Late Development of Pulmonary Venous Obstruction Following Mustard's Operation Using a Dacron Baffle

DAVID J. DRISCOLL, M.D., MICHAEL R. NIHILL, M.D., THOMAS A. VARGO, M.D., CHARLES E. MULLINS, M.D., AND DAN G. McNAMARA, M.D.

SUMMARY Three patients developed late pulmonary vein obstruction (PVO) following Mustard's operation for transposition of the great arteries. In all three the absence of PVO had been documented by an earlier postoperative cardiac catheterization. At reoperation shrinkage and kinking of the dacron baffle was evident.

After Mustard's operation, patients should be observed closely for symptoms and signs of PVO since it can occur insidiously despite previously proven absence of this complication.

PULMONARY VENOUS OBSTRUCTION (PVO) is a recognized complication following Mustard's operation for transposition of the great arteries (TGA). It can occur early as well as late in the postoperative period. The purpose of this communication is to report late development of PVO in three patients who had no evidence of PVO at the time of an initial postoperative catheterization.

Material and Methods

From April 1972 to July 1973, 32 patients underwent Mustard repair of TGA with insertion of a dacron baffle at the Texas Children's Hospital. Only one patient had enlargement of the pulmonary venous atrium (PVA) at the time of the Mustard procedure. The age of the patients at operation ranged from 15 to 60 months and their weights ranged from 9 to 14 kg.

Fifteen patients underwent cardiac catheterization (table 1) 0.5 to 25 (mean 9.3) months after surgery. Four patients had a second postoperative cardiac catheterization 10 to 35 (mean 23) months after surgery.

Cardiac catheterizations were performed after sedation with meperidine hydrochloride (1 mg/kg) and promethazine hydrochloride (0.5 mg/kg). To determine the presence of PVO, simultaneous pressures were recorded in the pulmonary wedge position and the PVA or sequential pressures were obtained in a pulmonary vein and the PVA. Additional evidence of obstruction to pulmonary venous drainage was obtained using biplane cineangiography with selective injection of Hypaque into either a pulmonary vein or the pulmonary artery. Pulmonary blood flow (Qp) and cardiac output were calculated using assumed oxygen consumption and measurement of blood oxygen saturation from the main pulmonary artery (or superior vena cava), a pulmonary vein (or PVA) and the femoral artery.

Results

There were seven deaths less than 30 days following surgery and three late deaths.

PVO was demonstrated 3 to 12 (mean 9.3) months postoperatively in 6 of 15 patients undergoing initial postoperative cardiac catheterization. One patient with PVO also had superior vena cava obstruction (SVO). Five of six patients with PVO had symptoms that included cough, exertional fatigue, resting tachypnea and cyanosis (table 1). Symptoms began 3 to 12 (mean 6.6) months postoperatively.

Three patients (A, B, C) who were free of PVO at the time of their first postoperative cardiac catheterization subsequently developed symptoms 10 to 28 (mean 17.6) months after surgery (table 1). Symptoms in these three patients also included cough, exertional fatigue, resting tachypnea, and...
cyanosis. Pneumonia had been erroneously diagnosed on two occasions in patient A. In two patients a high frequency diastolic murmur was heard at the lower left sternal border at the time symptoms developed. Cardiomegaly and increased pulmonary vascular markings absent at the time of the first postoperative cardiac catheterization became apparent on chest radiograms with the onset of symptoms. Cardiac catheterizations done 0.5 to 4 months after the onset of symptoms demonstrated PVO (table 2, figs. 1–4) as well as SVO in all three patients. Surgical enlargement of the PVA with a dacron gusset (fig. 5) was performed in two patients (A and B). Patient B also had replacement of the dacron intra-atrial baffle with pericardium. Patient C, who had had pulmonary venous atriotomy using a dacron gusset at the time of the Mustard operation, had further enlargement of the PVA using pericardium. At the time of surgery, the dacron intra-atrial baffle in each instance was contracted and rigid. Following atriotomy all three patients improved clinically and the diastolic murmur could no longer be heard. Cardiac catheterization in patient A performed eight months after atriotomy revealed no PVO (table 2).

**Discussion**

Because of the occurrence of PVO using a pericardial baffle, dacron was selected as an alternative material.1,4 Subsequently, however, PVO has been reported to occur more frequently following dacron baffle insertion4,6 and many surgeons6,7 have returned to using pericardial baffles.

