muscle at the level of the crista supraventricularis was then excised. In order to correct the valvar stenosis cardiac arrest was again carried out and the main pulmonary artery incised. The stenotic valve was opened to the wall of the artery along its three commissures. The pulmonary artery incision was then closed with a running arterial suture. The aortic clamp was then released and normal sinus rhythm returned after a brief episode of ventricular fibrillation. Right ventricular systolic pressure after repair was 50 mm/Hg. The patient awakened immediately at the end of the procedure and her postoperative convalescence has been totally uneventful.

Patient Number Seven: S.N. (Talihina, Oklahoma).

Referring Physician: D. E. Bendingfield, M.D. (Talihina, Oklahoma).

This seven-year-old Indian girl was noted to have a heart murmur at the age of one year and had been smaller for her age than any of her six siblings. Physical examination revealed a grade three stenotic systolic murmur maximal at the pulmonary area and a diminished pulmonary second sound. There was no evidence of cyanosis. X-ray examination revealed a heart with right ventricular contour and a normal pulmonary segment. The electrocardiogram showed an indeterminate axis and moderate right ventricular hypertrophy. She was admitted to the Children’s Memorial Hospital on 3-8-59, at which time a cardiac catheterization revealed valvar pulmonary stenosis with no evidence of left to right or right to left shunting. The right ventricular pressure was at systemic levels being 100 mm.Hg. On 3-18-59, open correction of the pulmonic valvar stenosis was carried out utilizing the pump oxygenator. The heart was exposed through a midline sternotomy incision. The pulmonary artery was somewhat enlarged but did not show a great deal of post-stenotic dilatation. After establishing total bypass the main pulmonary artery was opened and the pulmonary valve orifice visualized directly. It was 5 mm. in diameter. In order to minimize blood loss, cardiac arrest was established by aortic cross clamping. The stenotic valve was then opened all the way to the valve ring along its commissures. The surgeon’s finger was then introduced into the outflow tract in the right ventricle through the pulmonary valve. No infundibular stenosis was present. In order to be completely sure that there was not an associated small ventricular septal defect, a right ventricular cardiotomy was performed. Excellent visualization of the ventricular septum was obtained and no defect was found. The aortic clamp was released and cardiac rhythmicity spontaneously returned to normal. Right ventricular systolic pressure after correction was only 30 mm/Hg. The patient’s postoperative course thus far has been completely uneventful.

Discussion

We believe our complete lack of mortality and our very low postoperative morbidity rate is unique for a center’s initial clinical experience with cardiac bypass. Our success is, in large measure, due to the previous experience of our surgical team with this technique at other centers. The work here has also been greatly facilitated by the close teamwork between the various departments and groups involved, e.g., Surgery, Medicine, Pediatrics, Radiology, Anesthesiology, the Blood Bank, and the Nursing Service. The Blood Bank group under Dr. DeWitt Hunter has provided outstanding assistance in obtaining and providing adequate quantities of very fresh blood for each procedure.

Table One summarizes our experience to date with correction of congenital heart defects by cardiac bypass techniques at the Children’s Memorial Hospital. As is listed, all patients have survived their corrective procedures and as near as can be determined clinically, all have been totally corrected. The postoperative morbidity has been practically nil. No patient has had congestive heart failure, and no patient has had any significant pulmonary difficulty in the immediate postoperative period.

Table Two is a summary of our experi-
### TABLE I—CLINICAL DATA AND RESULTS (0% Mortality)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age—(Yrs.)</th>
<th>Weight (Pounds)</th>
<th>Diagnosis</th>
<th>Surgical Indications</th>
<th>Postoperative Course</th>
<th>Post-correction Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>76</td>
<td>A.S.D. 2&quot; Anomalous Drainage RL. P.V.</td>
<td>Progressive Exercise Intolerance. QP/QS = 4/1</td>
<td>&quot;Post-Commissurotomy&quot; Syndrome</td>
<td>S.V.C. &amp; Saturation = R.V. &amp; Saturation</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>46</td>
<td>P.S.</td>
<td>R.V. Sys. Pressure of 160 mm. Hg QS unchanged with exercise</td>
<td>Uneventful</td>
<td>R.V. Sys. Pressure 50 mm. Hg</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>41</td>
<td>P.S.</td>
<td>R.V. Sys. Pressure of 160 mm. Hg</td>
<td>Uneventful</td>
<td>R.V. Sys. Pressure 25 mm. Hg</td>
</tr>
<tr>
<td>4</td>
<td>4½</td>
<td>26</td>
<td>A.S.D. 1&quot; M.R. A.S.D. 2&quot;</td>
<td>Congestive Heart Failure QP/QS = 3/1</td>
<td>Pneumonia Pneumothorax</td>
<td>S.V.C. &amp; Saturation = R.V. &amp; Saturation</td>
</tr>
<tr>
<td>5</td>
<td>9</td>
<td>51</td>
<td>A.S.D. 1&quot; M.R.</td>
<td>Decreased Exercise Tolerance, QP/QS = 4/1</td>
<td>Uneventful</td>
<td>S.V.C. &amp; Saturation = R.V. &amp; Saturation</td>
</tr>
<tr>
<td>7</td>
<td>7</td>
<td>46</td>
<td>P.S.</td>
<td>R.V. Sys. Pressure of 160 mm. Hg</td>
<td>Uneventful</td>
<td>R.V. Sys. Pressure 30 mm. Hg</td>
</tr>
</tbody>
</table>

