**Congenital Mitral Insufficiency**

*By Norman S. Talner, M.D., Aaron M. Stern, M.D., and Herbert E. Sloan, Jr., M.D.*

The infrequent reports of mitral insufficiency on a congenital basis reflect the apparent rarity of this condition. Aneurysmal dilatation of the left atrium in a 5-year-old girl with clinical findings suggesting mitral insufficiency has been described by Semans and Taussig in 1938.1 Postmortem examination of this patient revealed thickening of the mitral and tricuspid valves, saccular dilatation of the left atrium, and cardiac hypertrophy. The chordae tendineae also were noted to be greatly shortened. Prior2 has described two adults with congenital malformations of the mitral valve. In one, duplication of the mitral orifice was present while in the other the posterior leaflet lacked the proper chordal attachment. Kjellberg and co-workers3 described the clinical and hemodynamic findings in three patients with congenital mitral incompetence. In two of these patients the left atrium was aneurysmally dilated and the appendage was prominent. In the third patient the malformation was present in association with corrected transposition of the great vessels. One of these patients died, and at postmortem examination the anterior cusp of the mitral valve was found to be lacking and the posterior cusp was rigid. Helmholtz et al.4 have described mitral insufficiency in association with corrected transposition of the great vessels. This has also been discussed by Anderson and associates5 in their review of corrected transposition. Linde and Adams6 reported mitral insufficiency in association with a patent ductus arteriosus in three patients, which they considered related to the presence of endocardial sclerosis involving the valve leaflets. Three patients with congenital mitral incompetence who underwent surgical repair were reported by Starkey.7 In these patients dilatation of the mitral valve ring was the dominant feature. Surgical repair with the open-heart approach was advised as the procedure of choice. Edwards and Burchell8 have described and classified the pathologic anatomy in congenital mitral insufficiency. This included inadequate valve substance as in isolated cleft of the anterior mitral leaflet and anomalous chordal insertion. The latter has been observed in association with the posterior leaflet, corrected transposition of the great vessels, endocardial sclerosis, and duplication of the mitral orifice.

Although rare, this abnormality may produce severe impairment of cardiac function necessitating surgical intervention. Ten patients with congenital mitral insufficiency have been evaluated at the University of Michigan Medical Center. This communication constitutes an analysis of the salient clinical, hemodynamic, and surgical findings in this group.

**Clinical Material and Methods of Study**

Ten patients have been studied. All had the typical clinical findings of mitral insufficiency. In six patients the diagnosis was confirmed at surgery (table 1). The mitral valve was explored and was found to be incompetent in one patient at the time of correction of a coarctation of the aorta. A left-sided catheterization in this patient had shown the characteristic left atrial pressure contour of mitral insufficiency. In two patients the diagnosis was confirmed at postmortem examination: one died while awaiting surgical repair; the other, with mitral insufficiency associated with corrected transposition and a ventricular septal defect, died during cardiotomy. The diagnosis was proved by left ventricular angiography in a last patient who is to have surgical correction.

Right heart catheterizations were carried out in...
eight patients. Left heart catheterizations via the percutaneous method of Bjork and associates were performed in three patients. In two patients a retrograde left heart catheterization as described by Vlad and Lambert was done with injection of contrast media into the left ventricle.

Mitral incompetence was considered to be the major defect in all of the 10 patients. Three patients had an associated coarctation of the aorta, in two patients corrected transposition of the great vessels was demonstrated, and two patients also had evidence of a patent ductus arteriosus. We have not included patients with mitral incompetence in association with a patent ductus arteriosus in whom the latter defect was the primary abnormality.

**Clinical Features**

Nine of the 10 patients had at least one bout of congestive heart failure (table 1). Fatigue on exertion, retardation of growth, and frequent respiratory tract infections were present in all of the patients. In no patient could a history of manifestations of rheumatic fever be elicited. Eight of the 10 patients were examined by physicians during the first year of life and found to have a significant cardiac murmur. Two patients were not ex-
a examined until age 5, when the murmurs were first detected. These patients had no evidence of rheumatic fever, were afebrile, had a normal erythrocyte sedimentation rate, and no elevation of the antistreptolysin titer.

