Discrete Subaortic Stenosis

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SUMMARY
Twenty-five patients with discrete subaortic stenosis were reviewed. Twenty were
operated on for severe obstruction. Review of available data suggests there is a wide
spectrum of the disease which may be divided into two main types designated I and II.
Type I is a thin discrete membrane immediately under the aortic valve obstructing the
outflow but not associated with narrowing of it. Type II is situated about 1 cm below the
valve and consists of a fibrous ring, thicker than type I, and associated with muscular hy-
pertrophy which narrows the outflow tract, encroaches on the anterior leaflet of the mitral
valve, and may extend 1–2 cm downward. Both have a characteristic angiographic ap-
pearance. Results of surgery are good for type I but not satisfactory for type II. Aortic
incompetence is likely to increase or develop following operation in both types.

Additional Indexing Words:
Angiographic classification type I and type II
Left ventricular hypertrophy
Aortic regurgitation

Discrete subaortic stenosis was
described by Chevers in 1842 and
is a frequent cause of left ventricular outflow
obstruction. It occurs about one fifth as often
as valvular stenosis. The exact etiology
remains in doubt. The traditional explanation
has been that subaortic fibrous stenosis
resulted from incomplete atrophy of the
bulbus cordis. However, Van Praagh has
stated that conal or infundibular tissue in the
definitive heart is muscular, not fibrous, and
that fibrous subaortic stenosis results from
maldevelopment of the endocardial cushion
tissue of the atrioventricular canal that usually
forms the anterior leaflet of the mitral valve.
From review of reported cases there appear
to be many types of discrete subaortic
obstruction. Among these are abnormalities of
the anterior leaflet of the mitral valve in its
attachments or from accessory tissue on it.
A discrete, soft, fibrous or fibromuscular ring
may be attached to the upper margin of the
anterior leaflet. A narrow outflow tract in
association with the above has also been
reported. Because the customary transaortic
operative approach limits visibility, the exact
definition of the type and spectrum of discrete
subaortic stenosis is incomplete. The variable
extent of surgery, often with removal of the
mitral valve, makes analysis imprecise. Data
on both the variation in anatomic types of this
disorder as seen at surgery, and on the results
and long-term follow-up of operated cases are
surprisingly sparse.

Methods
Twenty-five patients with discrete subaortic
stenosis were seen over a 26-year period. Patients
with supravalvar, valvar, or hypertrophic muscular
subaortic stenosis were excluded. Hemodynam-
ically significant obstruction to left ventricular
outflow was proven by left heart catheterization
in all except one patient. Anatomic confirmation
of discrete subvalvar stenosis was obtained in 21, one at autopsy and the rest at surgery. In
the remaining four, who have not been operated
on, left ventricular angiography showed the characteristic findings.

**Preoperative Evaluation**

**Patient Data**

Thirteen patients presented under 1 year of age, 11 between 1 and 5 years of age, and one at the age of 12. All were identified initially because of loud cardiac murmurs and were thought to have aortic stenosis. Associated congenital heart defects were found in six patients. Two had a patent ductus arteriosus; other defects were coarctation of the aorta, pulmonary stenosis, sinus of Valsalva aneurysm which later ruptured causing death, a small ventricular septal defect, and dextroversion with persistent left superior vena cava.

**Symptoms**

Eleven presented with dyspnea; in nine of these it was grade I and in two, grade III (New York Heart classification). Four had associated chest pain and one, syncopal attacks. Despite the difficulty of obtaining an accurate history of chest pain in children and adolescents, all four were judged to have angina pectoris. All proved to have severe outflow obstruction as did the patient with syncope on exertion.

![Figure 1](Circulation, Volume XLVI, August 1972)

*Figure 1*

Case 7. Phonocardiograms are taken with the patient supine, with simultaneous recording at lead II of the ECG and indirect right carotid arterial pressure. (A) Preoperative: sound recordings are taken at the second left sternal edge (2LSE) and the apex at high frequency (HF). At the 2LSE the aortic component of the second sound \( (A_2) \) is of less intensity than the pulmonary \( (P_2) \). There is no ejection sound, but an ejection systolic murmur is present. (B) Postoperative: In the same patient the same technic is used as in A. The aortic component of the second sound \( (A_2) \) has increased in relation to the pulmonary \( (P_2) \).
Physical Findings

Peripheral pulses were judged normal in all. Twenty-three of 25 patients had a prominent left ventricular lift. A precordial systolic thrill felt in all patients was usually present over the base of the heart and transmitted to the suprasternal notch. This was accompanied by a widespread, long, ejection-type systolic murmur peaking in late systole, radiating to the carotid arteries. The thrill and the murmur differed neither on auscultation nor on phonocardiogram, from the murmur associated with aortic valvar stenosis. One patient had an aortic ejection sound. Early diastolic murmurs were heard in nine patients before operation, and seven patients had middiastolic apical murmurs. The second sound was split normally in 21 patients but was single in four. The aortic component of the second sound was noticeably soft in many (fig. 1). Atrial sounds were heard at the apex in five patients. These patients and those who had single second sounds had severe outflow obstruction.

