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Hemodynamics after Surgical Repair with Right Ventricle to Pulmonary Artery Conduit

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SUMMARY To assess the results of cardiac repair utilizing a right
ventricular to pulmonary artery conduit, we reviewed postoperative
hemodynamic data in 16 patients catheterized 0.5 to 5 years after
repair. In 12 patients, a Hancock conduit (dacron conduit with porcine
carina) was used; the conduit in the remaining four patients
was made with an aortic homograft. All patients in whom an aortic homograft
was utilized developed severe obstruction and calcification of
dacron conduit with a porcine aortic valve was used to estab-
lish continuity between the functional right ventricle and the
main pulmonary artery or its major branches.

TWO SURGICAL TECHNIQUES to establish continuity
between the right ventricle and pulmonary arteries are now
well accepted. Either a homograft aortic valve with a segment of ascending aorta or a dacron conduit was used. These conduits have been used with
short-term success for the repair of severe forms of tetralogy of
Fallot,1-4 pulmonary atresia with ventricular septal defect,1,4 various forms of trans-
position of the great arteries with pulmonary stenosis and
ventricular septal defect.5,7 However, only a few patients
with aortic homograft, and none with the dacron conduit,
have been followed with long-term hemodynamic studies.5-10

The purpose of this study was to evaluate the hemody-
amic and angiocardiographic findings in the early and
late postoperative period in patients in whom either a homograft aortic valve with a segment of ascending aorta or a

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Boston, Massachusetts 02115.
Received May 10, 1976; revision accepted June 24, 1976.

Material and Methods

Between January 1974 and September 1975, 16 con-
secutive patients with a conduit from functional right ventri-
cle to pulmonary artery underwent cardiac catheterization
at The Children’s Hospital Medical Center of Boston.
Catheterization was performed 0.1 to 5 years (mean 1.8
years) following the surgical repair (table 1). Twelve of these
patients had severe forms of tetralogy of Fallot, two patients
had D-transposition of the great arteries with pulmonary
stenosis and a ventricular septal defect, one patient had trun-
cus arteriosus, and one patient had corrected transposition
with pulmonary atresia, and a ventricular septal defect.
The patients’ ages at surgery ranged between 2 and 24 years
(mean 11.3 years). The operation was performed at either
the Children’s Hospital Medical Center of Boston (13/16
patients) or at the Mayo Clinic (3/16 patients).

In four of the 16 patients, a frozen-irradiated homograft
aortic valve with a segment of the ascending aorta was used
as a conduit to establish continuity between the heart and
pulmonary arteries, while in the remaining 12 patients a
TABLE 1. Pertinent Clinical Information in 16 Patients with Right Ventricular to Pulmonary Artery Conduit

<table>
<thead>
<tr>
<th>Pt</th>
<th>Diagnosis</th>
<th>Previous operations</th>
<th>Age at repair</th>
<th>Type</th>
<th>Age at cath</th>
</tr>
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<tbody>
<tr>
<td>1/JK</td>
<td>Truncus arteriosus</td>
<td>None</td>
<td>10½</td>
<td>Homograft</td>
<td>15½</td>
</tr>
<tr>
<td>2/DM</td>
<td>TOF</td>
<td>Bil. B-T</td>
<td>17</td>
<td>Homograft</td>
<td>21</td>
</tr>
<tr>
<td>3/RW</td>
<td>TOF</td>
<td>None</td>
<td>2</td>
<td>Homograft</td>
<td>3½</td>
</tr>
<tr>
<td>4/RR</td>
<td>TOF</td>
<td>Bil. B-T</td>
<td>17</td>
<td>Homograft</td>
<td>22</td>
</tr>
<tr>
<td>5/KH</td>
<td>TOF</td>
<td>Lt. B-T &amp; Asc. Ao.</td>
<td>14</td>
<td>Hancock</td>
<td>16</td>
</tr>
<tr>
<td>6/RB</td>
<td>TOF</td>
<td>Asc. Ao. to RPA</td>
<td>11</td>
<td>Hancock</td>
<td>12</td>
</tr>
<tr>
<td>7/RN</td>
<td>TOF and absent LPA</td>
<td>None</td>
<td>10</td>
<td>Hancock</td>
<td>11</td>
</tr>
<tr>
<td>8/KM</td>
<td>TOF</td>
<td>Bil. B-T</td>
<td>14</td>
<td>Hancock</td>
<td>15</td>
</tr>
<tr>
<td>9/RL</td>
<td>TOF</td>
<td>Bil. B-T</td>
<td>9</td>
<td>Hancock</td>
<td>10</td>
</tr>
<tr>
<td>10/KD</td>
<td>TOF</td>
<td>Asc. Ao to RPA</td>
<td>9</td>
<td>Hancock</td>
<td>10</td>
</tr>
<tr>
<td>11/PF</td>
<td>SLT</td>
<td>Asc. Ao to MPA</td>
<td>7</td>
<td>Hancock</td>
<td>7½</td>
</tr>
<tr>
<td>12/EY</td>
<td>TOF</td>
<td>Rt. B-T &amp; Asc. Ao. to RPA</td>
<td>8</td>
<td>Hancock</td>
<td>8.1</td>
</tr>
<tr>
<td>13/KR</td>
<td>TOF</td>
<td>Asc. Ao. to RPA</td>
<td>3½</td>
<td>Hancock</td>
<td>4½</td>
</tr>
<tr>
<td>14/JP</td>
<td>DTGA</td>
<td>None</td>
<td>18</td>
<td>Hancock</td>
<td>21</td>
</tr>
<tr>
<td>15/DS</td>
<td>DTGA</td>
<td>Bil. B-T</td>
<td>24</td>
<td>Hancock</td>
<td>26</td>
</tr>
<tr>
<td>16/DV</td>
<td>TOF</td>
<td>Asc. Ao. to RPA</td>
<td>7</td>
<td>Hancock</td>
<td>8</td>
</tr>
</tbody>
</table>

