Nonrheumatic Mitral Insufficiency

Determination of Operability and Prognosis

By Martin Klughaupt, M.D., M. D. Flamm, Major, USAF (MC), E. William Hancock, M.D., F.A.C.P., and Donald C. Harrison, M.D., F.A.C.C.

SUMMARY

Twenty patients with mitral insufficiency (MI) due to ruptured chordae tendineae, papillary muscle infarction, or left ventricular enlargement with no organic disease of the mitral valve have been studied during the past 3 years. Nineteen had valve replacement, and one had plastic repair of ruptured chordae tendineae; all have been followed from 6 months to 3 years. Preoperative data were different in those who were improved by surgery (13 patients) and those who were not (seven patients). The group in which good results were obtained had a short history of heart failure, little or no evidence of left atrial (LA) enlargement in the electrocardiograms and x-rays, and striking LA v waves at catheterization. In the group demonstrating poor results, heart failure was of longer duration, gross four-chamber cardiomegaly was present, and LA v waves were only moderate. Thus, patients with nonrheumatic mitral insufficiency with high-pressure LA regurgitant waves and only moderate LA enlargement are likely to benefit from mitral valve surgery, even when the primary cardiovascular disease is hypertension, arteriosclerosis, or a cardiomyopathy. These patients generally have ruptured chordae tendineae.

Additional Indexing Words:

Hypertension Mitral valve surgery Ruptured chordae tendineae Left atrial v waves Atrial compliance

RECOGNITION of severe and rapidly progressive mitral insufficiency in patients with no background of rheumatic valvular disease has been increasing recently. These patients give no history of acute rheumatic fever or of a long-known heart murmur and typically present with advanced congestive heart failure, a murmur suggesting mitral insufficiency, and regular sinus rhythm. Often there is a background of arteriosclerotic heart disease, with or without myocardial infarction, or of systemic hypertension. The anatomic findings at operation, in those who have undergone mitral valve surgery or at postmortem examination, have included ruptured chordae tendineae,1-4 papillary muscle infarction with or without rupture,5,6 and left ventricular enlargement without associated organic defects of the mitral valve.7 Because mitral valve operations in these circumstances are relatively hazardous and may not alter the progressive course of cardiac failure, it is important that valid criteria for the selection of patients for operation be established. This report describes those clinical, hemodynamic, and radiographic factors that correlate with a favorable response to operation and that may serve as criteria of operability in nonrheumatic mitral insufficiency.

From the Cardiology Division, Department of Medicine, Stanford University School of Medicine, Palo Alto, California.

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Address for reprints: Donald C. Harrison, M.D., Cardiology Division, Stanford University School of Medicine, Palo Alto, California 94304.

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Methods

Our group consisted of 20 patients who survived mitral valve operation at Stanford University Hospital from 1964 to 1967, because of progressive congestive heart failure with signs of mitral insufficiency, not of rheumatic origin. None gave a history of rheumatic fever, and only one had a murmur that had been noted before the age of 40 years. Prior to surgery, each patient had a 12-lead electrocardiogram, chest x-rays, and a phonocardiogram. Each underwent routine right heart catheterization by standard methods. The mid-axillary line was used as the reference point for all intracardiac pressures. The Fick method was used to determine all cardiac outputs. In addition, 12 patients underwent transseptal left heart catheterization, five had left ventricular angiography, four had coronary angiography, and five underwent repeat catheterization postoperatively.

Preoperative cardiac catheterization in each case showed a left atrial or pulmonary artery wedge pulse contour consistent with significant mitral insufficiency, in other words, elevation of pressure with a dominant $v$ wave and rapid $y$ descent. Nineteen patients underwent mitral valve replacement with a Starr-Edwards prosthesis, and in one patient with ruptured chordae tendineae plastic repair was done. After 6-month to 3-year follow-ups, the patients were divided into two groups: (1) those who had distinctly benefited from operation, as evidenced by a postoperative reclassification using the New York Heart Association class at least two classes higher than the preoperative class (good result group); (2) and those whose status had not improved (poor result group), in which the postoperative course was one of progressive cardiac failure and death, or in which the New York Heart Association class was worse than, or equivalent to, that prior to surgery.

Results

Anatomic Findings

In no case was there found pathological or histological evidence of rheumatic valvulitis such as thickened, calcified leaflets or thickened, shortened chordae tendineae.