Radiology

Preoperative anteroposterior and lateral chest X-rays were available in 24 patients. Left ventricular prominence was present in 17 patients. No marked cardiomegaly was noted in the series and there was no correlation between general heart size and left ventricular aortic pressure difference. The ascending aorta and arch were dilated in seven of 24 patients, and this was noted first at as early as 3 years of age. Slight enlargement of the left atrium was found on barium swallow in six of the 24 patients. No calcification was noted on fluoroscopy.

Electrocardiograms

Twenty-four had sinus rhythm present, one had nodal rhythm, five had left atrial hypertrophy, and all had normal P-R interval. The average duration of QRS was 0.06 sec. The QRS frontal axis varied between +5 and +90. Sixteen patients had a pattern described as severe left ventricular hypertrophy with strain, but two with severe obstruction requiring operation had cardiograms which did not suggest much hypertrophy (fig. 2). Of two patients with mild obstruction thought not to require operation, one had a moderately severe left ventricular hypertrophy pattern and one had a normal electrocardiogram and vector, which had remained normal over a period of 15 years.

Postoperative Evaluation

Symptoms

All patients except one have been clinically reviewed within the last year. Four were symptomatic. Three of these had dyspnea and chest pain thought to be angina pectoris, and on restudy were found to have severe

Figure 2

Electrocardiogram of the patient who had a gradient of 100 mm Hg across the LV outflow tract. There is some increase in R-wave amplitude in the lateral chest leads but tracing is within normal limits.
obstruction. Two have had further surgery. The fourth symptomatic patient was a 12-year-old girl who presented initially with severe heart failure. Since operation she has had complete heart block which required pacing, and has severe aortic incompetence with grade 1 dyspnea.

### Physical Signs

All patients had residual aortic ejection murmurs. In those who had satisfactory reduction of gradient the murmur was reduced from IV-V/VI to I-II/VI. Seven patients developed an early diastolic murmur of aortic incompetence when none was heard before.

### Table 1

**Hemodynamic Data**

<table>
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<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Method</th>
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<th>Gradient</th>
<th>Output (liters/min)</th>
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<td>80</td>
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<td>LVP</td>
<td>256/17</td>
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<td>—</td>
</tr>
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<td>LVP</td>
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<td>91</td>
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<td>170/9</td>
<td>70</td>
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<tr>
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<td>92</td>
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<tr>
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<td>80</td>
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</tr>
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<td>33</td>
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<td>137/8</td>
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<tr>
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<td>TS</td>
<td>162/15</td>
<td>63</td>
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<td>23 Preop</td>
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<td>Retro</td>
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<td>49</td>
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</tr>
<tr>
<td>Preop 14</td>
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<td>141/21</td>
<td>52</td>
<td>6.2</td>
</tr>
<tr>
<td>24 Preop†</td>
<td>5</td>
<td>Retro</td>
<td>236/12</td>
<td>145</td>
<td>—</td>
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<tr>
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<td>142/7</td>
<td>23</td>
<td>4.5</td>
</tr>
<tr>
<td>Preop 17</td>
<td></td>
<td>TS</td>
<td>132/18</td>
<td>41</td>
<td>—</td>
</tr>
</tbody>
</table>

* Died of unrelated causes.
† Died preoperatively.

Abbreviations: TS = transeptal; Retro = retrograde; LVP = left ventricular puncture.
An additional eight who had signs of aortic incompetence preoperatively had early diastolic murmurs remaining. In one the diastolic murmur heard preoperatively had disappeared and aortography confirmed that there was no aortic incompetence. In two the aortic incompetence had increased significantly and was graded as moderate to severe. Four patients, all with residual obstruction, had middiastolic murmurs, but in three the middiastolic murmur had disappeared after resection of the obstruction.

No significant changes were noted in the postoperative radiographs. When the obstruction had been successfully removed, the left ventricular hypertrophy pattern on the electrocardiogram regressed postoperatively.

