Subaortic Obstruction after the Use of an Intracardiac Baffle to Tunnel the Left Ventricle to the Aorta

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SUMMARY Postoperative hemodynamic studies in five patients document subaortic obstruction after surgical repair utilizing an intracardiac baffle to establish continuity between the left ventricle and the aorta. Four of the patients had a Rastelli procedure for D-transposition of the great arteries with a ventricular septal defect and pulmonary stenosis; one patient had repair of double outlet right ventricle with a ventricular septal defect and pulmonary stenosis. The left ventricular outflow was shown to be a long narrow tunnel by angiography in four of five patients and by echocardiography in one patient.

ANATOMIC REPAIR OF D-TRANSPOSITION of the great arteries (DTGA) with a ventricular septal defect (VSD) and severe subpulmonic stenosis (PS), using an intracardiac tunnel to connect the left ventricle to the aorta and a valved conduit to connect the right ventricle to the pulmonary artery (Rastelli procedure), has become an acceptable surgical technique. The use of an intracardiac tunnel to connect the left ventricle to the aorta has also been used in the repair of some forms of double outlet right ventricle (DORV). The postoperative residual defects and sequelae observed in patients with DORV and DTGA with VSD and PS include residual VSD, right ventricular to pulmonary artery obstruction, recurrent paroxysmal tachycardia, and unexplained late deaths. There have been no reports of subaortic obstruction following this procedure. The hemodynamic data of five patients in whom a left ventricular outflow tract gradient was demonstrated at postoperative cardiac catheterization form the basis of this report.

Methods

The diagnostic files of the Department of Cardiology and Cardiovascular Surgery at the Children's Hospital Medical Center of Boston were searched and the records of five patients (four with DTGA, VSD, and PS, and one with DORV) who showed subaortic obstruction after repair were obtained and reviewed. The pre- and postoperative hemodynamic and angiographic data were examined. Summary of the clinical and hemodynamic findings in each patient is shown in table 1.

Case Reports

Surgical repair in the patients with DTGA, VSD, and subpulmonic stenosis was performed through a midline sternotomy. A synthetic patch (Dacron in patients 1, 3, 4, and Teflon in patient 2) was used to close the VSD in such a manner that the left ventricle drained through the septal defect to the aorta. The upper rim of the baffle was anchored to the rim of the ventriculotomy in all four patients, so as to give a wide left ventricular outflow tract. The main pulmonary artery was ligated or divided and a dacron conduit with a porcine aortic valve (Hancock Laboratories) was anastomosed to both the main pulmonary artery and the right ventricle. The Hancock conduits varied in size from 18 mm to 25 mm (table 1). Previously placed systemic-to-pulmonary artery shunts were closed. In the patient with DORV, VSD, and subpulmonary stenosis (5), the surgical repair was performed through a midline sternotomy. The VSD was enlarged, the subpulmonic area resected, and an Ivalon patch was used to tunnel the left ventricle into the aorta.

Following operation all but one patient (3) experienced marked clinical improvement with loss of cyanosis and exercise intolerance. Patients 1 and 2 have been followed for 1½ and 2 years postoperatively, respectively, and have been asymptomatic. Patient 3, however, underwent cardiac catheterization five days after repair because of persistent congestive heart failure. Following the catheterization (table 1), the patient was reoperated and a large residual VSD closed. After the second operation, the child remained asymptomatic. Patient 4, although initially experiencing marked clinical improvement, developed bacterial endocarditis and severe congestive heart failure two months postoperatively. Cardiac catheterization at that time demonstrated a large residual VSD, severe right ventricular outflow obstruction, and mild subaortic obstruction (table 1). At surgery, an infected VSD patch and Hancock graft were found and replaced; however, despite maximal medical therapy, the patient died. There was no evidence either at surgery or postmortem examination to suggest that vegetations were responsible for the observed subaortic gradient.

