Transaortic Resection of the Subaortic Membrane*
Treatment for Subvalvular Aortic Stenosis

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In an attempt to better understand congenital subaortic stenosis, we reviewed 19 consecutive pediatric patients undergoing surgery for this problem between 1973 and 1984. Preoperative cardiac catheterization demonstrated subvalvular stenosis in all patients and associated anomalies in 11 patients. All patients underwent transaortic resection of the subaortic membrane. Five patients underwent concurrent additional cardiac procedures. An 8-month-old infant with multiple cardiac anomalies was the only operative mortality. Follow-up extended to 16 years, with a mean of 6.9 years. In five symptomatic patients, cardiac catheterization revealed a mean systolic pressure gradient of 33.0 ± 31.94 mm Hg 24.7 months (mean) after surgery. Two of these patients (11.1 percent of survivors) required reoperation. Twelve of the remaining 13 asymptomatic patients underwent echocardiographic follow-up, and 4 were found to have recurrent stenosis with 1 requiring surgery (5.0 percent of survivors). Our experience shows that transaortic resection of the subaortic membrane is an acceptable treatment for subvalvular aortic stenosis, but is associated with a high incidence of recurrence requiring reoperation (3 of 18 or 16.7 percent). Consequently, it is reasonable to consider the role of alternative therapies which may help prevent recurrence in selected cases.

Key words: subarotic stenosis, surgery

Patients and Methods

Between 1973 and 1984, 19 patients underwent 21 operations for congenital fixed subvalvular aortic stenosis. Detailed assessments were made of these 19 consecutive and unselected patients both before and after operations by reviewing their medical records, echocardiograms, and cardiac catheterization data as well as interviewing the patients, their families, and their referring physicians.

The patients' ages at surgery ranged from 8 months to 18 years, with a mean age of 8.8 years. Eleven were males and eight were females. Eight patients were asymptomatic, whereas nine presented with symptoms of congestive heart failure, and four had angina pectoris (two of whom had coexistent symptoms of failure).

Five of the patients had undergone a total of six prior cardiac operations: one patient had closure of a patent ductus arteriosus, one patient underwent resection of infundibular pulmonic stenosis, one patient underwent resection of infundibular pulmonic stenosis with repair of a ventricular septal defect, and one patient had correction of Fallot’s tetralogy. Another patient underwent two prior cardiac operations: first, closure of a patent ductus arteriosus with repair of aortic coarctation, and 18.5 months later, a second coarctation repair.

All 19 patients had systolic ejection murmurs detected during the physical examination, 10 had palpable thrills, 3 had ejection clicks, and 4 had associated diastolic murmurs. Chest radiography revealed cardiomegaly in ten patients, aortic poststenotic dilatation in three patients, and evidence of increased pulmonic flow in two patients. Electrocardiogram revealed left ventricular hypertrophy in 11 patients with associated strain pattern in 3 patients. Preoperative 2-dimensional and Doppler echocardiograms were performed in 11 patients: 7 patients had clear evidence of subvalvular aortic stenosis with 4 of these revealing discrete subvalvular lesions, and 5 patients had evidence of left ventricular hypertrophy.

All patients underwent preoperative cardiac catheterization which documented the presence of subvalvular aortic stenosis (Fig 1). Valvular aortic stenosis also was present in three patients and subvalvular aortic stenosis was demonstrated in one.

Congenital aortic stenosis represents 3 to 5 percent of all congenital heart disease.1,2 In approximately half of all cases, the aortic stenosis is valvular, one third is subvalvular, and the remainder is either supravalvular or multilevel.3,4 Both dynamic and fixed forms of subvalvular aortic stenosis have been described.5 Dynamic obstruction is produced by myocardial hypertrophy; therefore, the outflow orifice is variable in size relating to left ventricular volume and contractility. Fixed obstruction is produced by a specific anatomic malformation, and the orifice size is unchanged by physiologic variables. Fixed obstruction may be of two types. Type 1 or membranous subvalvular aortic stenosis is more common and is caused by a discrete subvalvular membrane. Less commonly, type 2, fibromuscular, or tunnel subvalvular aortic stenosis, is caused by a diffusely narrowed segment of the left ventricular outflow tract.5,6

We have reviewed the clinical, pathologic, and operative findings of all patients undergoing surgery for this problem over a 12-year period at the University of Miami School of Medicine/Jackson Memorial Medical Center in order to compare our outcome with that previously reported in the literature.