Hemodynamics

Preoperative

Twenty-four of the 25 patients were studied preoperatively. Fifteen had right heart catheterization, and in four who had severe stenosis the pulmonary artery pressures were abnormal. Twenty-four patients had left ventricular pressures measured. The results are shown in table 1. Two of three patients who are living and have not had surgery were restudied after a 4-year interval. In one the electrocardiogram suggested increasing left ventricular hypertrophy; in the other it had remained the same. The obstruction had not appreciably altered in either. The end-diastolic pressure was elevated in 11 patients. Since only one had clinical left ventricular failure, in the others this elevation may represent change in compliance secondary to left ventricular hypertrophy. The cardiac output was available in 13 patients and was within the normal range, except in case 20 with severe left ventricular failure.

Postoperative

Postoperative catheterization was done in 13 of the 20 who had undergone surgery. Figure 3 shows the change in left ventricular outflow gradient in the 12 patients in whom it was measured before and after surgery. In five, severe obstruction remained, and in three it had increased. Two of the five had elevated right heart pressures.

Angiocardiography

Preoperative Left Ventriculograms

Discrete subaortic stenosis can be divided into two distinct types on angiographic criteria in the 17 patients who had it.

Type I

A thin, discrete membrane located immediately below the aortic valve with a structurally normal outflow tract and normal mitral valve movement. This was seen in five patients, all of whom had little or no residual gradient left after operation (figs. 4, 5).

Type II

A fibromuscular stenosis associated with considerable muscular hypertrophy and narrowing of the outflow tract. This was seen in 12 patients and appeared lower in the outflow tract than in patients with type I stenosis, usually involving the anterior leaflet of the mitral valve. Discrete obstruction was often difficult to see in these angiograms, but the persistence of a filling defect in various phases of the cardiac cycle at the distal end of the narrowing distinguished this from a pure
muscular left ventricular outflow obstruction. In all cases except one (case 19) the mitral valve appeared to move normally, and the incompetence and massive septal hypertrophy seen with muscular obstruction were not seen. The severity of involvement varied but always had a characteristic appearance (figs. 6–10).

Postoperative Left Ventriculograms

Twelve of the 20 patients had postoperative ventriculograms. In type I little residual stenosis could be seen. In type II, although the discrete filling defect was no longer present, considerable abnormality with narrowing of the outflow tract remained (figs. 8, 9).

Aortography

Preoperative

Seventeen aortograms were done in the 24 patients who had preoperative hemodynamic assessment. In seven there was a grade I incompetence, which had been noted clinically.

Postoperative

Twelve patients had aortograms and 10 showed aortic incompetence. In one patient (type I) it had disappeared, but had appeared in four when not present preoperatively. The degree of incompetence had increased in six, and in three was now grade II.

Operative Findings

Twenty patients had surgery (table 2). At operation the aortic valve was thought to be normal in 12 patients. In two, a bicuspid valve was present, one with associated minor stenosis and the other normal. In six some tethering and thickening of the aortic valve was observed. In 12 patients the fibrous ring was 1 cm or more below the aortic valve cusps, and the stenosis was described in these
patients as fibromuscular. This varied in severity and length from a circumscribed, almost discrete fibrous channel with considerable abnormal muscular hypertrophy underneath. The fibromuscular ring extended to the anterior leaflet of the mitral valve. All had severe narrowing of the outflow tract. One patient (case 15) with dextrocardia previously operated on via a transaortic approach had further surgery. Because of the posterior location of the aorta, a ventriculotomy was done, and the fibrous obstruction was excised through this incision. Accessory valve leaflet tissue which lay on the anterior aspect of the mitral valve, obstructing the left ventricular outflow tract, was easily identified and removed through this approach without compromising the valvar function. If the superior transaortic approach had been used, this structure would have been identified as part of the anterior leaflet of the mitral valve rather than as an appendage of it, and the whole valve would have been removed. One patient was said to have anomalous attachment of the anterior leaflet which obstructed the outflow tract, and the valve was removed. She was the only patient with mitral incompetence demonstrated preoperatively, but this was grade I. In eight of 20 patients, hypertrophied muscle was removed in the left ventricular outflow tract, mainly in the region of the septum.

In eight there was a more discrete fibrous ring located a few millimeters beneath the aortic valve cusps not accompanied by a fibromuscular hypertrophy and narrowing of the outflow tract. Considerable hypertrophy of the left ventricular muscle was seen, but it did not seem to narrow the outflow tract in the same way as the predominantly fibromuscular obstructions which did involve or attach to the anterior leaflet of the mitral valve. This discrete fibrous ring was often connected by a strand to one aortic cusp.