The physical findings were also remarkably uniform. All had some degree of cardiomegaly. A prominent lateral apical out-thrust indicating left ventricular enlargement was usually seen. The typical pansystolic regurgitant murmur of mitral insufficiency was heard in every instance. Seven of the 10 patients had an associated apical thrill. An apical protodiastolic rumble was also heard in every patient. This murmur has been described in patients with mitral insufficiency and probably results from an increased flow across the mitral valve. The typical murmurs are shown in figure 1. The pulmonary component of the second heart sound was thought to be accentuated in three instances, and this finding correlated with the level of the pulmonary artery pressure.

Electrocardiographic Findings

Table 2 summarizes the electrocardiographic findings. Evidence of left ventricular hypertrophy was present in six cases. Right ventricular hypertrophy was encountered in one patient who had corrected transposition of the great vessels and pulmonary hypertension. Another patient with corrected transposition had incomplete right bundle-branch block. Left atrial enlargement as indicated by the presence of a bifid P wave (P mitrale) (fig. 2) was encountered in all of the patients including the two with corrected transposition.

Roentgenologic Examination

The roentgen findings in this group of patients consisted primarily of left atrial and left ventricular enlargement. A double atrial contour, elevation and spreading of the mainstem bronchi, prominence of the left atrial appendage, and a posterior impression on the barium-filled esophagus were found as evidence of left atrial enlargement (fig. 3). In three instances the left atrium was aneurysmal in size. Left ventricular enlargement was suggested by protrusion of the apex beyond the vertebral margins in the left anterior oblique projection. The aorta was characteristically small while the pulmonary vascular markings were either normal or slightly increased. The peripheral pulmonary vessels did not appear narrow in any of the patients, including those with significant pulmonary hypertension. No intracardiac calcifications were noted. In the two patients with corrected transposition, the upper left border of the heart (ascending aorta) presented as a gentle convexity having a more gradual slope than is usually associated with the pulmonary artery of a normal heart. This latter finding has been described by Anderson and associates as a characteristic roentgenologic feature of corrected transposition.

Angiocardiography

All of the patients in the series had biplane angiocardiograms after injection of contrast material into a peripheral vein, pulmonary artery, or left ventricle. With the exception of the left ventricular injections, the studies were not diagnostic of mitral insufficiency, but they served to determine chamber size and the presence of associated defects. The left atrial chamber was usually markedly enlarged and formed the right cardiac border (fig. 4, upper). In addition, the right branch of the pulmonary artery was elevated by the enlarged left atrium (fig. 4, lower). We have also been impressed by the large volume changes encountered in the left atrium as demonstrated in systolic and diastolic exposures (fig. 5). Retrograde left heart
Table 1

Clinical Data

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Diagnosis established</th>
<th>Associated defects</th>
<th>Rheumatic fever</th>
<th>Ease of fatigue</th>
<th>Growth retardation</th>
<th>Congestive failure</th>
<th>Age murmur heard</th>
<th>Frequent respiratory infection</th>
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<td>1. LA</td>
<td>7</td>
<td>F</td>
<td>surgery</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>5 yr.*</td>
<td>+</td>
</tr>
<tr>
<td>2. PP</td>
<td>4</td>
<td>M</td>
<td>surgery</td>
<td>patent ductus</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>3 mo.</td>
<td>+</td>
</tr>
<tr>
<td>3. CP</td>
<td>8</td>
<td>F</td>
<td>surgery</td>
<td>corrected</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>5 yr.*</td>
<td>+</td>
</tr>
<tr>
<td>4. DP</td>
<td>6</td>
<td>M</td>
<td>surgery</td>
<td>transposition</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>2 mo.</td>
<td>+</td>
</tr>
<tr>
<td>5. RJ</td>
<td>3</td>
<td>M</td>
<td>surgery</td>
<td>coarctation</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>1 yr.</td>
<td>+</td>
</tr>
<tr>
<td>6. MB</td>
<td>5</td>
<td>M</td>
<td>surgery</td>
<td>corrected</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>1 mo.</td>
<td>+</td>
</tr>
<tr>
<td>7. SE</td>
<td>9 mo.</td>
<td>M</td>
<td>post mortem</td>
<td>transposition</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8 mo.</td>
<td>+</td>
</tr>
<tr>
<td>8. MP</td>
<td>7</td>
<td>F</td>
<td>post mortem</td>
<td>coarctation</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>2 mo.</td>
<td>+</td>
</tr>
<tr>
<td>9. DW</td>
<td>8</td>
<td>M</td>
<td>surgery</td>
<td>coarctation</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>1 mo.</td>
<td>+</td>
</tr>
<tr>
<td>10. SB</td>
<td>2</td>
<td>F</td>
<td>LV angiogram</td>
<td>patent ductus</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>1.5 mo.</td>
<td>+</td>
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</table>

