Nonrheumatic Subvalvular Mitral Regurgitation

Etiology and Clinical Aspects


SUMMARY

Thirty-three patients are reported with nonrheumatic mitral regurgitation due to rupture or stretching of the chordae tendineae or papillary muscles. Nineteen (58%) had no definite predisposing etiological factors (average age 59 years). Six (19%) had a previous myocardial infarction. It is proposed that rupture or stretching of the chordae resulted from degenerative or ischemic changes in the chordae and/or papillary muscles. Six young patients had ruptured chordae from other causes. Two patients had myocardial infarction resulting in rupture of the posteromedial papillary muscle.

Characteristic clinical findings included progressive dyspnea of sudden onset, sinus rhythm, systolic thrill, accentuated pulmonary valve closure sound, third heart sound, and widely propagated pansystolic murmur. The electrocardiogram frequently showed inferolateral ischemic changes. Plain chest radiographs usually showed a normal-sized heart and left atrium in patients in sinus rhythm. At cardiac catheterization a characteristic high V wave in the pulmonary artery wedge pressure was seen in 88% of patients. The left ventricular end-diastolic pressure was highest in the six postinfarct patients in whom the ejection fraction was significantly low.

Additional Indexing Words:
Ruptured chordae  Papillary muscle rupture  Myocardial ischemia
Plain chest radiographs  Ejection fraction

CHRONIC rheumatic heart disease causing destruction, fusion, shortening, and often calcification of the valve leaflets and chordae is the most common cause of organic mitral regurgitation. In such patients atrial fibrillation is usually present, and some degree of mitral stenosis or aortic valve disease is also common.

With the routine use of open-heart surgery, an increasing number of patients have been reported with nonrheumatic disorganization of the subvalvular mechanism resulting in pure regurgitation. Rupture or stretching of the chordae tendineae, and rupture or dysfunction of a papillary muscle have been described. Each of these lesions may produce a characteristic clinical syndrome.

This paper presents the clinical and hemodynamic findings in 33 patients with severe subvalvular mitral regurgitation, none of whom had any evidence of antecedent rheumatic carditis. Subvalvular mitral regurgitation is defined as the result of deformity, disruption or dysfunction of the chordae tendineae or papillary muscles in the absence of primary disease of the valve leaflets or annulus. The surgical management of these patients is the subject of a separate report.

Methods and Materials

During the period September 1965 to December 1970, 212 patients with mitral regurgitation were submitted to open operation by author M.P. In the majority of patients the valve demonstrated the typical effects of previous rheumatic infection. In a few of these ruptured chordae were also seen, but only as part of the rheumatic disorganization of the valve structures. Such patients have been excluded from this series. In 33 patients (16%) regurgitation was due to rupture or stretching of the chordae tendineae, or rupture of a papillary muscle. The leaflets and annulus were essentially normal, or showed only effects considered to be secondary to the subvalvular lesions, such as a dilated ring or redundant cusp tissue.
ASPECTS OF MITRAL REGURGITATION

Table 1

<table>
<thead>
<tr>
<th>Patient Classification According to Known Etiological Factors</th>
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<tbody>
<tr>
<td>Etiological factor</td>
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<tr>
<td>-------------------</td>
</tr>
<tr>
<td>1. Previous MI</td>
</tr>
<tr>
<td>2. Previous subacute bacterial endocarditis</td>
</tr>
<tr>
<td>3. Previous blunt chest trauma</td>
</tr>
<tr>
<td>4. Idiopathic</td>
</tr>
<tr>
<td>Acute MI</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

Classification

Chordal Abnormalities

The 31 patients with chordal abnormalities were classified into four groups (table 1). The affected chordae were related to the leaflet to which they were attached (table 2).

In six patients (mean age 61 years) there was a definite history and electrocardiographic evidence of old myocardial infarction. Three of these patients had stretched and redundant aortic leaflet chordae, two had rupture of the aortic leaflet chordae, and one had rupture of the posterior leaflet chordae.

Three women (mean age 43 years) had been diagnosed and treated in the past for bacterial endocarditis. They all presented with rupture of the chordae to the aortic leaflet.

A 33-year-old man with ruptured posterior leaflet chordae had sustained an injury to the left anterior chest wall. The underlying pericardium was densely adherent both to the ribs and the left ventricle.

