Acute Severe Mitral Regurgitation Secondary to Ruptured Chordae Tendineae

Clinical, Hemodynamic, and Pathologic Considerations

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THE MAJORITY of patients with severe mitral regurgitation have enlarged left atria, atrial fibrillation, a history of acute rheumatic fever, and a gradual onset of symptoms of cardiac decompensation that may persist for many years. This report describes 10 patients with severe mitral regurgitation caused by rupture of one or more chordae tendineae, and contrasts the clinical, radiographic, hemodynamic, and pathologic findings in these patients with those commonly noted in patients with the more usual form of mitral regurgitation. Thus, the majority of the patients with ruptured chordae tendineae had little left atrial enlargement, were in sinus rhythm, and had not had a history of acute rheumatic fever. The symptoms of cardiac decompensation generally appeared abruptly, and were usually present for only several months before they led to the patient’s death or necessitated operative treatment. An appreciation of this relatively pure, acute form of mitral regurgitation is important in the understanding of the wide range of physiologic, pathologic, and clinical findings that may accompany this valvular lesion.

Description of Patients

A summary of the clinical, electrocardiographic, hemodynamic and pathologic findings in the individual patients is presented in table 1.

History. The 10 patients ranged in age from 17 to 54 years. Six were women. Only one patient (L. L.) had a history suggestive of acute rheumatic fever, and none had a history of chorea or chest trauma. Six patients had a definite history of bacterial endocarditis, and each had been asymptomatic and fully active prior to the onset of the infection. Pregnancy appeared to have played a role in the appearance of the bacterial endocarditis in three patients; A. L. and A. A. developed the infection shortly after parturition, while patient R. H. developed pyogenic endometritis after an abortion which in turn was followed by bacterial endocarditis. In two patients each alpha streptococcus and staphylococcus aureus were the organisms responsible for the endocarditis, and in one patient each the causative organisms were streptococcus faecalis and staphylococcus albus. The onset of cardiac decompensation was abrupt in nine patients and was progressive thereafter, while in patient V. C., who did not have a history of bacterial endocarditis, symptoms developed more slowly. In seven patients the period of time from the onset of symptoms of cardiac failure to autopsy or operation was less than 16 months, while in the other three patients the interval ranged from 5 to 8 years. Seven patients had no knowledge of heart murmurs before the onset of bacterial endocarditis or cardiac failure. Three patients, two of whom had a history of bacterial endocarditis, were known to have had precordial murmurs for periods ranging from 12 to 43 years before operation or death. In one of these patients, the murmur may have resulted from the presence of associated partial anomalous pulmonary venous connection and a small atrial septal defect.

Each of the patients complained of easy fatigability, exertional and nocturnal dyspnea, and all but one had pedal edema. All were treated with digitalis and salt restriction, but despite these measures they were severely disabled and considered to be in functional classes III or IV.

Physical Examination. Each of the 10 patients had prominent right ventricular and less prominent left ventricular lifts as well as easily palpable apical systolic thrills. The sound of pulmonic valve closure was accentuated in all patients, and a third heart sound was almost always audible. In every patient, a blowing pansystolic murmur of at least grade IV/VI intensity was heard over the entire precordium, loudest over the cardiac apex, with radiation to the left axilla. In two patients the murmur at the cardiac apex was of whistling or...
cooing quality, and in two others the systolic murmur was also audible over the entire vertebral column. In five patients the systolic murmur along the lower left sternal border became louder during inspiration. Seven patients had short, low-pitched, mid-diastolic flow murmurs at the apex, and two patients had high-pitched, blowing, diastolic murmurs along the left sternal border. The liver was palpable and tender in the majority of the patients, and pulsed during systole in three of them.

Electrocardiograms (Fig. 1). Eight patients had sinus rhythm, and two had atrial fibrillation; in one of the latter this arrhythmia appeared shortly before death. Right axis deviation was present in five patients, and left axis deviation in one. Four patients had first-degree atrioventricular block. Right ventricular hypertrophy was present in seven patients and left ventricular hypertrophy as well in three. Seven patients exhibited the peaked P waves characteristic of right atrial enlargement, and one had left atrial enlargement.