Operative techniques, postoperative infection of the baffle, baffle shrinkage, and adhesions between the baffle and atrial wall have been proposed as mechanisms in development of PVO.1,4,7,13 Several studies have indicated that failure of the free wall of the PVA to grow probably is not the mechanism of obstruction.1,11,13 Although PVO has been demonstrated both early and late following Mustard repair, we found no reports of the development of PVO after previous documentation of no obstruction. Documented absence of PVO followed by development of PVO favors an ongoing process such as contraction and fibrosis of the baffle rather than intraoperative technique as the cause of late development of PVO. This process may relate to the type of baffle material employed. Others have demonstrated that dacron baffles become enveloped in a thicker neocardiomyct in than does pericardium.11 Although both materials contract in vivo, dacron has a tendency to kink, is more difficult to tailor and is stiffer than pericardium.8

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**Table 1. Relationship of Onset of Symptoms of PVO in 15 Patients to Time and Findings of Postoperative Cardiac Catheterizations**

<table>
<thead>
<tr>
<th>Symptom onset months postop</th>
<th>1st Postoperative catheterization</th>
<th>2nd Postoperative catheterization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient A</td>
<td></td>
<td></td>
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<tr>
<td>Patient B</td>
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<td></td>
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<tr>
<td>Patient C</td>
<td></td>
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</tbody>
</table>

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**Table 2. Cardiac Catheterization Data**

<table>
<thead>
<tr>
<th>Cath or surgery age</th>
<th>Wt (kg)</th>
<th>Qs (L/min/m²)</th>
<th>RV (mm Hg)</th>
<th>LV (mm Hg)</th>
<th>PA (mm Hg)</th>
<th>PAW (mm Hg)</th>
<th>PV (mm Hg)</th>
<th>PVA (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient A</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cath 1 (1 day)</td>
<td>4</td>
<td>60/0.25</td>
<td>60/0.15</td>
<td></td>
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<tr>
<td>Cath 2 (9 mo)</td>
<td>9.3</td>
<td>80/0.9</td>
<td>40/0.9</td>
<td></td>
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<tr>
<td>Mustard (15 mo)</td>
<td>10.3</td>
<td>90/0.8</td>
<td>32/4.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cath 3 (22 mo)</td>
<td>12.9</td>
<td>90/0.8</td>
<td>32/4.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cath 4 (47 mo)</td>
<td>14.5</td>
<td>130/0.10</td>
<td>150/0.10</td>
<td></td>
<td></td>
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<tr>
<td>Surgery (47 mo)</td>
<td>14.5</td>
<td>100/0.8</td>
<td>30/0.4</td>
<td>30/10</td>
<td>14</td>
<td></td>
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<tr>
<td>Patient B</td>
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<td></td>
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<td>Cath 1 (1 mo)</td>
<td>3.2</td>
<td>90/0.8</td>
<td>70/0.8</td>
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<tr>
<td>Cath 2 (11 mo)</td>
<td>7.7</td>
<td>100/0</td>
<td>30/0.9</td>
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<tr>
<td>Cath 3 (22 mo)</td>
<td>9</td>
<td>80/0.7</td>
<td>24/0.4</td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Mustard (23 mo)</td>
<td>9</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>Cath 4 (28 mo)</td>
<td>10</td>
<td>15/0</td>
<td>15/8</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cath 5 (33 mo)</td>
<td>10.6</td>
<td>95/0.8</td>
<td>55/0.5</td>
<td>52/20</td>
<td>32*</td>
<td>45*</td>
<td>3</td>
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<tr>
<td>Surgery (33 mo)</td>
<td>10.6</td>
<td></td>
<td></td>
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<tr>
<td>Patient C</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Cath 1 (2 mo)</td>
<td>12.3</td>
<td>102/0.10</td>
<td>62/10</td>
<td>60/40</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mustard (55 mo)</td>
<td>12.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cath 3 (56 mo)</td>
<td>12.3</td>
<td>83/9</td>
<td>40/6</td>
<td>38/18</td>
<td>5</td>
<td>9</td>
<td>4.5</td>
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<tr>
<td>Cath 4 (71 mo)</td>
<td>15.6</td>
<td>95/0.5</td>
<td>55/0.5</td>
<td>55/30</td>
<td>32</td>
<td>32</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Surgery (71 mo)</td>
<td>15.6</td>
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*Not simultaneous.