The largest group, 21 patients, had no obvious predisposing factor for chordal rupture or stretching. Nineteen of these patients were aged over 45 years (mean age 59 years); in 17 the chordae had ruptured and in two they were stretched and redundant. Chordae to the posterior leaflet only were involved in 14 patients and to the aortic leaflet only in three patients. One patient had rupture of the chordae to both leaflets at the posteromedial commissure and one patient, rupture of the chordae to both leaflets at the anterolateral commissure. The other two patients with idiopathic chordal rupture were a boy, aged 7, and a 19-year-old girl.

The involved chordae were also related to their papillary muscle of origin (table 3). Ruptured chordae to the central portion of a leaflet were always considered to have arisen from both papillary muscles. Chordae arising from the posteromedial papillary muscle only were involved in nine patients, from both papillary muscles in 19 patients, and from the anterolateral muscle only in three patients.

Papillary Muscle Rupture

These two patients (aged 59 and 71 years) had both ruptured the posteromedial papillary muscle following acute myocardial infarction.

Clinical Features

Chordal Abnormalities

There were 20 men and 11 women with chordal lesions (fig. 1). In the postinfarction and idiopathic groups there were 19 males and eight females, a ratio of 2.4 : 1.

In 24 patients (77%) dyspnea began quite suddenly and was progressive and disabling; ten (32%) dated their dyspnea from a sudden episode of retrosternal or epigastric discomfort. All but three of these 24 patients required surgical treatment within 1 year. Five patients experienced slowly progressive dyspnea for longer than 2 years. Three patients gave a history of several episodes of heart failure following bacterial endocarditis with increasing breathlessness and palpitations.

Twenty-five patients (81%) presented in sinus rhythm and six patients presented in atrial fibrillation. The central venous pressure was raised in 16 patients, of whom 12 were in sinus rhythm and showed a dominant a wave. Systemic hypertension was found in four patients.

A systolic thrill was palpable in 20 patients (65%). The pulmonary component of the second sound was accentuated in 24 patients (77%) and a third heart sound was heard in 18 patients (58%). All but one patient had a pansystolic murmur maximal in the mitral area but with wide radiation to the precordium and back.

Papillary Muscle Rupture in Two Patients

A woman, aged 71, suddenly developed pulmonary edema 1 month after inferior myocardial infarction.

Table 2

<table>
<thead>
<tr>
<th>Leaflet Involved in 31 Patients with Ruptured or Stretched Chordae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leaflet chordae</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Aortic</td>
</tr>
<tr>
<td>PM commissural</td>
</tr>
<tr>
<td>Posterior</td>
</tr>
<tr>
<td>AL commissural</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

Abbreviations: SBE = subacute bacterial endocarditis; PM = posteromedial; AL = anterolateral; S = stretched; R = ruptured.
**Table 3**

**Papillary Muscle Involved in 31 Patients with Chordal Lesions**

<table>
<thead>
<tr>
<th>Papillary muscle</th>
<th>Postinfarction</th>
<th>SBE</th>
<th>Trauma</th>
<th>Idiopathic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posteromedial</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Both</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>12</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>21</td>
<td>31</td>
</tr>
</tbody>
</table>

*Ruptured chordae to the central portion of a leaflet were assumed to have originated from both papillary muscles.

Sinus rhythm, a third heart sound, and a pansystolic murmur at the apex were noted. The blood pressure was normal. At operation 4 weeks later, that part of the posterior papillary muscle controlling the aortic leaflet was found to be ruptured.

A man, aged 59 years, suddenly developed gross heart failure and cardiogenic shock 9 days after acute myocardial infarction. He presented with peripheral vasoconstriction, sinus tachycardia, and a systolic blood pressure of 60 mm Hg. A soft pansystolic murmur was identified at the apex. Emergency operation was performed and the entire posterior papillary muscle was found to have ruptured.

**Electrocardiogram**

All six patients in the “postinfarct” group were in sinus rhythm and had electrocardiographic evidence of inferolateral ischemia and old myocardial infarction. In four patients the infarct was inferior, in one posterior, and in one anterior.

The three patients in the postinfarctive group showed ST-T changes similar to those of inferolateral ischemia; two were in atrial fibrillation and both had voltage criteria for left ventricular hypertrophy. The patient with an old chest injury was in sinus rhythm and had inferolateral ischemic changes.