Discussion

We have elected to divide the subaortic stenosis into two distinct types, as there appear to be several distinctive features in
Figure 6

Case 20. Left ventriculogram in the lateral projection; illustrates type II stenosis. Systole showing an extremely narrow outflow tract well below the valve cusps (at arrow) and the long segment of stenosis. The myocardial wall is very thick. The insert illustrates type II obstruction in the lateral projection. Ao = aorta; AL = anterior leaflet of the mitral valve; PL = posterior leaflet; LA = left atrium.

Each group. The salient features are illustrated in figures 4–9. Type I is a discrete, usually pliable fibrous ring immediately under the aortic cusps; it may or may not have some continuity with one of these cusps. The ring is 1–2 mm thick above the muscular portion of the outflow tract. The ring extends to the superior part of the anterior leaflet of the mitral valve. There is no narrowing of the outflow tract caused by excessive fibrous and muscular tissue. The membrane is easily excised in toto at surgery, and resection of the further adjacent tissue in the outflow is not necessary. On recatheterization there is little or no residual obstruction, and the cardiogram, which rarely manifests the degree of hypertrophy seen in the other type, usually returns to normal. Clinically, it is usually indistinguishable from aortic valve stenosis, although a diminished aortic component of the second sound and absence of an ejection sound may suggest it. At investigation, unless care is taken even on direct catheter withdrawal, the basic pressure contours may suggest aortic valve stenosis because the membrane is so close to the valve. On the left ventriculogram a thin linear obstruction (figs. 4, 5), which is often difficult to see, is present as a persistent filling defect in all phases of the cardiac cycle immediately under the aortic valve. Cineaortography may or may not show some degree of aortic incompetence, but the normal excursion of the nonthickened valve cusps in the presence of severe dynamic
obstruction is a helpful diagnostic feature (figs. 4, 5). Nine of 25 patients are in this group, and eight have had successful surgery.

The remaining patients are classified as type II (figs. 6–9). This obstruction is at a lower site, usually 1 cm or more below the aortic valve. Although it is essentially a fibrous ring, moderate muscular obstruction with narrowing of the left ventricular outflow tract is present, and the narrowing may extend for 1–2 cm below the fibrous ring. The membrane is attached to the anterior leaflet of the mitral valve and is often very fibrous and thick compared to the usually more soft and pliable membrane of type I. Accessory mitral valve tissue may be present as in case 15. Clinically, there are signs of aortic stenosis, but unlike type I severe left ventricular hypertrophy is usually present both clinically and on electrocardiogram. No patient had satisfactory resolution of the left ventricular hypertrophy pattern after surgery as did those with type I. This may reflect the residual obstruction. The seven patients who had midstiastic murmurs were all in this group. At operation, although there was a localized fibrous or fibromuscular ring present, this merged into a narrowed, hypertrophied outflow, and usually some muscular tissue was resected.

After surgery the physical signs suggest significant obstruction remains, and, in some, increased aortic regurgitation. At postoperative catheterization (table 1) significant residual obstruction was present in all, and in three it had actually increased (fig. 3). Two of these were thought not to have significant...
Figure 8
Case 20. Left ventriculogram in the lateral projection; illustrates type II stenosis. Postoperative when left ventricular pressure had been lowered from 296 to 161 mm Hg: during systole there is considerable relief of the stenosis but the outflow is still narrowed. A transvenous pacemaker is in the right ventricle.

localized obstruction at reoperation, but the outflow tract had a fibrous or "woody" narrowing 1–2 cm long. Some of this fibrous tissue plus muscle was removed, but extensive resection was not possible. The mitral valve was thought to be normal in each. Left ventricular pressures prior to a second operation in these patients were 189/20 and 310/25 mm Hg, which indicated considerable systolic dynamic obstruction. At operation the findings did not resemble hypertrophic muscular obstruction in that the outflow was narrow and covered with dense fibrous tissue and the disproportional hypertrophy of the septum was not present, and preoperatively the pulse contours did not suggest it. Clinically both patients now have signs of significant residual obstruction. In the noncontracting heart dur-

Figure 9
Case 20. Left ventriculogram in the lateral projection; illustrates type II stenosis. Postoperative diastole: the subvalvar filling defect is now absent.

ing cardiopulmonary bypass it appears that, as in the condition of muscular subaortic obstruction, the severity of the outflow narrowing cannot always be appreciated.