Roentgenograms (Fig. 2). Some degree of cardiac enlargement was evident on the chest roentgenograms of all 10 patients. The left atrium was mildly or moderately enlarged in eight patients, and markedly enlarged in only the two patients who had been symptomatic for the longest times, 5 and 8 years. Both ventricles were considered to be enlarged in all 10 patients. Calcification in the region of the mitral valve was detected in only one patient (L. L.). Angiographic studies with left ventricular injection in each of two patients (A. F. and L. S.) showed near-normal sized left atria which expanded strikingly during ventricular systole, but there was no evidence of prolapse of a mitral valve leaflet into the left atrium.

Hemodynamic Findings (Fig. 3). All nine patients who underwent right heart catheterization had markedly elevated pulmonary arterial and right ventricular systolic pressures, ranging from 61 to 110 mm. Hg. The mean left atrial pressures were elevated to between 15 and 32 mm. Hg in eight patients, and within normal limits (11 mm. Hg) in the ninth. The v-wave peaks were abnormally elevated in all nine patients and ranged from 23 to 66 mm. Hg (average, 45 mm. Hg). The left ventricular end-diastolic pressure was normal (≤12 mm. Hg) in six of the eight patients in whom it was measured, and was markedly elevated (20 and 27 mm. Hg) in two. The

Figure 1

Electrocardiogram of patient A.L. Right axis deviation, right atrial enlargement, right ventricular hypertrophy, and possible left ventricular hypertrophy are present, although mitral regurgitation had been present for only 16 months.
Table 1
Clinical, Electrocardiographic, Hemodynamic, and Pathologic Data from the 10 Patients with Ruptured Mitral Chordae Tendineae

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age at death or at operation (yr)</th>
<th>Length of symptoms (mo)</th>
<th>History of bacterial endocarditis (organism)</th>
<th>Electrocardiogram</th>
<th>Hemodynamic data</th>
<th>Pulmonary vascular resistance (dynes sec. cm.⁻²)</th>
<th>LA size at operation (O)</th>
<th>LA size at autopsy (A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. V.C.</td>
<td>41 yr.</td>
<td>5 yr.</td>
<td>AF, RAD, RAE, RVH, LVH</td>
<td>110/40 (67)</td>
<td>32–39*</td>
<td>1.4</td>
<td>1217</td>
<td>Dilated (A)</td>
</tr>
<tr>
<td>2. A.A.</td>
<td>37 yr.</td>
<td>7 mo.</td>
<td>NSR, RAE, RVH</td>
<td>24–52</td>
<td>92/27</td>
<td>“Normal”</td>
<td>“Normal”</td>
<td>(A)</td>
</tr>
<tr>
<td>3. A.L.</td>
<td>19 yr.</td>
<td>16 mo.</td>
<td>Streptococcus Group D</td>
<td>70/30</td>
<td>15–36</td>
<td>100/10</td>
<td>1.7</td>
<td>950</td>
</tr>
<tr>
<td>4. R.H.</td>
<td>24 yr.</td>
<td>5 mo.</td>
<td>Enterococcus</td>
<td>72/32</td>
<td>27–66</td>
<td>108/11</td>
<td>1.5</td>
<td>1000</td>
</tr>
<tr>
<td>5. P.H.</td>
<td>29 yr.</td>
<td>5 yr.</td>
<td>NSR, RAE, First-degree HB</td>
<td>105/40</td>
<td>15–35</td>
<td>1.6</td>
<td>2000</td>
<td>“Normal”</td>
</tr>
<tr>
<td>6. B.F.</td>
<td>21 yr.</td>
<td>13 mo.</td>
<td>Staphylococcus aureus</td>
<td>65/28†</td>
<td>11–23†</td>
<td>100/9†</td>
<td>1.4†</td>
<td>1100†</td>
</tr>
<tr>
<td>7. J.W.</td>
<td>54 yr.</td>
<td>8 yr.</td>
<td>Alpha streptococcus</td>
<td>32/11</td>
<td>6–11†</td>
<td>3.0†</td>
<td>179†</td>
<td>(O)</td>
</tr>
<tr>
<td>8. L.L.</td>
<td>53 yr.</td>
<td>9 mo.</td>
<td>AF, LAD</td>
<td>44/20 (28)†</td>
<td>7–10†</td>
<td>95/5†</td>
<td>2.8†</td>
<td>382†</td>
</tr>
<tr>
<td>9. A.F.</td>
<td>48 yr.</td>
<td>7 mo.</td>
<td>NSR, Nonspecific T-wave changes</td>
<td>84/30</td>
<td>25–65</td>
<td>100/20</td>
<td>1.8</td>
<td>609</td>
</tr>
<tr>
<td>10. L.S.</td>
<td>17 yr.</td>
<td>5 mo.</td>
<td>Staphylococcus aureus</td>
<td>71/41</td>
<td>24*</td>
<td>86/12</td>
<td>“Normal”</td>
<td>(A)</td>
</tr>
</tbody>
</table>

*Pulmonary arterial wedge pressures.
†Before operation.
‡Several months after operation.