At operation 10 patients in this series had ruptured chordae tendineae, which involved the chordal attachments of the anterior mitral leaflet in five and the posterior leaflet in five. Five of these patients had documented hypertensive or arteriosclerotic cardiovascular disease, or both, while in five no underlying cardiac disease was documented. Three of the patients with idiopathic chordal rupture had involvement of the chordae supporting the anterior mitral leaflet, while in two, those chordae serving the posterior leaflet were torn. This contrasts with Selzer and associates' description of the selective involvement of chordae supporting the posterior leaflet in isolated chordal rupture. Of the 10, all benefited from surgery except one (J. Mack.), who died 6 months after operation and was found at autopsy to have diffuse myocardial fibrosis with widely patent coronary arteries. He had had a long history of hypertension.

Two patients in the series were found to have infarction of the posterior papillary muscles with prolapse of the posterior mitral leaflet. In both, myocardial infarctions of the inferior wall had been documented. One (C. M.) had sustained, in addition, rupture of the chordae tendineae supporting the anterior mitral leaflet. Both patients did well following operation.

In nine patients, no specific histological lesion of the mitral valve, papillary muscles, or chordae tendineae was found. Two patients had myocardial infarctions of the inferior wall, and five others were hypertensive. Three of the patients in this group improved considerably after operation, one continued to have intermittent congestive heart failure and remained unchanged from his preoperative status, and five died after several months of progressive postoperative deterioration. The final anatomic diagnosis after autopsy in two of these patients was idiopathic cardiomyopathy.

Comparison of Preoperative Data in Good and Poor Result Groups

The clinical and hemodynamic characteristics of the good and poor result groups are listed in tables 1 and 2, respectively. Several features deserve further comment:

Age and Sex

There was no significant difference between the two groups in these regards. The average age in the good result group was 54 and in the poor result group, 56 years. There was a marked predominance of males in both groups in contrast to the prevalence of...
females in rheumatic mitral valvular disease.

**Duration of Murmur**

With the exception of one patient (S.M., in whom a systolic murmur had first been noted in an insurance examination at the age of 24), there was no record of a murmur in any patient prior to the age of 40, or more than 6 years before the date of surgery. A typical apical systolic murmur had appeared within the year prior to surgery in eight of 13 patients in the good result group, and in two of seven patients in the poor result group. In only one patient (R.G.) was the murmur, because of its prominent radiation to the base and ejection quality, suggestive of aortic stenosis. He was subsequently found to have rupture of the chordae tendineae serving the posterior mitral leaflet, a lesion which has been described as producing a murmur which may simulate that of aortic stenosis.

There were no significant differences between the good and poor result groups in regard to the intensity of the murmur or the presence or absence of a left ventricular heave, third or fourth heart sound, or mitral diastolic flow murmur.

**Duration of Symptoms of Congestive Heart Failure**

In the good result group, there was a relatively short history of congestive failure. The average duration of symptoms was 17 months prior to the time of surgery, with seven of 13 patients having been symptomatic for 6 months or less. In the poor result group, on the other hand, the average duration of symptoms was 42 months, with five of seven patients having been symptomatic for 3 years or longer. In the good result group the murmur either preceded, or appeared simultaneously with, the onset of symptoms in all but one patient (C.M.), whereas in the poor result group, symptoms of congestive failure had preceded the appearance of the murmur in five of seven patients.

**Incidence of Arteriosclerotic and Hypertensive Cardiovascular Disease**

Five of the 13 patients in the good result group had had documented myocardial infarctions; in three of the five the murmur and symptoms of congestive failure appeared with, or shortly after, the acute episode. Hypertension was present in three patients in this group. In the poor result group, four of the seven patients were hypertensive, and one had had a myocardial infarction 15 months prior to surgery.

**Cardiac Rhythm Disturbances**

Only three of 13 patients in the good result group and one of seven in the poor result group had atrial fibrillation; the remainder had regular sinus rhythm. This again contrasts with the high incidence of atrial fibrillation in rheumatic mitral valvular disease.

**Chamber Hypertrophy and Enlargement**

Electrocardiographic criteria for left ventricular hypertrophy were met in five of the 13 patients in the good result group, while cardiac x-rays revealed left ventricular enlargement in 12 patients. Left atrial hypertrophy, however, was present in the electrocardiograms of only three patients in this group and definite left atrial enlargement in only five of the 13, roentgenographically (fig. 1). In the poor result group, on the other hand, gross cardiomegaly with involvement of all chambers was the most common x-ray finding. Left atrial hypertrophy was present electrocardiographically in five of seven patients in the poor result group, and in all seven patients this chamber was enlarged roentgenographically (fig. 2). In no patient in either group was mitral valve calcification seen on the x-rays.