In the “idiopathic” group the youngest patient (aged 7) with ruptured chordae and the 19-year-old girl had voltage criteria for left ventricular hypertrophy but no ST-T wave changes. Both were in sinus rhythm. Twelve of the other 19 older patients had ST-T segment changes suggestive of inferior, lateral, or inferolateral ischemia, and seven of these 12 had voltage criteria for left ventricular hypertrophy; two were in atrial fibrillation. Two other patients had anterolateral ST-T segment changes suggestive of ischemia and one of these was in atrial fibrillation. Three other patients had normal ST-T segments; two had voltage criteria for left ventricular hypertrophy, and all were in sinus rhythm. The remaining two patients had electrocardiographic changes suggestive of previous myocardial infarction (one a true posterior infarct with left-axis deviation and atrial fibrillation, and the other an inferior infarct), but without any historical evidence of myocardial infarction.

**Figure 1**

Clinical features in 31 patients with ruptured or stretched chordae.
ASPECTS OF MITRAL REGURGITATION

The two patients with rupture of the posteromedial papillary muscle were in sinus rhythm and had electrocardiographic evidence of recent inferior myocardial infarction.

**Chest X-ray**

The preoperative chest radiographs were assessed with respect to heart size, left atrial size, the appearance of the upper lobe pulmonary veins, and the presence or absence of septal lines, pulmonary effusions, pleural effusions, and mitral valve calcification. Left atrial size was assessed in four grades (0–III) according to the criteria of Goodwin et al. The appearance of the upper lobe pulmonary veins was assessed in four grades according to Simon. Assessment of chest radiograph was impossible in one patient who had previously undergone left pneumonectomy and thoracoplasty for tuberculosis.

**Chordal Abnormalities**

Twenty-four patients who were in sinus rhythm had an average cardiothoracic index of 54% (range 43–63%). Fourteen of these had a normal or slightly enlarged cardiac diameter (cardiothoracic index less than 55%) and a normal or only slightly enlarged (grade 0) left atrium (fig. 2). The remaining 10 patients had a cardiothoracic index between 55% and 63% but none had more than moderate enlargement (grade I) of the left atrium. In contrast, the six patients who were in atrial fibrillation had a average cardiothoracic index of 63% (range 58–71%), and all but one had grade II enlargement of the left atrium. Aneurysmal dilatation of the left atrium (grade III) was not seen.

Upper-lobe pulmonary venous dilatation was present in all but one patient. In three patients this was marked (grade IV), in 14 patients it was obvious (grade II–III), and in 12 patients it was slight (grade I). Only one patient with a chordal lesion (who also had a left ventricular aneurysm) presented in pulmonary edema. This patient and four others had septal lines (Kerley B). A pleural effusion (fig. 3) was present in six patients in this series. Mitral valve calcification was never seen.

**Papillary Muscle Rupture**

The chest radiograph in both patients showed pulmonary edema with a normal left atrial size. The cardiothoracic indices were 48% and 51%, respectively.

**Cardiac Catheterization and Angiography**

Routine right and left heart catheterization was carried out in 25 patients, pressure measurement being referred to the midchest level. Cardiac output was determined according to the Fick principle. Mitral regurgitation was assessed by left ventricular cineangiography in the right anterior oblique position, which

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Posteranterior chest radiograph in a patient with gross mitral regurgitation in sinus rhythm due to rupture of chordae to the posterior leaflet. The cardiothoracic ratio is normal and there is marked dilatation of the upper-lobe veins.

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also permitted measurement of the ejection fraction. Coronary arteriograms were not performed.

The patients were divided into the "postinfarct" group (six patients), and the remainder (19 patients) which included the man with blunt chest trauma, one patient in the postinfective group, and 17 patients in the idiopathic group. The results are shown in table 4.

There was a wide variation in hemodynamic findings in these 25 patients. Since no patient had mitral stenosis the usually elevated mean pulmonary artery wedge pressures reflected elevation of the left ventricular end-diastolic pressures. An apparent gradient across the mitral valve was occasionally seen (fig. 4). This phenomenon may be partially explained by a phase lag in the pulmonary artery wedge pressure (PAWP) trace. The absence of a true gradient across the mitral valve is indicated by the rapid descent of the V wave and the reversal of the PAWP/LV gradient in end-diastole. The average values for the V wave in the wedge pressure and the average pulmonary artery pressures were very high, but there was no significant difference between these values in the postinfarct group and the other patients. The characteristic high-peaked V wave of subvalvular mitral regurgitation (fig. 4) was seen in all but three of the 25 patients. The mean valve for the cardiac index in the 25 patients was normal but tended to be lower in the postinfarct group.

The left ventricular end-diastolic pressure was significantly higher in the postinfarct group than in the other patients ($P = 0.02$), but the most significant difference ($P < 0.01$) between these two groups of patients was in the ejection fraction. The ejection fraction was low in every patient in the postinfarct group (mean $0.44 \pm 0.12$) but nearly always normal in the other patients (mean $0.70 \pm 0.10$). No hemodynamic distinction was possible between patients with stretched and patients with ruptured chordae.