*First physical examination was pre-school physical

Table 2

Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case</th>
<th>Rate</th>
<th>P-R interval</th>
<th>Axis</th>
<th>Right ventricular hypertrophy</th>
<th>Left ventricular hypertrophy</th>
<th>P mitrale</th>
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<tr>
<td>1. LA</td>
<td>120</td>
<td>0.15</td>
<td>+67</td>
<td>-</td>
<td>+</td>
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<tr>
<td>2. PP</td>
<td>110</td>
<td>0.20</td>
<td>+6</td>
<td>-</td>
<td>+</td>
<td>+</td>
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<tr>
<td>3. CP</td>
<td>88</td>
<td>0.14</td>
<td>+70</td>
<td>+110</td>
<td>+</td>
<td>+</td>
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<td>4. DP</td>
<td>100</td>
<td>0.18</td>
<td>+110</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<tr>
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<td>0.14</td>
<td>+8</td>
<td>-</td>
<td>+</td>
<td>+</td>
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<td>6. MB</td>
<td>87</td>
<td>0.24</td>
<td>+113</td>
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<td>+</td>
<td>+</td>
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<td>7. SE</td>
<td>148</td>
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<td>+65</td>
<td>-</td>
<td>+</td>
<td>-</td>
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<td>8. MP</td>
<td>133</td>
<td>0.15</td>
<td>+81</td>
<td>-</td>
<td>+</td>
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<tr>
<td>9. DW</td>
<td>92</td>
<td>0.14</td>
<td>+100</td>
<td>-</td>
<td>+</td>
<td>+</td>
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<td>10. SB</td>
<td>126</td>
<td>0.14</td>
<td>+84</td>
<td>-</td>
<td>+</td>
<td>+</td>
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</table>

*The contrast material used was the Ditriokon brand of sodium diprotirozate and diatriazoate injection, supplied by Mallinekrodt Chemical Works.

catheterization with injection of contrast material* into the left ventricle demonstrated regurgitation of contrast material into the left atrium in two instances. This is the technic we presently prefer for proving mitral regurgitation (figs. 6 and 7). These studies were carried out with electrocardiographic monitoring so as to correlate the angiocardiographic findings with phases of the cardiac cycle and to rule out the possibility that regurgitation could have occurred because of premature ventricular contraction during atrial systole.

Right Heart Catheterization

The findings obtained at right heart catheterization are shown in table 3. In only one patient was the pulmonary artery pressure within normal limits. One patient had severe pulmonary hypertension, whereas in the others moderate elevation of the pulmonary artery pressure was encountered. The pulmonary capillary wedge pressure was elevated in five of the patients studied in whom this pressure could be obtained. In three instances
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Figure 5
Case 1. Large left atrial volume changes during ventricular diastole (left) and ventricular systole (right).

descent in the first 0.1 second to the mean left atrial pressure. In each of the patients the ratio of the V to A wave was greater than 1.4 and the ratio of the y descent in the first 0.1 second to the mean left atrial pressure equaled or exceeded 0.5, supporting the diagnosis of mitral insufficiency. A typical left atrial pressure pulse contour is seen in figure 8.

Diastasis, the phase of diastole preceding atrial contraction, was definitely present in two of the three patients in whom a left atrial pressure pulse was obtained. This phase is absent in patients with mitral stenosis. In children with rapid cardiac rates, however, diastasis could be masked.