Abbreviations: TOF = tetralogy of Fallot; SLT = eustus solitus of the visera; L-loop and L-transposition of the great arteries; DTGA = D-transposition of the great arteries; Bil = bilateral; B-T = Blalock-Taussig shunt; Asc. Ao to RPA = ascending aorta to right pulmonary artery shunt; Lt = Left; Rt = Right; LPA = left pulmonary artery; MPA = main pulmonary artery.

TABLE 2. Anatomic and Hemodynamic Findings in Patients with Aortic Homograft

<table>
<thead>
<tr>
<th>No.</th>
<th>Wt (kg)</th>
<th>RV (mm Hg)</th>
<th>Ao (mm Hg)</th>
<th>SBF L/min/m²</th>
<th>PBF L/min/m²</th>
<th>Gradient across homograft (mm Hg)</th>
<th>Total</th>
<th>Prox</th>
<th>Val</th>
<th>Dist</th>
<th>Calcif.</th>
<th>Residual defects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>47.0</td>
<td>205</td>
<td>185</td>
<td>—</td>
<td>—</td>
<td>*PA NE</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>Severe truncal regurgitation</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>60</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>43.7</td>
<td>115</td>
<td>115</td>
<td>2.2</td>
<td>8.9</td>
<td>60</td>
<td>60</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>VSD, PAH (moderate)</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>60</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13.0</td>
<td>120</td>
<td>110</td>
<td>3.9</td>
<td>3.9</td>
<td>92</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>Tricuspid regurgitation</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>55</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>66.7</td>
<td>75</td>
<td>100</td>
<td>2.2</td>
<td>2.2</td>
<td>**PA NE</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>Tricuspid regurgitation</td>
</tr>
<tr>
<td></td>
<td>NA</td>
<td>NA</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Severely obstructed homograft at surgery.
**Distal obstruction delineated by right ventricular angiography.

Abbreviations: wt = weight; RV = right ventricular pressure; Ao = aortic pressure; SBF = systolic blood flow; PBF = pulmonary blood flow; Prox = proximal; Val = valvar; Dist = distal; Calcif = calcification; VSD = ventricular septal defect; PAH = pulmonary artery hypertension; NE = not entered; NA = not available.
shown in figure 2. There are dense collagen fibers with areas of calcification, ossification, and almost no elastic tissue.

The homograft was markedly obstructed in all four patients, as documented by a large pressure gradient in two, angiography in one, and surgery in one. Two of the patients (1 and 3) had suprasystemic pressures in the right ventricle when right ventricular pressures were measured simultaneously with aortic pressures. Right ventricular end-diastolic pressure was elevated in three patients and systemic blood flow was abnormally low in two. A murmur of pulmonary insufficiency was not detected in any of the patients. All four patients had congestive heart failure and two patients had arrhythmias.

Table 3 summarizes the hemodynamic results of postoperative cardiac catheterization in the 12 patients with dacron conduit and porcine aortic valve. None of the patients had any evidence of calcification of the conduit either on chest X-ray or at fluoroscopy. However, all 12 patients had some degree of conduit obstruction (figs. 3-6). Pressure gradients across the right ventricular to pulmonary artery conduit ranged from 12 to 98 mm Hg, with the majority (nine out of 12) having a gradient of less than 45 mm Hg. Obstructions tended to be in three distinct areas: the proximal end of the conduit (junction with the ventricle) in eight of the 12 patients; the porcine aortic valve in four of the 12 patients; and the distal end of the conduit (junction with pulmonary artery) in six of the 12 patients. More than one obstruction was present in half of the patients.