**Findings at Cardiac Catheterization**

The most striking difference between the two groups hemodynamically was the amplitude of the left atrial or pulmonary artery "wedge" v wave (figs. 3 and 4). In the good result group, the v wave had an average height of 52 mm Hg and was 40 mm Hg or greater in all patients but one (fig. 4). In
### Table 1

**Nonrheumatic Mitral Insufficiency With Good Postoperative Result**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age(yr) and sex</th>
<th>Duration of murrur</th>
<th>Duration of symptoms of CHF*</th>
<th>Electrocardiogram†</th>
<th>Hemodynamic data‡</th>
<th>Operative findings</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.A.</td>
<td>60M</td>
<td>6 yr</td>
<td>5 yr</td>
<td>AF, LAH, LVE++</td>
<td>LA or PAW wave/mean: -/60/23 PA mean: 30 LV-EDP: 2.5 CI: 2.5</td>
<td>Ruptured chordae posterior leaflet; valve normal</td>
<td>Hypertension to 190/112</td>
</tr>
<tr>
<td>E.A.</td>
<td>56M</td>
<td>1 yr</td>
<td>1 yr</td>
<td>RSR, None, LVE+++ LAE+</td>
<td>18/55/27 PA mean: 50 LV-EDP: 1.6 CI: 1.6</td>
<td>Inferior wall myocardial infarction 1 yr prior to surgery</td>
<td>Preop. and P.O. diagnosis: cardiomyopathy</td>
</tr>
<tr>
<td>L.A.</td>
<td>52M</td>
<td>1 yr</td>
<td>1 yr</td>
<td>RSR, LVH, LVE+</td>
<td>18/46/26 PA mean: 41 LV-EDP: 3.7 CI: 3.7</td>
<td>LVE; valve normal</td>
<td>Inferior wall myocardial infarction 6 mo prior to surgery</td>
</tr>
<tr>
<td>F.G.</td>
<td>48M</td>
<td>6 mo</td>
<td>6 mo</td>
<td>RSR, LVH, LVE+ LAE+</td>
<td>34/52/29 PA mean: 50 LV-EDP: 3.1 CI: 3.1</td>
<td>Old infarct, posterior papillary muscle; LVE</td>
<td>Inferior wall myocardial infarction 6 mo prior to surgery</td>
</tr>
<tr>
<td>R.G.</td>
<td>45M</td>
<td>4 yr</td>
<td>2 mo</td>
<td>RSR, LVH, LAH, LVE+ LAE+</td>
<td>14/42/18 PA mean: 47 LV-EDP: 2.0 CI: 2.0</td>
<td>Ruptured chordae posterior leaflet; valve normal</td>
<td>Inferior wall myocardial infarction 6 mo prior to surgery</td>
</tr>
<tr>
<td>J.H.</td>
<td>45M</td>
<td>2 yr</td>
<td>2 yr</td>
<td>RSR, None, LVE+</td>
<td>14/51/18 PA mean: 9 LV-EDP: 2.0 CI: 2.0</td>
<td>Mild LVE; valve normal</td>
<td>Mild hypertension, to 160/100</td>
</tr>
<tr>
<td>N.J.</td>
<td>49M</td>
<td>4 mo</td>
<td>4 mo</td>
<td>RSR, None, LVE+</td>
<td>-/52/21 PA mean: 51 LV-EDP: 1.8 CI: 1.8</td>
<td>Ruptured chordae posterior leaflet; valve normal</td>
<td>Inferior wall myocardial infarction 6 yr prior to surgery</td>
</tr>
<tr>
<td>M.K.</td>
<td>56F</td>
<td>3 yr</td>
<td>5 mo</td>
<td>RSR, None, LVE+ LAE+</td>
<td>-/33/13 PA mean: 45 LV-EDP: 1.7 CI: 1.7</td>
<td>Ruptured chordae anterior leaflet; valve normal</td>
<td>Inferior wall myocardial infarction 6 yr prior to surgery</td>
</tr>
<tr>
<td>M.M.</td>
<td>64M</td>
<td>4 mo</td>
<td>4 mo</td>
<td>RSR, LAH, LVE+++</td>
<td>29/71/31 PA mean: 57 LV-EDP: 2.2 CI: 2.2</td>
<td>Ruptured chordae anterior leaflet; valve normal</td>
<td>Inferior wall myocardial infarction 6 yr prior to surgery</td>
</tr>
<tr>
<td>C.M.</td>
<td>51M</td>
<td>4 mo</td>
<td>4 yr</td>
<td>RSR, LVH, LVE+ LAE+</td>
<td>22/40/19 PA mean: 43 LV-EDP: 1.8 CI: 1.8</td>
<td>Ruptured chordae anterior leaflet; valve normal</td>
<td>Inferior wall myocardial infarction 6 yr prior to surgery</td>
</tr>
<tr>
<td>S.M.</td>
<td>55M</td>
<td>30 yr</td>
<td>4 yr</td>
<td>AF, None, LVE+++</td>
<td>-/64/26 PA mean: 41 LV-EDP: 1.5 CI: 1.5</td>
<td>Ruptured chordae anterior leaflet; valve normal</td>
<td>Asthmatic bronchitis and bronchiectasis since childhood</td>
</tr>
</tbody>
</table>
the poor result group, the average v wave amplitude was 34 mm Hg and was less than 40 mm Hg in six of the seven patients (fig. 4). It is noteworthy that the one patient in this group who did have a v wave greater than 40 mm Hg (J.H.) did not have progressive congestive failure postoperatively as did the others, but died suddenly while driving after 4 months of continued improvement. Mean left atrial or pulmonary artery wedge pressures, mean pulmonary artery pressures, and cardiac indices did not differ significantly in the two groups.