In one patient the left ventricular end-diastolic pressure was elevated as seen in figure 1. The left ventricular end-diastolic pressure is reported as not being elevated in patients with acquired mitral insufficiency. The “washing phenomenon” in which the catheter enters the left ventricle in diastole only to recoil back in the atrium with the regurgitant flow was observed in one patient. The left ventricular pressure pulse showed a rapid fall during late ejection in three patients (fig. 1). This contour has been described by Davila and is attributed to the inability of the ventricle to maintain ejection pressure during the period when regurgitant flow is maximal.

Pathologic Findings

Postmortem examination was performed on two patients. The autopsy on case 8 was ca-
Table 3
Hemodynamic Findings, Right Heart Catheterization

<table>
<thead>
<tr>
<th>Case</th>
<th>Right ventricular pressure S/D</th>
<th>Pulmonary artery pressure S/D</th>
<th>Pulmonary capillary wedge (M)</th>
<th>Wedge pressure pulse</th>
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</thead>
<tbody>
<tr>
<td>1. LA</td>
<td>37/0</td>
<td>37/25</td>
<td>16</td>
<td>nonphasic</td>
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<tr>
<td>2. PP</td>
<td>40/0</td>
<td>42/24</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3. CP</td>
<td>26/2</td>
<td>25/15</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>4. DP</td>
<td>44/0</td>
<td>44/14</td>
<td>18</td>
<td>V wave elevation</td>
</tr>
<tr>
<td>6. MB</td>
<td>62/0</td>
<td>60/25</td>
<td>20</td>
<td>V wave elevation</td>
</tr>
<tr>
<td>7. SE</td>
<td>80/0</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>8. MP</td>
<td>50/12</td>
<td>50/37</td>
<td>132</td>
<td>V wave</td>
</tr>
<tr>
<td>9. DW</td>
<td>38/0</td>
<td>35/25</td>
<td>20</td>
<td>nonphasic</td>
</tr>
</tbody>
</table>

Carried out at a neighboring hospital. Gross findings consisted of enlargement of the left atrium and left ventricle, with a thickened, widely dilated mitral valve annulus. The chordae tendineae were short and appeared to restrict leaflet mobility. Microscopically there was evidence of endocardial sclerosis, primarily in the region of the mitral valve.

The second patient (case 7) died during ventriculotomy for repair of a ventricular septal defect. Examination showed the typical anatomic arrangement in corrected transposition of the great vessels, with the aorta arising anteriorly from the left ventricle and the pulmonary outflow tract arising medially.

Figure 6
Case 10. Upper, posteroanterior view; lower, lateral view. Left ventricular angiogram during early systole (left), late systole (center), and diastole (right) showing marked regurgitation of contrast material into the left atrium. Note variation in left atrial volume during cardiac cycle.
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Figure 7
Case 5. Upper, posteroanterior view; lower, lateral view. Left ventricular angiogram showing regurgitation of contrast material into the left atrium. Left, diastole; right, systole.

and somewhat posteriorly from the right ventricle. The left atrioventricular valve was tricuspid and markedly deformed. The valve leaflets were thickened, the chordae tendineae were shortened, and the annulus was dilated. There were dilatation and hypertrophy of the left atrium and ventricle. Several pinpoint openings were present in the ventricular septum, which was translucent. Microscopic examination of the small pulmonary vessels demonstrated vascular lesions consisting of medial hypertrophy and intimal thickening. This patient had marked elevation of pulmonary artery pressure. Vascular lesions have been reported in acquired mitral insufficiency by Becker et al.,16 and may be attributed to increased pulmonary venous pressure and stasis.

Surgical Results
Six patients have had surgical repair of their defects. Repair of the insufficient mitral valve was carried out through a right posterolateral incision through the fifth intercostal

Table 4

<table>
<thead>
<tr>
<th>Case</th>
<th>Left atrial mean pressure mm. Hg</th>
<th>V/A*</th>
<th>y/MLAP† (0.1)</th>
<th>Diastasis</th>
<th>Left ventricular pressure S/D mm. Hg</th>
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</thead>
<tbody>
<tr>
<td>2. PP</td>
<td>26</td>
<td>2.5</td>
<td>.7</td>
<td>+</td>
<td>—</td>
</tr>
<tr>
<td>6. MB</td>
<td>19</td>
<td>1.5</td>
<td>.5</td>
<td>±</td>
<td>86/0</td>
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<tr>
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<td>25</td>
<td>2.0</td>
<td>.6</td>
<td>+</td>
<td>75/12</td>
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<tr>
<td>5. RJ</td>
<td>Retrograde left heart study</td>
<td></td>
<td></td>
<td></td>
<td>118/20</td>
</tr>
<tr>
<td>10. SB</td>
<td>Retrograde left heart study</td>
<td></td>
<td></td>
<td></td>
<td>115/0</td>
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</table>