Following surgery, patient 5 remained asymptomatic for nine years. An elective catheterization eight years postoperatively demonstrated a residual VSD, mild right ventricular outflow tract obstruction, and significant left ventricular outflow tract obstruction (table 1). One year later,
angina and decreased exercise tolerance developed. A graded treadmill exercise study at this time showed limited exercise tolerance for age (Bruce protocol; 11:18 min; normal in our laboratory is 13 min), bursts of ventricular bigeminy during the recovery phase, but no electrocardiographic changes suggestive of myocardial ischemia. Reoperation is planned with enlargement of the VSD and revision of the patch.

Postoperative cardiac catheterization was performed at intervals of from two days to eight years postoperatively. A resting left ventricular outflow pressure gradient, ranging from 10 to 42 mm Hg (mean 25), was documented in all patients. The obstruction was localized at catheterization to the proximal end of the left ventricle to aortic tunnel (i.e., site of the VSD). All of the left ventricular to aortic pressure gradients were established by withdrawal of the catheter from the LV to the aorta. Figure 1 illustrates the pressure gradient obtained from patient 1. Isometric exercise in the same patient, using a hand grip dynamometer at 50% of maximal voluntary capacity, resulted in a 12 mm Hg increase in the left ventricular outflow tract gradient (fig. 2). The left ventricular outflow tract was shown by either angiography or echocardiography to be a long, narrow tunnel (figs. 3, 4).

**Discussion**

The use of an intracardiac baffle to tunnel the left ventricle to the aorta and a valve-carrying conduit to connect the right ventricle to the pulmonary artery has become the preferred surgical approach for repair of DTGA with a VSD and PS.

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**TABLE 1. Pertinent Clinical and Hemodynamic Findings Prior to and After Surgical Repair**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Diagnosis</th>
<th>Age at cath (yr)</th>
<th>Age at repair (yr)</th>
<th>Status</th>
<th>SatO₂ %</th>
<th>Flow</th>
<th>Pressure (mm Hg)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>DTGA, PS VSD, ASD, Bil. B-T shunts</td>
<td>23</td>
<td>24</td>
<td>Preop</td>
<td>88</td>
<td>2.4</td>
<td>11.2</td>
<td>1.9</td>
</tr>
<tr>
<td>2</td>
<td>DTGA, PS, VSD, ASD</td>
<td>16.9</td>
<td>19</td>
<td>Postop</td>
<td>25.5</td>
<td>96</td>
<td>4.0</td>
<td>4.0</td>
</tr>
<tr>
<td>3</td>
<td>DTGA, PS, VSD, ASD, AsAo-RFA shunt</td>
<td>21</td>
<td>8</td>
<td>Postop</td>
<td>21</td>
<td>97</td>
<td>3.5</td>
<td>3.5</td>
</tr>
<tr>
<td>4</td>
<td>DTGA, PS, VSD, Potts shunt</td>
<td>19</td>
<td>20</td>
<td>Postop</td>
<td>20.6</td>
<td>80</td>
<td>3.5</td>
<td>5.1</td>
</tr>
<tr>
<td>5</td>
<td>DORV, PS, VSD</td>
<td>15.2</td>
<td></td>
<td>Postop</td>
<td>15.2</td>
<td>97</td>
<td>2.8</td>
<td>5.0</td>
</tr>
</tbody>
</table>

Abbreviations: No = number; Cath = catheterization; SatO₂ = systemic arterial oxygen saturation; Sys = systemic; Pul = pulmonary; Eff = effective; RV = right ventricle; PA = pulmonary artery; LV = left ventricle; Ao = aorta; Yrs = years; DTGA = D-transposition of the great arteries; VSD = ventricular septal defect; PS = pulmonary stenosis; ASD = atrial septal defect; AsAo = ascending aorta; B-T = Blalock-Taussig; Bil = bilateral; Preop = preoperative; Postop = postoperative; Mod = moderate; Obst. = obstructive.
and some forms of DORV.\textsuperscript{7,8} However, little is known of the long-term hemodynamic results of this operation. With regard to the Rastelli operation for DTGA, McGoon et al.\textsuperscript{4} have reported a high early mortality of 25% and they and others\textsuperscript{9-11} have reported late complications such as residual VSDs, right ventricular-pulmonary artery obstruction, arrhythmias, and late postoperative deaths. Similar results have been noted after repair of DORV.\textsuperscript{7,8} The subaortic obstruction, observed in our patients, adds yet another possible serious sequelae to these operations.