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Systolic pressure gradients across the subvalvular membrane were 74.53 ± 31.49 mm Hg. Eleven patients demonstrated a wide range of associated cardiac anomalies including three with ventricular septal defect, three with pulmonic stenosis, and two with a single main coronary artery (Table 1).

RESULTS

Transaortic resection of the subaortic membrane was performed in all patients with a mean cardiopulmonary bypass time of 78.8 ± 72.3 min (range: 35 to 302 min), systemic hypothermia at 28°C, and a mean aortic cross-clamp time of 31.0 ± 15.7 min (range: 15 to 66 min) with cold (4°C) crystalloid cardioplegia. Five patients underwent additional concurrent cardiac procedures (Table 1). There was one operative mortality (5.3 percent), an 8-month-old infant with multiple cardiac anomalies (a ventricular septal defect, an atrial septal defect, a single main coronary artery, cor triatriatum, pulmonary hypertension, and a double outlet right ventricle [Table 1]).

Pathologic examination of specimens revealed fibrous membranes or rings resected from the subvalvular region of the 18 surviving patients. The patient who died intraoperatively was found to have endocardial fibrosis, membranous subaortic stenosis, as well as previously listed anomalies (Table 1).

Postoperative complications were seen in four patients: one case of culture-negative postpericardiotomy syndrome controlled with aspirin, one case of short-term renal failure treated with temporary peritoneal dialysis prior to eventual resolution, one urinary tract infection caused by Pseudomonas organisms and treated with antibiotics, and one Staphylococcus aureus wound infection treated with surgical debridement and antibiotics.

Follow-up ranged from 1 month to 16 years, with a mean of 6.9 years. No patient demonstrated postoperative heart block or surgically induced mitral valve dysfunction. Postoperative cardiac catheterization was carried out in five symptomatic patients revealing systolic pressure gradients across the subaortic membrane of 33.0 ± 31.94 mm Hg 26.8 months (mean) after surgery (Fig 2, 3). Two of these five patients required reoperation for recurrent subaortic stenosis 23 and 34 months, respectively, after their initial operations. One of these patients also had a tubular fibrous subvalvular stenosis as well as supravalvular and subvalvular mitral stenosis (Fig 4). This patient died 34 months after the second mitral valve replacement (4.5 years after the initial operation). This patient represents the only late mortality in our series (1 out of 18 or 5.6 percent).

Of the remaining 13 asymptomatic patients, 12 underwent two-dimensional and Doppler echocardiograms. Four of these patients were found to have recurrent subaortic stenosis with systolic pressure gradients between 55 and 85 mm Hg. Of these four
patients, one underwent successful reoperation 101 months after her initial operation. A second of these four underwent catheterization 107 months after initial surgery and was found to have an insignificant aortic gradient of only 23 mm Hg. This patient is currently asymptomatic and doing well more than 13 years after the operation. A third patient refused catheterization or further intervention and is currently doing well. The fourth patient was unavailable for follow-up (Fig 5).

In our series, 6 patients had evidence of restenosis (6 of 18 or 33.3 percent of survivors; 2 symptomatic patients whose conditions were manifested by catheterization and 4 asymptomatic patients whose conditions were shown by echocardiography). Three patients required reoperation for recurrent stenosis (3 of 18 or 16.7 percent of survivors). Two of these three patients are currently doing well. The third patient expired, as previously described.