*Ratio of the pressure at the peak of the V wave to the pressure at the peak of the A wave.
†Ratio of the y descent in the first 0.1 second to the mean left atrial pressure.
space. The venae cavae and the right superficial femoral artery were cannulated. Extracorporeal circulation was accomplished with an apparatus employing roller pumps and a rotating-disk oxygenator. The mitral valve was exposed through a longitudinal incision in the left atrium, which bulged markedly to the right in each patient. The mitral insufficiency was corrected by placing plication sutures of heavy silk through the mitral annulus at one or both commissures. In one patient a cleft of the anterior leaflet was sutured. The surgical findings and follow-up clinical and roentgenologic examinations are presented for each of the patients.

Case Histories

Case 1

This 5-year-old girl had been in severe congestive heart failure and was only slightly improved on a medical program. At the time of operation...
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the left atrium was found to be considerably enlarged. A regurgitant systolic jet was palpable over the left atrium. The annulus measured 4 to 5 cm. in greatest diameter. The mitral valve leaflets were somewhat thickened and the leaflets in the region of the posterior commissure seemed to lack proper chordal attachment, so that they flapped in the blood stream. The incompetence was materially reduced by plicating the annulus posteriorly with mattress sutures of 3-0 silk. Approximately one third of the length of the mitral valve was closed posteriorly. The patient tolerated the procedure quite well and the postoperative course was uneventful. From a clinical standpoint the girl was markedly improved.

Over 2 years later the patient was fully active, no longer receiving any cardiac medications and had a normal exercise tolerance. Physical findings consisted of minimal cardiomegaly with a soft apical pansystolic murmur. Roentgenograms reveal a marked decrease in cardiac size, less prominence of the left atrium and left ventricle, and diminished spreading of the bronchi (fig. 9).

Case 2

This 2-year-old boy had been observed since the age of 2 months with frequent respiratory tract infections, growth retardation, and two bouts of congestive heart failure. Surgical repair of his mitral valve lesion was undertaken because of increasing cardiac symptoms and progressive cardiomegaly, despite an intensive medical program. At operation the heart was found to be tremendously enlarged and the left atrium aneurysmally dilated. The mitral valve ring was widely dilated and the leaflets were thick and rubbery. The left atrial wall was increased in thickness and showed endocardial sclerosis microscopically. Regurgitation occurred in the region of the posterior portion of the valve, the leaflets being restricted by anomalous insertion of short, thickened chordae tendineae. Horizontal mattress sutures posteriorly across the annulus decreased the diameter of the mitral valve ring considerably. A small amount of insufficiency remained but additional suturing was thought unwise because of the risk of creating stenosis. The postoperative course was uneventful and during the subsequent 2-year period the boy has resumed a normal growth pattern, is no longer receiving cardiac medications, and has a normal exercise tolerance. Roentgenograms reveal a marked decrease in the over-all cardiac size, particularly of the left atrium and left ventricle (fig. 10).

Case 3

This 6-year-old girl was the least incapacitated of the group although there was ease of fatigue and growth retardation. At surgery the left atrium was enlarged and a systolic thrill was felt over it. The mitral valve appeared thickened and white, and a cleft of the anterior leaflet was noted and was closed with figure-of-eight sutures. The postoperative course was complicated by a prolonged febrile course, which was thought to be the postcardiotomy syndrome. A marked decrease in cardiac size has not been noted, and a residual murmur of mitral insufficiency has persisted in the subsequent year. The child has had an increase in exercise tolerance, has gained 6 pounds in weight, and appears to be progressing quite satisfactorily.