Subaortic obstruction may develop prior to surgical repair due to a restrictive VSD, subaortic conal muscle, or endocardial cushion tissue in patients with DORV\textsuperscript{9-11} and secondary to a restrictive VSD in patients with DTGA, VSD and PS.\textsuperscript{4} Muscular subaortic obstruction due to hypertrophy of the conal septum may occur after pulmonary artery banding in patients with VSD and normal\textsuperscript{19} or transposed great arteries.\textsuperscript{19} To our knowledge, however, this is the first report of subaortic obstruction following repair with the use of an intracardiac baffle to tunnel the left ventricle to the aorta.

A left ventricular outflow pressure gradient was documented in all five of our patients. Although the magnitude of the pressure gradients was only mild to moderate (ranging from 13 to 42 mm Hg), the gradient in patient 1 did increase with isometric hand grip exercise and in patient 5 the subaortic obstruction, along with a residual VSD, was also associated with angina and decreased exercise tolerance. The LV outflow tract in each patient was a long, narrow tunnel with the obstruction localized to the proximal end of the tunnel (i.e., site of the VSD).

The mechanism for the development of subaortic obstruction is uncertain. However, since all five LV outflow gradients were located at the entrance of the LV baffle, the gradients are likely to have been caused by the size of the VSD and/or the geometry of the proximal portion of the baffle. The VSDs in all the patients were felt at preoperative catheterization (both angiographically and hemodynamically, as indicated by equalization of right ventricular, left ventricular, and aortic pressure) and at surgery to be nonrestrictive. Nonetheless, placement of the intraventricular baffle may decrease the effective size of the VSD and make a marginal VSD restrictive.

The decrease in effective VSD size may be due to either the baffle itself or the secondary proliferation of fibrous tissue along the rim of the VSD and/or on the baffle itself. Since McGoon\textsuperscript{4} has reported no significant increase in risk to patients who required VSD enlargement, excision of the subaortic conal muscle to enlarge the VSD should probably become an integral part of the surgical procedure.

Other possible etiologies for the development of left ventricular to aortic obstruction in our patients include redundancy or kinking of the baffle, shrinkage of the baffle with

\textbf{Figure 2.} The relationship between left ventricular (LV) to aortic (Ao) outflow tract gradient and systemic blood flow during isometric exercise in patient 1. Isometric exercise resulted in an increase of systemic blood flow with consequent increase in pressure gradient.

\textbf{Figure 3.} An anterior-posterior and lateral angiogram obtained in patient 1 demonstrating narrowing of the left ventricular (LV) outflow tract at the level of the intracardiac baffle (arrow). The metallic ring to support the porcine valve within the Hancock conduit is also visualized. Ao = aorta.
Fig. 4. An echocardiogram obtained from patient 5, demonstrating obstruction of the left ventricular (LV) outflow tract at the level of the intracardiac baffle (arrows). The baffle diameter is only 1.5 cm compared to an aortic (Ao) diameter of 2.5 cm. LA = left atrium.

time, and spontaneous diminution of the VSD orifice.

Irrespective of the mechanism involved, the development of subaortic obstruction after the use of an intracardiac baffle to connect the left ventricle to aorta is a serious complication. The obstruction may be hemodynamically significant and the patients may develop symptoms. The obstruction may also progress with time, especially if the operation is performed at a young age. Left ventricular pressures should be recorded intraoperatively or immediately postoperatively, and one year and four years postoperatively to monitor any progression toward obstruction. In addition, echocardiographic studies may be useful.

References

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