Abbreviations: PA, pulmonary arterial pressure; LA, left atrial pressure; LV, left ventricular pressure; s/d (mean), systolic/diastolic (mean) pressures; AF, atrial fibrillation; RBBB, right bundle-branch block; NSR, normal sinus rhythm; RVH, right ventricular hypertrophy; RAE, right atrial enlargement; LVH, left ventricular hypertrophy; LAE, left atrial enlargement; LAD, left axis deviation; HB, heart block. “Normal,” near normal size or only moderately dilated.
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Figure 2
Roentgenograms obtained preoperatively (upper) and 7 months postoperatively (lower) in patient B.F. This patient had had severe mitral regurgitation for 13 months before operation, but there was only minimal left atrial enlargement.

Cardiac output was markedly reduced in each of the seven patients in whom it was measured, and ranged from 0.9 to 1.8 L/min./M², with an average of 1.5 L/min./M². Pulmonary vascular resistance was calculated in seven patients and ranged from 609 to 2,000 with an average of 1135 dynes sec. cm⁻⁵. Indicator-dilution curves recorded from a systemic artery following injection into the left ventricle or left atrium in seven patients showed the prolonged descending limb characteristic of severe mitral regurgitation.

Pathologic Findings (Figs. 4-11). The mitral valves and chordae tendineae were examined in six patients at necropsy. In the other four patients the mitral valve was examined after it had been surgically excised with the chordae tendineae and papillary muscles intact. One patient died of heart failure, one from a complication of cardiac catheterization, three following mitral annuloplas-ty, and one following Starr-Edwards mitral valve replacement. Ruptured chordae tendineae were responsible for mitral regurgitation in every patient, and in six of them the rupture was clearly the result of
bacterial endocarditis. Active endocarditis was present in two patients (R. H. and L. S.); it involved the posterior half of the aortic leaflet of the mitral valve in one (fig. 6); and both anterior and posterior leaflets in the other patient. All chordae that had inserted into these portions of the valve had been destroyed. Organisms, however, were not found in sections of the valves of either of these patients. In six patients most or all of the chordae to one half of the anterior leaflet of the mitral valve were ruptured, in one patient all of the chordae that had been attached to the posterior leaflet were involved, and in three patients several chordae from both leaflets were destroyed. With the exception of the two patients with active endocarditis, there were no essential differences between the pathologic findings in the patients with and those without a history of bacterial endocarditis. Although some fibrous thickening of the mitral leaflet adjacent to the ruptured chordae was usually evident, the remaining mitral valve leaflets, commissures, and remaining chordae showed no apparent abnormalities. The aortic valve was normal in the six autopsied patients. In the other four patients, no abnormalities of the aortic valve were detected at the time of operation. The tricuspid and pulmonic valves also were normal in the hearts examined at necropsy.

The left atrial chamber was near normal size, or only moderately enlarged, in the six autopsied patients, and in each of them the left atrial wall was considerably thickened. In two of the patients, who had had symptoms for 5 and 8 years, the left atrium was severely dilated. Jet lesions were present on the endocardial surface of the left atrium in all 10 patients, their location depending upon the position of the ruptured chordae. The right atrium was not dilated, but the left ventricle was considerably dilated. The anterior mitral leaflet (A.M.L.) is now seen from the ventricular aspect. The aortic valve (A.V.) is normal.
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ventricle was dilated and hypertrophied in the six patients studied at necropsy.