In the patient with complete rupture of the posteromedial papillary muscle transseptal cardiac catheterization showed the left atrial V wave to approximate to the left ventricular systolic pressure.

Figure 3

Posteroanterior chest radiograph 3 weeks after onset of symptoms due to rupture of chordae to the posterior leaflet. Bilateral pleural effusions are present although the heart and left atrium are still of normal size.
Table 4

Cardiac Catheterization Data in 25 Patients*

<table>
<thead>
<tr>
<th>Catheter finding</th>
<th>Infarct group (N = 6)</th>
<th>Noninfarct group (N = 19)</th>
<th>All patients (N = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PAWP (mm Hg)</td>
<td>28.8 ± 13.1</td>
<td>20.0 ± 9.0</td>
<td>22.3 ± 10.7</td>
</tr>
<tr>
<td>PAWP (mm Hg); V wave</td>
<td>50.0 ± 21.0</td>
<td>40.0 ± 14.0</td>
<td>42.0 ± 17.0</td>
</tr>
<tr>
<td>PAP (mm Hg):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>71.0 ± 27.0</td>
<td>54.0 ± 22.0</td>
<td>59.0 ± 25.0</td>
</tr>
<tr>
<td>Diastolic</td>
<td>34.0 ± 13.0</td>
<td>26.0 ± 11.0</td>
<td>28.0 ± 12.0</td>
</tr>
<tr>
<td>Mean</td>
<td>49.0 ± 18.0</td>
<td>36.0 ± 15.0</td>
<td>40.0 ± 17.0</td>
</tr>
<tr>
<td>PVR (units/m²)</td>
<td>9.6 ± 6.6</td>
<td>5.3 ± 5.2</td>
<td>6.4 ± 6.5</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>28.0 ± 12.0†</td>
<td>16.0 ± 7.0†</td>
<td>19.0 ± 11.0</td>
</tr>
<tr>
<td>CI (liters/min/m²)</td>
<td>2.1 ± 0.7</td>
<td>3.0 ± 1.0</td>
<td>2.8 ± 1.0</td>
</tr>
<tr>
<td>EF</td>
<td>0.44 ± 0.12†</td>
<td>0.70 ± 0.10†</td>
<td>0.57 ± 0.17</td>
</tr>
</tbody>
</table>

*The findings in the six postinfarct patients are compared with those in 19 of the other patients with chordal lesions. Only the differences between the LVEDP and EF are statistically significant.

†P = 0.02.

| Abbreviations: PAWP = pulmonary artery wedge pressure; PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; EF = ejection fraction; N = number. |

Discussion

Although there have been several series of patients with ruptured chordae reported in the last decade, most authors have included patients with a rheumatic etiology. As a result, confusing and often contradictory statements have been made about the incidence, etiology, clinical presentation, and surgical management of subvalvular mitral regurgitation. This study of 33 carefully documented and fully investigated patients was carried out in an attempt to identify the etiology for "idiopathic" chordal rupture and to clarify the preoperative diagnosis of nonrheumatic mitral regurgitation.

Etiology

Idiopathic Chordal Rupture

In classifying this series no obvious cause for chordal rupture could be identified in 19 patients. Seventeen were over the age of 45 and their average age (59 years) corresponded closely with that reported in other series of similar patients. This higher age range suggests that degenerative changes are the most likely cause of chordal rupture in these patients. Cystic, mucoid, and hyaline degeneration of the leaflets and chordae have been described, but the pathologic evidence in every series has been scanty. In this series the mitral valves were rarely excised but the few leaflets and chordae examined microscopically looked remarkably normal. It has also been suggested that other factors more common in old age, such as aortic regurgitation and systemic hypertension, may place an additional strain on the mitral chordae. None of the patients in this series had aortic regurgitation, and only four had moderate systemic hypertension.