**Angiographic Findings**

Left ventricular cineangiography was done preoperatively in five patients in this series, and therefore, the findings are of limited significance. However, in two patients in the good result group, marked mitral regurgitation with relatively effective left ventricular contractility was seen, while in three patients in the poor result group the degree of regurgitation ranged from moderate to severe, with relatively poor ventricular contractility in each case.

**Discussion**

Normal function of the mitral valve depends on both the anatomic integrity of the valve leaflets, chordae tendineae, and papillary muscles, and the maintenance of appropriate spatial relationships between these elements.7

The patient who presents with congestive heart failure and an apical systolic murmur of relatively recent onset without evidence of long-standing rheumatic valvular disease may have surgically correctable mitral insufficiency due to any one of several distortions not involving the valve leaflets themselves. In recent years, the most frequently reported of these lesions has been rupture of the chordal tendineae which may occur in association with other diseases of the heart or as an “isolated” or “spontaneous” event. Myocardial infarction with involvement of a papillary muscle may produce mitral insufficiency either through frank rupture of the muscle or as the result of
### Table 2

**Nonrheumatic Mitral Insufficiency with Poor Postoperative Result***

| Patient | Age (yr) and sex | Duration of murmur | Duration of symptoms of CHF | Electrocardiogram | Cardiac hypertrophy | Cardiac series | LA or PAW a wave/mean | PA mean | LV-EDP | CI | Operative findings | Remarks
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>L.C.</td>
<td>55M</td>
<td>3½ yr</td>
<td>3½ yr</td>
<td>RSR</td>
<td>LVH</td>
<td>Four-chamber</td>
<td>30/36/28</td>
<td>55</td>
<td>26</td>
<td>3.4</td>
<td>Marked LVE; valve normal</td>
<td>Hypertension to 226/120. Autopsy: prosthetic valve normal; focal coronary arteriosclerosis without occlusion</td>
</tr>
<tr>
<td>J.H.</td>
<td>61M</td>
<td>Unknown</td>
<td>6 yr</td>
<td>RSR</td>
<td>None (left bundle-branch block)</td>
<td>Four-chamber</td>
<td>—/-54/34</td>
<td>2.3</td>
<td></td>
<td></td>
<td>LVE; valve normal</td>
<td>Autopsy: prosthetic valve normal; coronaries patent; probable cardiomyopathy</td>
</tr>
<tr>
<td>E.K.</td>
<td>60F</td>
<td>19 mo</td>
<td>6 yr</td>
<td>RSR</td>
<td>LVH</td>
<td>Four-chamber</td>
<td>—/-35/24</td>
<td>38</td>
<td>3.8</td>
<td>1.8</td>
<td>LVE; aortic leaflet thinned</td>
<td>Autopsy: prosthetic valve normal; coronaries patent; probable cardiomyopathy</td>
</tr>
<tr>
<td>J.M.</td>
<td>56M</td>
<td>5 mo</td>
<td>10 mo</td>
<td>RSR</td>
<td>LAH</td>
<td>LV+ + LAH+ +</td>
<td>16/33/15</td>
<td>33</td>
<td>2.7</td>
<td></td>
<td>LVE; normal valve</td>
<td>Inferior wall myocardial infarction 15 mo prior to surgery. Autopsy: prosthetic valve normal; severe coronary arteriosclerosis with myocardial infarcts</td>
</tr>
<tr>
<td>J.MacK.</td>
<td>52M</td>
<td>4 yr</td>
<td>8 mo</td>
<td>RSR</td>
<td>LVH</td>
<td>Four-chamber</td>
<td>11/26/17 (—/-26/18)</td>
<td>30</td>
<td>12</td>
<td>1.8</td>
<td>Ruptured chordae posterior leaflet; LVE</td>
<td>Hypertension to 170/112. Autopsy: prosthetic valve normal; coronaries patent; probable cardiomyopathy</td>
</tr>
<tr>
<td>J.S.</td>
<td>55M</td>
<td>18 mo</td>
<td>3½ yr</td>
<td>RSR</td>
<td>LVH</td>
<td>Four-chamber</td>
<td>23/39/25</td>
<td>25 (34 1 yr P.O.)</td>
<td>1.8</td>
<td>LVE; valve normal</td>
<td>Hypertension to 170/110; died; no autopsy</td>
<td></td>
</tr>
<tr>
<td>R.T.</td>