On microscopic examination, there were no Aschoff bodies in sections of the heart from any of the six autopsied patients, and sections of the left atrial walls disclosed severe hypertrophy of the myocardial fibers and marked thickening of the endocardium (fig. 8). Histologic examination of sections from the lungs of the six autopsied patients disclosed grade 3/6 hypertensive pulmonary vascular changes in each (classification of Heath and Edwards2). Three patients had ossific nodules scattered in the lungs. Examples of microscopic changes in the lungs of two patients are shown in figures 9 and 10. The pulmonary vascular changes in patients R. H. and A. A. were particularly noteworthy, since these patients had had clinical evidence of mitral regurgitation for only 5 and 7 months, respectively, before death. There was severe centrolobular hepatic congestion in each of the six autopsied patients, an example of which is shown in figure 11.

Discussion

In the past, the conditions considered to be responsible for ruptured chordae tendineae of the mitral valve included rheumatic fever,5–7 trauma,8 so-called spontaneous rupture,7 and bacterial endocarditis involving either diseased or normal valves.8–10 Most authors have considered that chordal rupture occurs predominantly in previously diseased valves. In the National Institutes of Health collection of almost 150 hearts with acquired mitral valvular disease, there are only two with ruptured mitral chordae, and in both patients this was clearly of secondary importance in the resultant regurgitation. On the other hand, we have encountered the 10 patients, described in this report, who had ruptured mitral chordae involving a valve that otherwise appeared not to be grossly diseased. Critical review of earlier reports, including published photographs of ruptured mitral chordae, indicated numerous instances of ruptured mitral chordae in valves that also did not appear grossly abnormal,4, 5, 7, 10–17 although some of the authors had considered that rheumatic heart disease was the underlying process. Although bacterial endocarditis tends to affect obviously malformed or previously diseased valves most often, and

Figure 5

Photographs of opened left atrium (L.A.), mitral valve, and left ventricle (L.V.) of patient 3 (A.L.) left, and patient 5 (P.H.) right. Chordae tendineae that had attached to the anterior (A.M.L.) and posterior (P.L.) mitral leaflets adjacent to the anterolateral commissure are absent in the heart of patient 3 (left). The arrow points to the site of jet lesion in the left atrium in this patient. All chordae tendineae from the posterior portion of the anterior mitral leaflet (A.M.L.) are severed in patient 5 (right). The nonruptured chordae in each patient are normal, and in neither is the left atrium dilated.
chronic rheumatic involvement might thus predispose to chordal rupture, it seems entirely possible that the marked thickening and shortening of the chordae tendineae of a rheumatic valve may actually serve to protect these structures from rupture. On the basis of the experience with the patients described in this report, as well as the many published illustrations of ruptured chordae in valves that do not appear grossly abnormal, it is suggested that bacterial endocarditis occurring in hearts without detectable anatomic valvular abnormalities is an important cause of chordal rupture.

Immediately following rupture of a single chorda in the dog, the left atrial pressure, particularly the v-wave peak, rises abruptly, a systolic thrill and murmur become detectable in this chamber, and the left atrial appendage begins to balloon during each ventricular systole. The animals rapidly develop congestive cardiac failure, and their course is usually one of rapid progression. From the hemodynamic point of view, the

Figure 6
Photographs of heart of patient 4 (R.H.). Upper left. The mitral valve is exposed by opening the left ventricle (L.V.), aortic valve (A.V.), and aorta. All chordae tendineae that had attached to the posterior portion of the anterior mitral leaflet (A.M.L.) are severed. The endocarditis that ruptured the chordae is active. The aortic valve is normal. L.A.A., left atrial appendage. Lower left. The size of the incompetent mitral valve orifice (M.V.O.) is apparent from this view. A.V., aortic valve cusps. Upper right. The left atrium (L.A.), mitral valve, and left ventricle (L.V.) are opened. The nonruptured mitral chordae are normal. A.M.L., anterior mitral leaflet. Lower right. Close-up of the ruptured mitral chordae of the anterior leaflet.
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Figure 7
Photograph of heart of patient 1 (V.C.). The left atrium (L.A.), mitral valve, and left ventricle (L.V.) are opened. Both chambers are considerably dilated as is the mitral value annulus. This patient had known mitral regurgitation for at least 5 years. At least three chordae tendineae (arrows) from the anterior mitral leaflet (A.M.L.) are severed and each of these is thickened as is a portion of this leaflet. The non-ruptured chordae are elongated, but otherwise normal. The posterior leaflet is normal.