The observation that patients with a myocardial infarct could develop mitral regurgitation due to chordal abnormalities first suggested that ischemia of the papillary muscles and chordae could be a cause of "idiopathic" chordal rupture in the older patients. It has been customary to relate ruptured

Figure 4

Tracing taken from simultaneous left ventricular (L.V.) and pulmonary artery wedge pressure (P.A.W.P.) traces showing the high V wave in the left atrium following ruptured mitral chordae.
chordae to the leaflet to which they were attached. However, in this series it was usual for chordae to rupture near the papillary muscle tip suggesting that it might be more relevant to consider the papillary muscle from which the chordae had become detached. It is possible that papillary muscle ischemia weakens the chordal attachments and causes them to become attenuated and rupture. The vulnerability of the papillary muscles to an inadequate blood supply has been previously recognized. If myocardial ischemia were a factor, then the area of myocardium most likely to be affected would be the posterior wall of the left ventricle and the posteromedial papillary muscle tip which are supplied by small terminal branches of the coronary arteries furthest from the main source. Extensive patchy fibrosis of the posteromedial papillary muscle has been frequently observed in patients with myocardial ischemia and papillary muscle fibrosis has also been noted at postmortem in patients with “idiopathic” ruptured chordae.

We found that chordae attached to the posteromedial papillary muscle were involved in all but one of the 17 older patients with “idiopathic” chordal rupture but the most usual finding was rupture of chordae arising from both papillary muscles. The findings at operation did not indicate which chorda was the first to rupture and initiate the derangement. (Once a chorda has ruptured, an additional strain is immediately placed upon adjacent chordae, and progressive stretching and rupture of successive chordae probably occur.) The ST-T segment abnormalities seen in 15 of these 17 patients, although not specific, could have been due to myocardial ischemia and impairment of the papillary muscle blood supply.

This hypothesis does not entirely explain why chordae attached to the posterior leaflet are preferentially ruptured in the idiopathic group. The aortic leaflet of the mitral valve is larger, more mobile, and lies between the “inflow” and “outflow” tracts of the left ventricle, and is therefore subjected to greater stresses and strains. The chordae to its medial half also arise from the posteromedial papillary muscle, and might be expected to rupture even earlier than those to the medial half of the posterior leaflet. Preoperative coronary arteriography in patients with ruptured chordae and postmortem studies of the blood supply to the papillary muscles are being pursued in an attempt to gain further information on this subject.

Two patients in the “idiopathic” group had stretching and attenuation of chordae producing severe mitral regurgitation. We believe that stretching of the chordae may occur initially in every patient as the chordae immediately adjacent to ruptured chordae are always considerably elongated. Further stretching or later rupture of some chordae eventually results in severe mitral regurgitation. Spontaneous rupture of mitral chordae in young people as seen in two patients in this group is very uncommon, but may be associated with congenital malformation of the valve leaflets or chordae.

Postinfec tive and Traumatic Chordal Rupture

Chordal rupture following subacute bacterial endocarditis has been recognized for many years, and such patients form the majority in earlier reports. Bacterial endocarditis more frequently attacks the aortic leaflet and chordae as might be expected from its position in the left ventricle, and rupture of these chordae was found in all three of our patients. Chordal rupture is rare following blunt chest trauma.

Postinfarction Mitral Regurgitation

In our experience mitral regurgitation requiring surgery in patients with a definite antecedent myocardial infarct has been due to a different form of subvalvular defect. The most common finding (five out of six patients) was stretching and attenuation, and sometimes rupture of all the chordae to the aortic leaflet, causing gross overshoot of this leaflet into the left atrium. Only one patient had rupture of the posterior leaflet chordae similar to that usually seen in the idiopathic group.

All six patients had impaired left ventricular function as judged by left ventricular cineangiography. The presence of akinetic or dyskinetic areas in the myocardium may alter the force of direction of papillary muscle contraction, and thus impose an abnormal strain on the chordae. Involvement of a papillary muscle in the infarct may also result in stretching and rupture of the chordae originating from it, although fibrosis of the posteromedial papillary muscle was only seen at operation in one patient. These hypothetical mechanisms by which left ventricular and papillary muscle dysfunction could produce mitral regurgitation were first proposed by Burch and associates. Few surgeons have been able to demonstrate such dysfunction of the mitral valve in the beating heart at operation, and in our experience it is the structural change of chordal attenuation and rupture that most commonly produces mitral regurgitation following myocardial
infarction. In this group of patients it seems likely that it is the abnormal stresses and strains (to which the aortic leaflet is especially subjected because of its size and position) which produce the chordal abnormalities.

Complete rupture of the posteromedial papillary muscle following acute myocardial infarction has often been reported at autopsy, but only occasionally at surgery, as rapid deterioration and death from the resulting gross mitral regurgitation is the usual outcome. Survival for over a year may rarely occur, but is usually associated with only partial rupture of the muscle, as was found in the first of our two patients.