**DISCUSSION**

Fixed subvalvular aortic stenosis was first described in 1842 by Chevers.7 There are no published data documenting the natural history of unoperated fixed subvalvular stenosis.5 Campbell, however, reported a 1.4 percent per year overall death rate and 0.9 percent per year sudden death rate in the review of 2,816 collective nonsurgically treated cases of either valvular or subvalvular aortic stenosis from six separate series. He also documented the progressive nature of congenital aortic stenosis with continued worsening of the patients' functional status with time.9

Moses and associates6 reported long-term survival

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**Table 1—Patient Data***

<table>
<thead>
<tr>
<th>Case</th>
<th>Associated Anomalies</th>
<th>Operative Findings</th>
<th>Additional Concurrent Procedures</th>
<th>Subvalvular Pathologic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>Fibrous ring immediatly below aortic valve</td>
<td>None</td>
<td>Fibrous ring</td>
</tr>
<tr>
<td>2</td>
<td>SMCA</td>
<td>Fibrous subvalvular ring beneath aortic cusps</td>
<td>None</td>
<td>Fibrous membrane</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>Subvalvular membrane</td>
<td>None</td>
<td>Fibrous band</td>
</tr>
<tr>
<td>4</td>
<td>MS, MR</td>
<td>Subvalvular membrane</td>
<td>Correction of supravalvular and subvalvular MS, mitral valve replacement</td>
<td>Fibrous membrane</td>
</tr>
<tr>
<td>5</td>
<td>PDA</td>
<td>Fibrous membrane beneath aortic valve</td>
<td>None</td>
<td>Fibrous membrane</td>
</tr>
<tr>
<td>6</td>
<td>VAS</td>
<td>Subvalvular fibrous tissue 0.5 cm below aortic valve</td>
<td>Valvuloplasty (commissurotomy)</td>
<td>Fibrous membrane with focal mucinous degeneration</td>
</tr>
<tr>
<td>7</td>
<td>PS</td>
<td>Discrete fibrous ring below aortic valve</td>
<td>None</td>
<td>Fibrous ring with myxomatous changes and hyalinization</td>
</tr>
<tr>
<td>8</td>
<td>VAS</td>
<td>Subaortic fibrous membrane</td>
<td>Valvuloplasty (commissurotomy), closure of ASD</td>
<td>Fibrous ring with focal hyalinization</td>
</tr>
<tr>
<td>9</td>
<td>None</td>
<td>Fibrous subaortic ridge 1.0 cm below aortic valve</td>
<td>None</td>
<td>Fibrous ridge</td>
</tr>
<tr>
<td>10</td>
<td>None</td>
<td>3.0-mm thick fibrous ring circumferentially below aortic valve</td>
<td>None</td>
<td>Dense and loose connective tissue</td>
</tr>
<tr>
<td>11</td>
<td>None</td>
<td>Fibrous membrane 1.0 cm below aortic valve</td>
<td>None</td>
<td>Fibrous membrane</td>
</tr>
<tr>
<td>12</td>
<td>VSD</td>
<td>Fibrous subaortic membrane</td>
<td>None</td>
<td>Fibrous membrane, endocardium</td>
</tr>
<tr>
<td>13</td>
<td>None</td>
<td>Fibrous ridge 1.0 cm below aortic valve</td>
<td>None</td>
<td>Fibrous ridge, cellular and myxoid fibrous tissue</td>
</tr>
<tr>
<td>14</td>
<td>SMCA, PH, ASD, VSD, CTA, DORV, SAS</td>
<td>DORV with fibrous subaortic stenosis</td>
<td>Repair of SAS, enlargement of left atrium</td>
<td>Endocardial fibrosis</td>
</tr>
<tr>
<td>15</td>
<td>VAS, PS, COA</td>
<td>Fibrous area 1.0 cm below aortic valve</td>
<td>None</td>
<td>Fibrous membrane</td>
</tr>
<tr>
<td>16</td>
<td>None</td>
<td>Fibrous ridge 2.0 cm below aortic valve: 270°</td>
<td>None</td>
<td>Fibrous ridge, fibrointimal hyperplasia</td>
</tr>
<tr>
<td>17</td>
<td>TOF</td>
<td>Subvalvular membrane</td>
<td>None</td>
<td>Fibromuscular band</td>
</tr>
<tr>
<td>18</td>
<td>None</td>
<td>Fibrous ring 1.0 cm below aortic valve</td>
<td>None</td>
<td>Fibrous ridge, focal myxoid degeneration</td>
</tr>
<tr>
<td>19</td>
<td>VSD, PS</td>
<td>Subvalvular membrane</td>
<td>VSD correction</td>
<td>Fibrous band</td>
</tr>
</tbody>
</table>