Figure 8
Photomicrographs of sections of posterior wall of left atrium in four patients each taken from a site approximately 3 cm. above the mitral annulus. Left. Left atrial wall in 30-year-old woman dying of acute leukemia (for control). The endocardium is above, the epicardium, below. Mid-left. Left atrial wall in patient 4 (R.H.) showing marked thickening. This patient died 5 months after the onset of cardiac decompensation. Mid-right. Left atrial wall in patient 5 (P.H.) who lived for 5 years after the onset of cardiac decompensation. The endocardium is thicker, but the myocardium is thinner than in patient R.H. Right. Left atrial wall of a patient with mitral regurgitation, a giant left atrium, and normal left atrial pressures. The myocardial fibers are extremely elongated and thinned. The smooth muscle immediately beneath the endocardium is hypertrophied. Hematoxylin and eosin stains, original magnification in each x 20.

regurgitation produced suddenly in experimental animals more closely resembles the mitral regurgitation that developed acutely in the patients described herein than the regurgitation observed in patients with chronic rheumatic mitral regurgitation. In the latter condition, the regurgitation usually develops gradually, and sufficient time is available for the usual adjustments to this lesion to take place. In addition, the myocardium of the left atrium and of the left ventricle may also be affected by the rheumatic process. In the present group of patients with ruptured mitral chordae tendineae, however, the myocardium and pulmonary vascular bed were presumably normal at the time the chordae ruptured, and the resultant clinical, hemodynamic, and anatomic abnormalities may be attributed entirely or almost entirely to the mechanical defect.

The unusual hemodynamic and clinical features of the majority of patients described herein may be emphasized by comparing them with other groups of patients having severe mitral regurgitation. Patients with se-
vere mitral regurgitation were previously separated into three major groups on the basis of obvious differences in atrial compliance.20

I. A group with the sudden development of mitral regurgitation in whom there was mild to moderate enlargement of the left atrium, striking elevation of the mean left atrial pressure, of the left atrial v wave, the pulmonary vascular resistance, and in whom severe right heart failure was often present.

II. A group who had normal left atrial and pulmonary arterial pressures, normal pulmonary vascular resistance, a low cardiac output, huge left atria, atrial fibrillation, a history of acute rheumatic fever, and a long-standing history of heart failure (the syndrome of severe mitral regurgitation with normal left atrial pressure).20

III. The largest number of patients with severe mitral regurgitation lies between these two extremes, and they exhibit both gross left atrial enlargement and an

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**Figure 9**

Photomicrographs of sections of pulmonary blood vessels in patient 4 (R.H.) who lived for only 5 months after the onset of mitral regurgitation. Upper left. Pulmonary vein showing severe intimal proliferation and luminal narrowing. Upper right. Small muscular pulmonary artery with arteriolar branch showing severe intimal proliferation in each and luminal obliteration in the arteriole. Lower. Larger muscular pulmonary arteries showing medial hypertrophy and mild intimal proliferation. An adjacent small pulmonary artery is markedly narrowed by intimal fibrosis. Elastic tissue stains; original magnification $\times 340$ (two upper), $\times 125$ (lower).

**Figure 10**

Photomicrographs of sections of lung in patient 2 (A.A.), who survived 7 months after the onset of mitral regurgitation. Upper. Pulmonary vein showing severe intimal fibrosis. Lower. Ossific nodule. These "lung bones" were seen in sections of lungs in three of the six autopsied patients. Elastic tissue stain (upper), hematoxylin and eosin stain (lower); original magnification $\times 210$ (upper), $\times 135$ (lower).
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Figure 11

Photomicrograph of section of liver in patient 4 (R.H.), showing massive centrilobular necrosis (dark areas). The few remaining hepatic cells are located adjacent to the portal areas. Similar degrees of hepatic congestion and necrosis were seen in each of the six autopsied patients in this series. Phosphotungstic-acid hematoxylin stain, original magnification X 32.

elevated left atrial pressure. Thus, the patients with the syndrome of severe mitral regurgitation with normal left atrial pressure represent one end of the spectrum of mitral regurgitation, and the seven patients described in this report whose cardiac failure progressed rapidly represent the opposite end.