Clinical and Hemodynamic Findings

This series of patients with ruptured and stretched mitral chordae clearly illustrates the clinical syndromes of nonrheumatic subvalvular regurgitation. While chordal rupture may rarely occur before the age of 50, it was most common in the sixth and seventh decades and affected males much more frequently than females. In contrast to the findings of others, we have found that patients with sudden onset of symptoms (77%) due to ruptured or stretched chordae nearly all deteriorate and require surgical treatment within 1 year. The duration of symptoms was shortest in the postinfarct patients in whom the left ventricle was least able to accommodate the hemodynamic burden of sudden mitral regurgitation.

The presence of sinus rhythm (81%) is an important diagnostic finding in these patients and contrasts markedly with the finding of atrial fibrillation in most patients with symptomatic rheumatic mitral regurgitation. It should be noted that atrial fibrillation can occur with idiopathic chordal rupture and its development was noted in two patients in the idiopathic group within weeks of the onset of symptoms.

While an accentuated pulmonary component of the second sound and a third heart sound were frequently recorded in this series, a fourth heart sound was not heard clinically, although increased atrial contraction (double impulse) was frequently detected by palpation. Cohen and associates believed that an atrial sound was an important clue to the diagnosis of ruptured chordae.

In contrast to the findings of others, the radiation of the pansystolic murmur in this series had no diagnostic value in determining which chordae were ruptured. Selzer, Kelly, Vanmitanby, Walker, Gerbode, and Kerth believed that the auscultatory findings following rupture of the chordae to the posterior leaflet mimicked those of aortic stenosis. We have not found this to be so although few of our patients had the typical lesion found in their series, i.e., rupture of a few chordae in the center of the posterior leaflet producing a hoodlike deformity of the central portion of this leaflet. The “murmur on top of the head” described by Merendino and Hessel must be rare, although the murmur of ruptured chordae was often heard over the spine from neck to sacrum with involvement of either leaflet. This wide radiation, and the frequently associated systolic thrill, are characteristic of subvalvular mitral regurgitation and are presumably due to the powerful regurgitant jet impinging on the walls of a small left atrium.

In our experience the ECG is of more value in determining the leaflet involved since five out of six patients with ECG evidence of antecedent myocardial infarction have involvement of the aortic leaflet. The identification of these postinfarct patients is important as we have found that their prognosis is poor following mitral valve surgery.

A detailed survey of the plain chest radiograph showed that the presence or absence of atrial fibrillation was the most important determinant of cardiac and left atrial size. A small left atrium has been described as “the most useful sign in ruptured mitral chordae” and was found in approximately 50% of the patients in this series, all of whom were in sinus rhythm and had a normal cardiothoracic ratio. Another third of the patients had mild-to-moderate left atrial enlargement with sinus rhythm, whereas five out of six patients in atrial fibrillation had considerable left atrial enlargement and increase in heart size. While a direct relationship between the cardiac and left atrial size and the duration of symptoms was usually found, it was not inevitable, and some patients very quickly develop considerable cardiac enlargement in sinus rhythm. It has been stated that the pulmonary vasculature is normal in patients with ruptured chordae, but dilatation of the upper lobe pulmonary veins was almost always present in this series, reflecting the elevated left atrial pressures.

At cardiac catheterization the patients in the idiopathic group usually showed the typical findings of a high V wave in the pulmonary artery wedge pressure trace, elevated pulmonary artery pressures, a normal cardiac output, and good left ventricular function. By contrast, left ventricular contractility was always impaired on the left ventricular cineangiogram in the postinfarct patients, and the
calculated ejection fractions in these patients were always significantly lower than the usually normal values found in the other patients. The ejection fraction may not be a strictly accurate method for estimating left ventricular function in patients with severe mitral regurgitation, since it will be influenced by such factors as the absolute end-diastolic volume, the degree of mitral regurgitation, and the size and compliance of the left atrium. However, there were no significant differences in these other factors between the postinfarct and the remaining patients submitted to cardiac catheterization. Assessment of left ventricular function prior to surgery is important since we found that patients with impaired left ventricular contractility and a low ejection fraction did poorly following mitral valve replacement. Coronary arteriography was not performed but we believe that it would be a valuable additional investigation in the postinfarct group.

Rupture of a papillary muscle following a myocardial infarct presents with gross pulmonary edema and shock as was seen in both patients in this series. The finding of a systolic murmur, which is often soft and unaccompanied by a thrill, may be the only clinical indication of mitral regurgitation. In the few patients who survive for any length of time, the chief differential diagnosis is perforation of the ventricular septum.

Acknowledgment

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