*SD=atrial septal defect; COA=coarctation of aorta; CTA=cor triatriatum; DORV=double outlet right ventricle; MR=mitra regurgitation; MS=mtral stenosis; PDA=patent ductus arteriosus; PH=pulmonary hypertension; PS=pulmonic stenosis; SAS=supravalvular aortic stenosis; SMCA=single main coronary artery; TOF=Fallot's tetralogy; VAS=valvular aortic stenosis; VSD=ventricular septal defect. |
Figure 3. Left ventriculogram in end systole in a 20-year-old patient 26 months after transaortic resection of subaortic membrane. There was no postoperative pressure gradient between the left ventricle and the ascending aorta. A: Right oblique view. B: Left oblique view.

ranging from 63 to 93 percent after surgery for subvalvular aortic stenosis in a collective review of eight series. The two patients who died in our series had multiple cardiac anomalies. The majority of our survivors (13 of 18 or 72 percent) have remained asymptomatic since their initial operations; some are active in sports, and at least one is known to have delivered a baby.

Transaortic resection of the subaortic membrane has been the standard surgical approach for treating subvalvular aortic stenosis for many years.10-12 This procedure carries a very low operative mortality.5,8,13,14 However, certain potential complications must be avoided. When excising the subaortic membrane, certain anatomic relationships must be kept in mind. Beneath the noncoronary cusp, the membrane may be attached to the ventricular septum; excessive excision in this area can perforate the septum and damage the conduction bundle causing either a ventricular septal defect, heart block, or both. Be-

Postoperative Echocardiographic Studies In 12 Asymptomatic Patients

4 Patients With Recurrent Subaortic Stenosis
- systolic pressure gradient of 55 mm Hg to 85 mm Hg

8 Patients Without Recurrent Subaortic Stenosis

1 Patient Underwent Successful Reoperation

1 Patient Managed Nonoperatively
- no significant restenosis by catheterization

1 Patient Refuses Further Workup

1 Patient Lost to Follow-Up

Figure 4. Postoperative left ventriculogram in patient 4 showing tubular subaortic stenosis (arrow).

Figure 5. Postoperative echocardiography was performed in 12 asymptomatic patients. Only 4 had evidence of recurrent stenosis and only 1 required reoperation.
neath the left coronary cusp lies the base of the aortic leaflet of the mitral valve; overaggressive excision here can lead to mitral valve damage.\textsuperscript{13}

Postoperative recurrence of the subvalvular aortic stenosis has been reported to occur at rates ranging from 5.5 to 36 percent.\textsuperscript{8,11,14-16} Three of our patients (16.7 percent) required reoperation for recurrent stenosis. Because of high recurrence rates, some authors view this operation merely as palliative.\textsuperscript{13} Moreover, some believe that untreated or inadequately treated membranous obstruction may actually progress to fibromuscular tunnel obstruction due to fibromuscular proliferation in the area of the left ventricular outflow tract.\textsuperscript{14} This concept leads to the theory that the Kelly classification of two types of subvalvular aortic stenosis is actually an oversimplification and that in reality type 1 membranous obstruction and type 2 tunnel obstruction actually represent two ends of a spectrum of anatomic entities causing subvalvular aortic stenosis.\textsuperscript{17} Our patient who required reoperation 23 months after his initial operation demonstrated this progression of disease. Initially he underwent resection of the subaortic membrane, but 23 months later he underwent resection of the subaortic tubular fibrous tunnel.

Because of the potential for recurrent subaortic stenosis, we feel that echocardiography offers an important noninvasive method to serially follow patients after transaortic resection of the subaortic membrane. Echocardiography can identify the presence of subvalvular stenosis and its anatomic type.\textsuperscript{18} As shown by our series, patients with surgically corrected subvalvular aortic stenosis may develop restenosis and remain asymptomatic. Echocardiography represents an inexpensive and noninvasive method of screening for recurrent stenosis in those asymptomatic patients.