The severe pulmonary vascular changes observed in these patients with ruptured chordae tendineae are interpreted as anatomic expressions of the markedly elevated left atrial and pulmonary arterial pressures. The advanced pulmonary vascular changes, in association with little or no left atrial enlargement, contrasts sharply with the absence of these changes in patients with huge left atria secondary to long-standing mitral regurgitation.20 As a consequence of the abnormalities in the pulmonary vascular bed, right ventricular hypertrophy was always present in the patients with acute mitral regurgitation and relatively small left atria, whereas right ventricular hypertrophy is rare in the patients with extremely dilated left atria.21

It is likely that the rapidity with which regurgitation develops is the primary determinant of hemodynamic, pathologic, and clinical changes observed in patients with severe mitral insufficiency. It is apparent that the walls of the left atria, and of the pulmonary vessels, were far less compliant in the nine patients with ruptured chordae tendineae who developed heart failure rapidly than in the group of patients with severely dilated left atria and normal left atrial pressures described previously.20 Long-standing, gradually developing mitral regurgitation would appear, in some instances, to alter the elastic properties of the left atrial wall and, thereby, displace the atrial pressure-volume curve to make this chamber more compliant, so that a normal pressure may exist in a greatly enlarged atrium. Mitral regurgitation was present for only 5 to 16 months in seven of the eight patients in this series with normal or only moderately enlarged left atria. In contrast, symptoms of cardiac decompensation had been present for 5 to 8 years in the two patients with ruptured chordae tendineae who had very large left atria. Additional support for the view that the atrial compliance may increase as a late development in the course of mitral regurgitation was gained from observations in a 9-year-old patient with congenital mitral regurgitation in whom serial catheterization studies were carried out.22 He was in sinus rhythm and exhibited slight disability at the time his left atrial pressure was grossly elevated (mean, 25; v peak, 45 mm. Hg). Subsequently, the patient developed cardiac failure, atrial fibrillation, and roentgenographic evidence of further increase in size of the left atrium. These changes were accompanied by a decline of left atrial pressure (mean, 15; v peak, 24 mm. Hg). Thus, in this patient a decrease in the mean left atrial pressure and the atrial v-wave peak pressure occurred in the face of an increase in the left atrial size, further substantiating the notion that the elastic properties of the left atrial wall had been altered.

In other reports of patients with ruptured mitral chordae tendineae, the left atrial chamber is usually described as being quite dilated. This finding was present in the six patients with ruptured mitral chordae reported by Menges et al.,12 and the five described in
detail by Osmundson et al. These patients had been symptomatic for a number of years and, in general, the size of the left atrium correlated with the duration of the illness. In contrast, the heart of the first reported patient with ruptured mitral chordae, described by Corvisart in 1812, was not grossly enlarged, but this patient survived for only 10 days after the onset of cardiac symptoms. Similarly, in the patient with ruptured mitral chordae reported by Frothingham and Hass, who had been symptomatic for only 8 months, the left atrium was apparently not enlarged. In the patients described in these reports and others, a good correlation existed between left atrial size and the presence or absence of atrial fibrillation. The patients with atrial fibrillation were generally those with symptoms of cardiac disease for several years and very large left atria. The presence of atrial fibrillation in all of the patients with markedly dilated left atrial chambers, but normal atrial pressures, and the absence of this arrhythmia in the patients with ruptured chordae tendineae, as well as in the child with congenital mitral regurgitation discussed above, suggest the possibility that the process that increases atrial compliance may be related to the development of atrial fibrillation.

Operative treatment was undertaken in seven of the 10 patients described herein. Mitral annuloplasty, with or without insertion of artificial chordae, which was originally utilized in this clinic for repair of ruptured chordae, was attempted in two patients, but each of them died in the immediate postoperative period. Mitral valve replacement with a Starr-Edwards prosthesis was carried out in the other five patients, and four have shown considerable improvement. The fifth died 2 weeks after operation of severe pulmonary infection including pulmonary abscess and empyema. Total valve replacement appears to be the most effective operative method for correcting ruptured chordae, although others have reported good results with mitral plication and insertion of artificial chordae tendineae.