We feel that these cases all have the same type of pathology but represent varying grades of it, and the most extreme form is the left ventricular tunnel where the left ventricular outflow tract appears hypoplastic with fibrous thickened endocardium and muscular narrowing. A small stenotic valve ring may also be present. Reis and associates described nine patients with tunnel stenosis and showed that when accompanied by discrete subvalvar obstruction the surgical results are satisfactory. We have had only one patient (case 13) with valvar and subvalvar obstruction. Operation was ineffective and has not been recommended again. Our two other patients described above with long narrow
outflow tracts and discrete subvalvar obstruction, but normal valves and valve rings, have not had their obstruction satisfactorily relieved.

Two patients did appear to have associated anomalies of the mitral valve. In one with grade I mitral incompetence, when her subaortic stenosis was resected, the mitral valve was thought to be leaking significantly and the attachments were obstructing the outflow tract. It was removed. Severe outflow obstruction was still present at postoperative catheterization. The second patient was the boy with dextrocardia who had anomalous mitral valve tissue lying on the anterior mitral valve, obstructing the left ventricular outflow tract. This could be resected easily at repeat surgery when a left ventricular, rather than a transaortic, approach was used because of the malposition of his heart and great vessels.

With this different surgical approach the exact mitral valve anatomy could be seen. When the transaortic approach is used, accessory tissue may be identified as an abnormal anterior leaflet of the mitral valve rather than an appendage to a normal leaflet, and may modify surgical treatment. A previous report has stressed the difficulty in assessing the obstructive element at operation in these examples of accessory mitral valve tissue. Anomalous insertion of mitral chordae or papillary muscle was not seen.

Although one patient (type I) had mild aortic regurgitation which disappeared after operation, and in each instance was confirmed by aortogram, seven developed mild regurgitation following surgery. Three had mild regurgitation preoperatively; now it is moderate to severe. The aortic valve, therefore, seems to be either damaged at the time of
### Table 2

**Operative Findings**

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<td>Normal</td>
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<tr>
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<td>6</td>
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<td>Normal</td>
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</tr>
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<td>1969</td>
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</tr>
<tr>
<td>6</td>
<td>1969</td>
<td>3</td>
<td>Normal</td>
<td>Normal</td>
<td>2-mm high thin membrane</td>
<td>Hypertrophied muscle removed</td>
</tr>
<tr>
<td>7</td>
<td>1970</td>
<td>12</td>
<td>Cusp thickening</td>
<td>Normal</td>
<td>High thin</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>1970</td>
<td>12</td>
<td>Cusp thickening</td>
<td>Normal</td>
<td>Thin, 2 mm under aortic cusps</td>
<td>—</td>
</tr>
</tbody>
</table>

| **Type II:** | | | | | | |
| 9   | 1957 | 18 | Normal | Not described | 3 cm below valve, thick | — |
| 10  | 1958 | 14 | Normal | Normal | 2 cm below valve | — |
| 11  | 1958 | 9  | Normal | Not described | 2 cm below valve, fibrous | — |
| 12  | 1958 | 13 | Normal | Not described | 1 cm below valve | — |
| 13  | 1959 | 10 | Cusp thickening | Not described | 5 mm thick, 2 cm below valve | — |
| 14  | 1960 | 5  | Normal | Normal | 1-cm long fibrous tunnel, thin membrane extending to aortic valve | Hypertrophied muscle excised |

| 15* | 1962 | 7  | Normal | Attached to anterior leaflet | Thick, 1 cm below valve, extending 1.5 cm down | — |
| 16* | 1965 | 9  | Cusp thickening | Ring attached to anterior leaflet | 1 cm below aortic valve, extending 1.5 cm down | — |
| 17  | 1965 | 6  | Normal | Not described | 1 cm below valve, extending down | — |
| 18* | 1967 | 8  | Cusp thickening | Adherent to anterior leaflet | Long fibrous stenosis, 4 mm thick | — |
| 19  | 1967 | 7  | Normal | ? Anomalous attachment to anterior leaflet | 2 cm below valve, small outflow | — |
| 20  | 1969 | 12 | Cusp thickening, abnormal | Attached to leaflet | 1 cm below valve, thick fibromuscular | — |

*All had reoperation.
surgery, or resection of the membrane causes damage to the closing mechanism and is a significant factor postoperatively. Six aortic valves showed some thickening. This is thought to be due to turbulence of blood flow from the severe stenosis below it. Aortic regurgitation has been postulated to be due to the same mechanism. However, many with normal valves at operation had incompetence prior to surgery, and the turbulence of stenotic lesion just under the valve probably hinders the closing of a normal valve and thus produces regurgitation. In type I, strands were noted to attach to the valve, and this too may be responsible for failure of complete cusp closing. In the one patient who had regurgitation preoperatively and now afterward, such a strand was noted and excised at surgery. Bacterial endocarditis with appearance of severe regurgitation has been noted with discrete subaortic stenosis, but no example was seen in our series.