Abbreviations: RV = right ventricle; PA = pulmonary artery; PAW = pulmonary artery wedge; LV = left ventricle; PV = pulmonary vein; PVA = pulmonary venous atrium; Qs = calculated pulmonary blood flow.
In a previous report from this institution, it was proposed that placement of the cranial portion of the posterior suture line of the baffle either too near the orifice of the SVC or too near the orifices of the pulmonary veins was an important cause of SVC and pulmonary vein obstruction, respectively.8 In this report, while only one of six (15%) of the patients with PVO documented at the time of the first postoperative catheterization also had SVO, each of the three patients with late development of PVO also had SVO. Although the position of the cranial portion of the posterior suture line may be
related to the early onset of SVO or PVO, progressive fibrosis around the dacron baffle may result in late development of both PVO and associated SVO.

Rapid postoperative body growth or increased cardiac output could result in the development of a pressure gradient between the pulmonary wedge position and PVA if the pulmonary venous outflow orifice were fixed by a rigid baffle. However, case A increased only 11.5% in weight, case B only 6% and case C 21% during the period that PVO developed. Furthermore, pulmonary blood flow ($Q_p$) remained essentially the same during this period in case A and decreased in case C after development of PVO. The increase in $Q_p$ in patient B reflects a left-to-right shunt at the atrial level.

Development of pulmonary symptoms suggestive of pneumonia or asthma may indicate development of PVO. Patients in whom PVO was demonstrated at the first postoperative cardiac catheterization developed symptoms earlier (mean 9.3 months) following surgery than those in whom PVO was demonstrated only at the second postoperative cardiac catheterization (mean 17.6 months).

The appearance of a continuous or diastolic murmur at the lower right or left sternal border may be a clue to the presence of obstruction. Prominent pulmonary venous markings roentgenographically suggest PVO. Unilateral PVO may result in decreased arterial vascular markings on the affected side. Radionucleotide scanning of the lungs may show unequal distribution of pulmonary blood flow if the obstruction is unilateral. 14

A membrane-like shadow between the confluence of the pulmonary veins and the PVA (fig. 1, left) can be seen in the lateral projection of the cineangiogram in the absence of PVO. However, when this area assumes an hourglass configuration (fig. 1, right) PVO is likely. Unequal filling of the pulmonary arteries or unilateral obstruction to pulmonary venous drainage in the anteroposterior projection of the left ventricular or pulmonary artery cineangiogram suggests PVO. Furthermore, echocardiographic determination of the

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**FIGURE 4. Patient C. Left) Anteroposterior view of LV injection demonstrating decreased flow to the left lung. Right) Lateral view of the levophase of LV injection demonstrating narrow channel between pulmonary vein and PVA.**

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**FIGURE 5. Artist’s concept of gusset enlargement of PVA. a) PVA viewed with dacron baffle in place and position of incision shown by dotted lines. b) Dacron gusset in place. c) Atrium closed following gusset placement. (Reprinted with permission from reference 5.)**
PEP/LVET ratio may be a noninvasive method of predicting elevated pulmonary artery pressure, which would suggest development of PVO.\textsuperscript{14}

It is difficult to determine how effective gusset enlargement of the PVA at the time of the Mustard operation is in preventing PVO. Champsaur et al.\textsuperscript{16} reported the development of PVO in three of 123 patients following the Mustard operation. Although 47 patients had had pulmonary venous atrialplasty at the time of the Mustard procedure, the authors did not specify if those who developed PVO had had atrialplasty performed. Two of 49 patients reported by Clarkson et al.\textsuperscript{16} developed PVO and none had had atrialplasty. Patient C in this report developed PVO despite pulmonary venous atrialplasty at the time of the Mustard procedure. Nor is gusset enlargement of the PVA to relieve PVO always successful. Although Reul et al.\textsuperscript{5} reported improvement in six patients following atrialplasty, others\textsuperscript{4, 12, 15} have noted failure of this procedure to relieve PVO in some patients.

Despite early symptomatic improvement after Mustard repair of TGA, patients require continued observation for the late development of PVO. Indeed, even with hemodynamically and angiographically documented absence of PVO one must be alert to its insidious development. Those responsible for the patient’s primary care should be aware that the first symptoms of this potentially lethal sequel are similar to those of pneumonia or asthma.

If PVO is suspected, cardiac catheterization is indicated to further investigate this possibility.

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10. Clarkson PM, Neutze JM, Barratt-Boyes BG, Brandt PWT: Late postoperative hemodynamic results and cineangiocardiographic findings after Mustard atrial baffle repair for transposition of the great arteries. Circulation \textbf{53}: 525, 1976


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