Increased right ventricular end-diastolic pressure was present in only two of the 12 patients and none had decreased systemic blood flow. A murmur of pulmonary insufficiency was present in two patients (11 and 13). In one of these, a pressure gradient was measured across the porcine valve and pulmonary artery pressure was normal. The second patient had severe pulmonary artery hypertension with no obstruction across the porcine valve. All but one of the patients with this type of conduit were asymptomatic. Patient 11 had congestive heart failure. Five of the 12 patients had isometric exercise studies during cardiac catheterization. An increase in pulmonary blood flow during isometric exercise was demonstrated in all five patients (fig. 7). The increase in flow ranged from 31 to 81% (mean 49%). The four patients in whom a pulmonary artery to right ventricular pressure was obtained during exercise demonstrated an increase in the pressure gradient which was proportional to the increase in pulmonary flow.

Seven of the 12 patients in whom a dacron conduit was used had a residual ventricular septal defect, only one of which caused hemodynamically significant left-to-right
TABLE 3. Anatomic and Hemodynamic Findings in Patients with Dacron Conduit and Porcine Valve

<table>
<thead>
<tr>
<th>No.</th>
<th>Wt (kg)</th>
<th>Conduit size</th>
<th>RVp mm Hg</th>
<th>Aop mm Hg</th>
<th>Gradient across conduit</th>
<th>SBF L/min/m²</th>
<th>PBF</th>
<th>Residual defects</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>40.0</td>
<td>20</td>
<td>102</td>
<td>110</td>
<td>78 36 42</td>
<td>3.9</td>
<td>7.0</td>
<td>VSD</td>
</tr>
<tr>
<td>6</td>
<td>37.0</td>
<td>18</td>
<td>72</td>
<td>120</td>
<td>40 — — 40</td>
<td>3.1</td>
<td>6.3</td>
<td>VSD, PAH (mild)</td>
</tr>
<tr>
<td>7</td>
<td>36.0</td>
<td>20</td>
<td>65</td>
<td>125</td>
<td>34 — — 34</td>
<td>3.3</td>
<td>3.3</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>31.2</td>
<td>22</td>
<td>65</td>
<td>98</td>
<td>39 39 — —</td>
<td>3.7</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>38.5</td>
<td>20</td>
<td>48</td>
<td>95</td>
<td>25 — — 25</td>
<td>3.0</td>
<td>3.2</td>
<td>VSD, Patent B-T Shunt</td>
</tr>
<tr>
<td>10</td>
<td>30.6</td>
<td>20</td>
<td>58</td>
<td>100</td>
<td>44 — — 44</td>
<td>3.5</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>20.3</td>
<td>16</td>
<td>100</td>
<td>100</td>
<td>34 — — 34</td>
<td>3.1</td>
<td>3.7</td>
<td>VSD, Tricuspid regurgitation</td>
</tr>
<tr>
<td>12</td>
<td>25.2</td>
<td>18</td>
<td>90</td>
<td>105</td>
<td>73 — — 62</td>
<td>3.0</td>
<td>4.0</td>
<td>PAH (mod), pulmonary regurgitation</td>
</tr>
<tr>
<td>13</td>
<td>14.1</td>
<td>16</td>
<td>65</td>
<td>100</td>
<td>38 13 25 —</td>
<td>3.5</td>
<td>5.0</td>
<td>VSD, Pulmonary regurgitation</td>
</tr>
<tr>
<td>14</td>
<td>62.8</td>
<td>25</td>
<td>20</td>
<td>100</td>
<td>12 9 — 3</td>
<td>3.0</td>
<td>3.0</td>
<td>Subaortic obstruction</td>
</tr>
<tr>
<td>15</td>
<td>67.4</td>
<td>22</td>
<td>118</td>
<td>118</td>
<td>98 60 — 38</td>
<td>4.0</td>
<td>4.0</td>
<td>Subaortic obstruction (gradient 40 mm Hg)</td>
</tr>
<tr>
<td>16</td>
<td>18.1</td>
<td>16</td>
<td>80</td>
<td>105</td>
<td>41 28 13 —</td>
<td>4.5</td>
<td>7.6</td>
<td>VSD, PAH (mild)</td>
</tr>
</tbody>
</table>

Abbreviations: Sub Ao Obf,t = subaortic obstruction; Mod = moderate; for others see tables 1 and 2.

shunt (pulmonary to systemic flow ratio ≥ 2:1) (table 3, #6). (The high incidence of residual ventricular septal defect was probably related to the use of nonpledged sutures when the patch was anchored to the ventricular septal defect.) The increased pulmonary blood flow resulting from a residual left-to-right shunt may contribute to the measured gradient across the conduit.