Associated congenital heart defects were found in six patients. Two had patent ductus arteriosus and one, coarctation of the aorta in association with a bicuspid aortic valve. The valve showed no stenosis and the discrete obstruction was type I.

In a review of congenital aortic stenosis, exertional dyspnea was found to occur commonly with a gradient of 70 mm Hg or over. Sixteen of our patients had symptoms. This is unusual in patients presenting in the pediatric age group with left ventricular outflow obstruction and does emphasize that discrete subaortic stenosis tends to be severe rather than a moderate-to-mild lesion. Only three of the 25 patients were thought not to require operation.

Discrete subaortic stenosis is difficult to distinguish clinically from aortic valve stenosis, but the absence of an aortic ejection sound may be a helpful diagnostic point. One of our 25 patients did have an aortic ejection sound documented by phonocardiogram, but it may have come from the associated bicuspid valve. Severe aortic valve stenosis may not have an aortic ejection sound because of lack of mobility of the valve. Therefore, in severe obstruction, which is the usual finding in discrete subvalve stenosis, the presence or absence of an ejection sound may not be as diagnostically helpful as previously thought.

Diastolic murmurs from aortic regurgitation are stated to occur more frequently than valvar stenosis in fixed subvalvar obstruction, but this too is not a distinguishing feature. The majority of our patients preoperatively did not have early aortic diastolic murmurs or aortic incompetence on aortography. Seven patients, however, had middiastolic rumbles audible at the apex. This sign was noted only in type II obstruction. We felt this was a helpful sign and that it suggested some interference with left ventricular filling due to mitral valve abnormality, presumably due to the encroachment of the stenosis on the anterior leaflet of the mitral valve tissue or to an anomaly of the mitral valve. In three of this diastolic murmur disappeared after surgery. This may suggest satisfactory relief of obstruction because in the four where it remained severe obstruction persisted. Diminished intensity of the aortic second sound (fig. 1) may also be helpful in separating valvar from subvalvar stenosis.

The overall heart size of patients with congenital aortic stenosis is usually normal, or the degree of enlargement is minimal. Left ventricular prominence was judged to be present in all of our patients, even though the cardiothoracic ratio was within normal limits. These findings are in agreement with those of Braunwald who observed 75% of patients with subvalvar aortic stenosis showed mild-to-moderate cardiac enlargement, whereas the finding was observed in only 39% of those with valvar obstruction. This again suggests the severity of the subaortic stenosis group. The ascending aorta is said not be dilated in this condition but usually is in valvar aortic stenosis, so that the finding of a dilated ascending aorta strongly suggests valve obstruction. However, in 25% of our patients the ascending aorta was thought to be abnormally dilated for the age.

The electrocardiogram is thought to be a valuable means of assessing severity of left
ventricular outflow obstruction.\(^{17, 18}\) There are many contrary views, however, and patients who have normal electrocardiograms have been known to die of severe outflow obstruction.\(^{19, 20}\) Hugenholtz and associates\(^ {21}\) reported that of their patients with severe congenital aortic stenosis only three fourths had electrocardiographic evidence of left ventricular hypertrophy and one fourth with mild stenosis exhibited electrocardiographic evidence of left ventricular hypertrophy. In our series, all patients who had type II subaortic stenosis had severe left ventricular hypertrophy and strain. However, in two of the patients with significant type I obstruction which required operation, the cardiograms did not reflect the severity (fig. 2), and one patient (type II) who had only mild outflow obstruction on direct measurement had a severe left ventricular hypertrophy pattern. Thus, it would appear that in discrete subaortic stenosis as in valvar stenosis the electrocardiogram is not a reliable index of the severity of outflow obstruction in the pediatric age group.

**Acknowledgment**

We would like to thank Drs. Vincent Gott, Harvey Bender, Jerome Krovetz, and Glenn Rosenquist for their help.

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*Circulation, Volume XLVI, August 1972*
Discrete Subaortic Stenosis
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doi: 10.1161/01.CIR.46.2.309
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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