Since transaortic resection of the subaortic membrane is associated with a significant recurrence rate, it is reasonable to consider alternative therapies for this disease process. Other surgical approaches have been applied to correct subvalvular aortic stenosis, especially in complex cases. Cain and colleagues\textsuperscript{14} reported on the use of resection of the subvalvular membrane combined with left ventricular myectomy as the primary treatment for subaortic stenosis. They feel that purely membranous obstructions are rare and that hypertrophic muscular obstruction often is coexistent. However, they found no significant difference in recurrence rates between subaortic membrane resection alone and subaortic membrane resection combined with myectomy, although the trend favored adding myectomy.\textsuperscript{14} Furthermore, Lupinetti and associates\textsuperscript{15} recently reported markedly reduced recurrence rates when myectomy was added to membrane excision during the original operation.

The procedure of Konno et al\textsuperscript{19} was first described in 1975 for patients with aortic stenosis associated with hypoplasia of the aortic valve annulus. This procedure involves excision of the aortic valve, opening of the left ventricular outflow tract, annulus, and aorta, and then reconstruction with patches and a prosthetic valve.\textsuperscript{20} Also, Rastan and Knoocz\textsuperscript{21} described aortoventriculoplasty as a technique which creates a surgical aortoseptal defect which is then patched with knitted Dacron in such a way as to provide the largest possible outflow to the left ventricle. In a later study, Rastan and colleagues\textsuperscript{22} reported that aortoventriculoplasty combined with aortic valve replacement is an effective operation for complex types of subvalvular stenosis resulting in relief of obstruction in the overwhelming majority but with potentially higher mortality and complication rate than subvalvular membrane resection alone. Others also have used aortoventriculoplasty for a variety of indications ranging from usage for all forms of congenital valvular and subvalvular aortic stenosis to usage for complex cases of subvalvular stenosis including reoperations and tunnel obstructions.\textsuperscript{14} Vouhe and colleagues\textsuperscript{23} reported a modification of the Rastan aortoventriculoplasty in which an aortic valve replacement is avoided as is the usage of the prosthetic patch. They avoided excising the aortic valve by dividing the aortic annulus across the commissure between the right and left aortic cusps so as to preserve the aortic leaflets. The use of prosthetic mesh is avoided by modifying the various incisions so that they can be closed primarily without patching the left ventricular outflow tract. They feel this aortoseptal modification may be the procedure of choice for diffuse stenosis limited to the subvalvular area and that aortoventriculoplasty as described by Konno et al,\textsuperscript{19} Misbach et al,\textsuperscript{20} and Rastan and colleagues\textsuperscript{21,22} is still applicable for tunnel lesions which also have involvement of valvular or supravalvular levels. Recently, Vouhe and Neveux\textsuperscript{17} reported that the anatomic features of the subvalvular aortic stenosis will determine their choice of surgical intervention: transaortic resection of the subvalvular membrane is used for localized discrete lesions and aortoventriculoplasty is used for diffuse lesions with preservation of the native aortic valve whenever possible.

We feel that transaortic resection of the subvalvular membrane is an acceptable method of treatment for fixed membranous subvalvular aortic stenosis, but is associated with a significant incidence of recurrence. Other more complex procedures are available for patients with more complicated types of subvalvular aortic stenosis, such as tunnel subvalvular stenosis, recurrent subvalvular stenosis, and stenosis with hypoplastic aortic annulus. As experience with...
these more complex procedures grows, their application may be broadened to replace or complement transaortic resection of the subvalvular membrane in selected patients to avoid the problem of recurrence.

Conclusion

Transaortic resection of the subaortic membrane is an acceptable treatment for fixed membranous subvalvular aortic stenosis, but is associated with a high incidence of recurrence. Echocardiography represents a noninvasive and inexpensive method to follow up patients serially after transaortic resection of the subaortic membrane. Mortality associated with fixed membranous subvalvular aortic stenosis often is related to coexisting cardiac anomalies. Many alternative surgical options are available for patients with more complex types of subvalvular stenosis or recurrent stenosis. In time, these alternative procedures may replace or complement transaortic resection of the subaortic membrane in selected cases to avoid the problem of recurrence.

References

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