Since, with any given magnitude of regurgitant flow, the atrial pressure varies inversely with the compliance of the atrium, the extent to which the atrial pressure can be expected to decline following a corrective operation will also vary inversely with atrial compliance. Accordingly, the clinical and hemodynamic benefits of operation may be expected to be greatest in patients with relatively small left atria and high left atrial pressures. This may be illustrated by two patients in this series who have had postoperative catheterization following mitral valve replacement. B. F., a 21-year-old woman, was completely bedridden immediately before operation, although she had been well until 13 months earlier, when bacterial endocarditis was soon followed by the development of mitral regurgitation. At the time of the operation she weighed only 33 Kg. Preoperatively, the left atrial (mean, 11; v peak, 23 mm. Hg) and pulmonary arterial (65/28, mean 38 mm. Hg) pressures were elevated, roentgenographically the left atrium was only mildly enlarged, and on a standard oral sodium tolerance test she was unable to excrete any sodium. Following replacement of the mitral valve with a Starr-Edwards prosthesis, she has been entirely asymptomatic. Seven months after operation she weighed 50 Kg. and the pulmonary arterial wedge (mean, 6; v peak, 11 mm. Hg) and pulmonary arterial (32/11, mean 16 mm. Hg) pressures were normal, the heart was smaller roentgenographically (fig. 2), and her sodium tolerance had become normal. J. W., a 54-year-old man, also was incapacitated before operation. He was emaciated, in severe cardiac failure, and the mean left atrial pressure was 15 with v waves of 32 mm. Hg, and the pulmonary arterial pressure was 70/27 mm. Hg. Twelve months after replacement of his mitral valve, the left atrial mean pressure had fallen to 7, the v waves to 10 mm. Hg, and the pulmonary arterial pressure to 44/20 mm. Hg. This patient has improved symptomatically, but he continues to accumulate fluid despite diuretic therapy, presumably consequent to persistent "relative" tricuspid regurgitation, and his heart had remained enlarged. Although this man also developed severe
mitral regurgitation following rupture of chordae tendineae as a consequence of bacterial endocarditis, 8 years had elapsed from the onset of the valvular regurgitation until the valve was replaced, and the left atrium dilated during this interval. It is suspected that the left atrial pressures were greatly elevated immediately following the mitral chordal rupture, but as the left atrial chamber dilated the left atrial pressures decreased to levels approaching normal.

Summary and Conclusions
This report describes the clinical, radiographic, hemodynamic, and pathologic findings in 10 patients, in each of whom ruptured mitral chordae tendineae were responsible for severe mitral regurgitation. In six patients, the chordal rupture was the result of proved bacterial endocarditis, and in the other four, although the cause was not determined, it is considered that bacterial endocarditis may have been the etiologic factor in them as well. Only one of the patients had a history suggestive of acute rheumatic fever. From examination of the mitral valves at autopsy, or after operative excision, no gross abnormalities of the valves were apparent, other than the ruptured chordae and mild thickening of the adjacent leaflet. The signs and symptoms of mitral regurgitation appeared abruptly in all but one patient and were unremitting thereafter. Eight patients had sinus rhythm and two had atrial fibrillation. Each of the latter had had symptoms of cardiac decompensation for several years, and their left atria were markedly enlarged. The left atrial chamber in the other eight patients was of near normal size or only moderately enlarged. The interval from the onset of symptoms of mitral regurgitation to operation in the two patients with dilated left atria averaged 6½ years, whereas the interval in the eight patients with near normal or only moderately dilated left atria averaged 15 months. In all patients the pulmonary arterial and the left atrial pressures, particularly the v-wave peak, were elevated. The cardiac index was low, and the calculated pulmonary vascular resistance was elevated in the seven patients in whom it was measured. Mitral annuloplasty was performed in two patients, with insertion of artificial chordae, but both died in the early postoperative period. Replacement of the mitral valve with a Starr-Edward prosthesis was performed in five patients, with considerable improvement in four. The fifth died of a pulmonary complication 2 weeks after operation.

The distinctive clinical and hemodynamic findings in the majority of these patients, i.e., the rapid progression of heart failure, sinus rhythm, normal-sized or only moderately enlarged left atria, and the greatly elevated pressures in the pulmonary artery and left atrium are attributed to the sudden onset of the mitral regurgitation. These findings are contrasted to those in patients with chronic rheumatic mitral regurgitation, whose disability progresses slowly and who exhibit atrial fibrillation, marked left atrial enlargement, and sometimes normal pulmonary arterial and left atrial pressures. It is proposed that the majority of patients with ruptured chordae cannot sustain the acutely imposed hemodynamic burden for periods of time sufficient to allow the increase in left atrial compliance so frequently observed in patients with chronic rheumatic mitral regurgitation.

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