**Index of Abbreviations:**
- A.S.D. 2" — Atrial Septal Defect, Secundum Type
- A.S.D. 1" — Atrial Septal Defect, Primum Type
- M.R. — Mitral Regurgitation
- P.S. — Pulmonary Stenosis
- P.V. — Pulmonary Veins
- QP — Pulmonary Blood Flow
- QS — Systemic Blood Flow
- R.V. — Right Ventricle
- S.V.C. — Superior Vena Cava
- Sys. — Systolic
- V.S.D. — Ventricular Septal Defect

...}

ence with perfusion in these seven patients. The average perfusion rate has been 1850 ml/min/m². No postperfusion acidosis has been noted and no significant postoperative arrhythmia or heart block has occurred.

The use of an open technique is mandatory for the repair of atrial septal defects of the primum type (patients 4 and 5) and for ventricular septal defects (patient 6). Partial anomalous pulmonary venous drainage (patient 1) can be corrected by closed methods, but is done with much greater accuracy by the open method. Valvar pulmonary stenosis (patients 2, 3 and 7) can also be done satisfactorily by closed methods, but we think that here also repair under direct vision is superior for most patients. The open technique also has the advantage of allowing the surgeon to deal with any unexpected complicating lesion such as infundibular stenosis or an associated ventricular septal defect.

Our choice of the cardiac bypass technique for open heart work rather than hypothermia and inflow occlusion, is based on the former technique being more versatile and allowing the surgeon a greater margin of safety if any complication should arise.

Neither techniques for surgical correction or cardiac bypass are static. The present methods are being improved and replaced constantly and certainly optimal methods have not as yet been developed. Currently we are working with a disc-type oxygenator* in the experimental laboratory at the...
TABLE II—CARDIAC BY-PASS PERFUSION DATA

<table>
<thead>
<tr>
<th>Patient</th>
<th>B.S.A. (m²)</th>
<th>Total Perfusion Time (Min.)</th>
<th>Total Duration Of Cardiac Arrest</th>
<th>Perfusion Rate (ml/min./m²)</th>
<th>Post-Perfusion pH</th>
<th>Post-Perfusion Plasma Hgb. (mgm%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.2</td>
<td>16</td>
<td>0</td>
<td>1500</td>
<td>7.31</td>
<td>81</td>
<td>Difficulty with coronary sinus suction.</td>
</tr>
<tr>
<td>2</td>
<td>0.8</td>
<td>9½</td>
<td>0</td>
<td>1800</td>
<td>7.46</td>
<td>32</td>
<td>No complications</td>
</tr>
<tr>
<td>3</td>
<td>0.8</td>
<td>9</td>
<td>0</td>
<td>1830</td>
<td>7.40</td>
<td>82</td>
<td>No complications</td>
</tr>
<tr>
<td>4</td>
<td>0.6</td>
<td>28</td>
<td>16 min.</td>
<td>1830</td>
<td>7.33</td>
<td>77</td>
<td>No complications</td>
</tr>
<tr>
<td>5</td>
<td>1.0</td>
<td>23</td>
<td>10 min.</td>
<td>1850</td>
<td>7.50</td>
<td>71</td>
<td>No complications</td>
</tr>
<tr>
<td>6</td>
<td>0.8</td>
<td>44</td>
<td>11 min.</td>
<td>2000</td>
<td>7.38</td>
<td>75</td>
<td>No complications</td>
</tr>
<tr>
<td>7</td>
<td>0.8</td>
<td>17</td>
<td>7 min.</td>
<td>2060 Average 1850 ml/min/m²</td>
<td>7.39</td>
<td>82</td>
<td>No complications</td>
</tr>
</tbody>
</table>

*Body Surface Area in Square Meters.

University of Oklahoma Medical Center. This apparatus lends itself to higher flow volumes and will prove of help for more prolonged periods of perfusion and for obtaining the increased perfusion rates required for larger patients. Techniques for making cardiac bypass safer for small children and infants are also being investigated.

Summary

The initial experiences with cardiac bypass for surgical correction of congenital heart defects at the University of Oklahoma Medical Center is presented. Corrective procedures have been accomplished in seven patients to date. There has been no operative mortality. Operative morbidity has been limited to one case of "post-commissurotomy" syndrome and one case of postoperative pneumonia and pneumothorax. Both these patients responded to appropriate therapy. Postoperative clinical evaluation shows that none of the seven patients has any significant residual defect.

REFERENCES


ADDENDUM

Since the submission of this report patients 4, 5, 6 and 7 have been discharged from the hospital. In addition three more patients have undergone cardiopulmonary by-pass and successful corrective surgery for (1) atrial septal defect with anomalous right pulmonary veins, (2) Tetralogy of Fallot and (3) infundibular pulmonic stenosis.