Discussion

Since 1966 when Ross and Somerville used a homograft aortic valve with a segment of ascending aorta to reconstruct the right ventricular outflow tract in a patient with pulmonary atresia, the use of a valve-carrying conduit to establish continuity between the right ventricle and pulmonary arteries has become a common treatment for some con-

![Figure 3. Pressure tracing from patient 12 demonstrating a gradient at the distal anastomotic site and at the porcine valve. Abbreviations: MPA = main pulmonary artery; Distal Cond. = distal conduit; Prox cond. = proximal conduit; RV = right ventricular; EKG = electrocardiogram.](image-url)
genital cardiac malformations. However, despite the wide use of conduits, little is known about the long-term hemodynamic effects of these devices on patients. There have been a number of reports of severe calcification and obstruction in patients in whom a homograft aortic valve with a segment of ascending aorta was used to establish continuity.\textsuperscript{13-16} Four of our 16 patients had an aortic homograft used in repair, and each demonstrated calcification and obstruction of the homograft at recatheterization two to five years postoperatively. In one patient, the homograft was severely calcified with complete destruction of the valvular cusps and severe obstruction to flow. Similar results were observed by Merin and McGoon\textsuperscript{17} in one of their patients. The calcification of the implanted homograft valve and vessel is thought to be related to techniques of the sterilization and preservation of the homograft.\textsuperscript{15, 16} Because of the high incidence of calcification, severe obstruction, and pseudoaneurysm formation, we have abandoned the use of homograft conduits and at present prefer the use of glutaraldehyde-preserved dacron conduits with a porcine aortic valve.

In none of the 12 patients with dacron conduits and porcine aortic valve was calcification present; however, these patients have only been followed for 0.1 to 3 years (mean 1.2 years) while the patients with calcified homografts have been followed for 1.5 to 5 years (mean 3.8 years). Nine of the 12 patients with dacron conduit and porcine aortic valve had mild to moderate pulmonary outflow tract obstruction (less than 44 mm Hg). This degree of right ventricular outflow tract obstruction represents an acceptable gradient.\textsuperscript{16, 17} However, three of the patients did have severe conduit obstruction and may require reoperation for relief of the conduit obstruction in the future. At present our major indicators for reoperation are persistent congestive heart failure and/or suprasystolic right ventricular pressures.

**FIGURE 4.** A lateral angiogram from patient 12 demonstrating narrowing at the junction of the Hancock conduit with the pulmonary arteries (arrow). The porcine valve and a virtually atretic right ventricular infundibulum are also well visualized. Abbreviations: XV = porcine valve; RV = right ventricle; CND = Hancock conduit; PV = pulmonary valve.

**FIGURE 5.** Pressure tracing from patient 5 demonstrating a gradient at the porcine valve and at the proximal anastomosis between the right ventricle and conduit. Abbreviations: LPA = left pulmonary artery; Distal cond. = distal conduit; Prox. cond. = Proximal conduit; RV = right ventricle; RA = right atrium; Ao = Aorta; EKG = electrocardiogram.
The conduit obstruction was documented to occur at three sites: at the egress from the right ventricle, at the porcine valve, and at the pulmonary artery to conduit junction. Obstruction distally, at the pulmonary artery junction, was the most common site, occurring in eight out of 12 patients. Six of the eight patients with a distal obstruction had a narrowed or deformed main or right pulmonary artery secondary to a previous systemic-to-pulmonary shunt. This probably represents the predominant mechanism for obstruction at the distal end of the conduit. Hypoplasia of the pulmonary arteries in patients with markedly diminished pulmonary flow may also contribute to distal obstruction. In each of the six patients with proximal obstruction, the obstruction was documented by angiography to result from kinking of the graft as it exited from the right ventricle. Finally, four of the patients exhibited obstruction at the level of the porcine valve. In one of these patients a murmur of pulmonary insufficiency was also present, suggesting a structural abnormality of the porcine valve itself rather than a relatively small anulus as the cause of the obstruction. We have no data on which to base recommendations for optimal conduit size but believe increasing obstruction with growth is a possible complication, especially when the conduit is inserted in very young patients.

Isometric exercise increased the magnitude of conduit obstruction (fig. 7). An approximate increase in pulmonary blood flow of 50% resulted in an average increase of gradient across the conduit of 16 mm Hg. Since isometric exercise causes relatively small increases in flow, it is likely that during rhythmic exercise, which brings about a great increase in pulmonary flow, a much larger increase in conduit gradient will occur.

A dacron conduit with a porcine aortic valve is a hemodynamically more satisfactory means of re-establishing continuity between the heart and pulmonary arteries in certain forms of congenital heart disease, but most of our patients developed obstruction even with dacron conduits.

**References**

Hemodynamics after surgical repair with right ventricle to pulmonary artery conduit.
A P Rocchini, A Rosenthal, J F Keane, A R Castaneda and A S Nadas

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