Case 4

This patient had correction of his defect at the age of 6 years. At surgery the left atrium was markedly dilated and a systolic thrill was felt over it. The aorta lay far to the left in the typical position of corrected transposition. The "mitral" valve was noted to be markedly incompetent. The annulus was widely dilated and the valve appeared to have three cusps. The valve leaflets were thickened and were bound down by shortened chordae tendineae. The "mitral" annulus was narrowed anteriorly and posteriorly by a series of horizontal mattress sutures. The amount of insufficiency was reduced dramatically.

The postoperative course was entirely satisfactory. A soft apical systolic and a diastolic murmur have persisted, despite marked clinical improvement. The boy is now fully active, attends school, and is not taking cardiac medications. Roentgenograms reveal a marked decrease in cardiac size.
Case 5

After repair of a coarctation of the aorta at age of 2 years in this boy, findings of mitral insufficiency persisted, symptoms continued and one bout of congestive heart failure occurred. The defect was repaired at age of 4 years. At surgery a tremendously enlarged left atrium was found, which pushed the right atrium into the superior vena cava anteriorly. A systolic thrill was felt in it. The mitral annulus was moderately dilated and the anterior leaflet did not approximate the posterior leaflet but appeared to overshoot it. The thickened anterior leaflet seemed to be attached by a few long chordae tendineae that allowed eversion of the leaflets during ventricular systole. The mitral valve annulus was reduced by horizontal mattress sutures through the anterior and posterior commissures. The valve then appeared to be completely competent. The procedure was tolerated quite well, and there were no postoperative complications. During his 6 months of follow-up there has been a marked increase in exercise tolerance, a 4-pound weight gain, and he has been taken off cardiac medications. Roentgenograms show a decrease in cardiac size, particularly of the left atrium and left ventricle.

Case 6

This boy was admitted at the age of 2½ years in severe congestive heart failure. At surgery a large left atrium was seen to bulge forward from a posterior position, and a systolic thrill was felt over it. The mitral valve annulus was widely dilated, the valve leaflets were thickened and white, and the chordae tendineae were shortened. The anterior leaflet seemed to lack proper chordal attachment at the midpoint. During left ventricular contraction the leaflets did not approximate. Horizontal mattress sutures across the mitral annulus anteriorly and posteriorly reduced the size of the annulus. The valve leaflets seemed to approximate much better and the valve opening was reduced to 1½ fingers. Postoperatively the patient did well although there was a residual, less intense systolic murmur at the apex. Since discharge he has gained 3 pounds and increased his activity but is still taking digitoxin. Roentgenograms reveal a considerable decrease in the over-all cardiac size, with diminution in the size of the left atrium and left ventricle.

Comment

All the patients who have had surgical repair of their mitral valve lesions were severely incapacitated preoperatively. Following the surgical procedure there has been a dramatic relief of symptoms in all patients and, with one exception, a remarkable decrease in cardiac size. The murmur of mitral incompetence has persisted but has diminished in intensity.

The types of malformations encountered in our series are shown in figure 11. Anomalous chordal insertion with regurgitation in the region of the posterior commissure appears to be the commonest. In one instance a cleft in the anterior leaflet was sutured. Recently Edwards has called attention to the presence of accessory chordae tendineae in association with clefts of the anterior leaflets with partial and complete atrial ventricular canal. The question can be raised in the patient with the cleft of the anterior leaflet whether persistence of mitral insufficiency is related to the presence of accessory chordae that may continue to restrict leaflet mobility.

Summary

The clinical and hemodynamic findings in 10 patients with mitral insufficiency on a congenital basis are described.

Six of these patients have had repair of the defects with dramatic clinical improvement noted. The findings at the time of surgical repair have been summarized.

The severity of the symptoms and the benefits to be derived from surgical repair should encourage vigorous attempts to establish the correct diagnosis. This is best accomplished by either recording the typical left atrial pressure pulse of mitral insufficiency during left heart catheterization or by left ventricular angiography to demonstrate the regurgitation of contrast material into the left atrium.

References

4. Helmholtz, H. F., Daugherty, G. W., and Edwards, J. E.: Congenital "mitral" insufficiency in association with corrected transposi-
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Sydenham was called "a man of many doubts" and therein lay the secret of his great strength.—SIR WILLIAM OSLER. Aphorisms from His Bedside Teachings and Writings. Edited by William Bennett Bean, M.D. New York, Henry Schuman, Inc., 1960, p. 112.
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