<td>56M</td>
<td>5 mo</td>
<td>4 yr</td>
<td>AF</td>
<td>LVH</td>
<td>LVE+ LAE+</td>
<td>—/21/14 (—/20/11 6 mo P.O.)</td>
<td>26</td>
<td>2.6</td>
<td></td>
<td>LVE; valve normal</td>
<td>Hypertension to 200/110</td>
</tr>
</tbody>
</table>

* Same abbreviations as in table 1.
functionally impairment of the infarcted but anatomically uninterrupted muscle. Finally, it has been recognized that left ventricular enlargement resulting from a variety of causes may be associated with mitral insufficiency without any anatomic lesion of the valve elements being present. Although this is often attributed to “dilatation of the mitral ring” it would, perhaps, be more appropriate to speak in terms of a distortion of the normal spatial relationship between the valve leaflets, papillary muscles, and chordae tendineae. In contrast to primary lesions of the chordae and papillary muscles, there is a paucity of information in the literature regarding the results of surgical intervention in this last but common category of mitral insufficiency.

Bacterial endocarditis occurring in hearts without anatomic valve disease has been cited as an important cause of chordal rupture. However, each of the four patients who presented at this institution during the study period with mitral insufficiency and
a history of endocarditis clearly had a background of rheumatic valvular disease with either a history of rheumatic fever or one of long-standing mitral murmur. In one of these patients, chordal rupture was responsible for the insufficiency; in the others, the valve leaflets were severely scarred and deformed by vegetations.

In reporting their experience with ruptured chordae tendineae, Roberts and co-workers postulated that patients with high left atrial pressures and relatively small left atrial size would be most likely to benefit from surgery. Presumably, this combination of findings would suggest that the insufficiency was of relatively short duration and that the muscular and elastic properties of the left atrial wall had not been sufficiently altered to produce a greatly compliant chamber in which pressure would be moderated by an expanded volume. Similarly, the patients of Austen and his co-workers, who underwent surgery for postinfarction papillary muscle rupture with fairly good results, exhibited small left atria radiographically in the face of tall left atrial v waves at catheterization.

Our findings, in general, are in agreement with these reports. Thus, the patients in our good result group had short histories of congestive heart failure, striking left atrial or pulmonary artery wedge v waves, and relatively small left atria, radiologically. In the poor result group, on the other hand, there was a longer history of congestive heart failure, gross left atrial enlargement, and relatively small left atrial or pulmonary artery wedge v waves. Furthermore it would seem, on the basis of our experience, that these principles apply not only to those patients with anatomic lesions of the valve apparatus but also to those who have sustained distortion of the spatial relationship of its components due to changes in the left ventricle. Thus, three patients in this series who responded favorably to mitral valve
replacement had no specific anatomic lesion of any of the valve elements when examined pathologically. All three had relatively short histories of congestive heart failure, prominent left atrial or pulmonary artery wedge v waves, and unremarkable left atrial radiographically.

One would expect that the patients most likely to benefit from mitral valve surgery would be those with a great deal of mitral regurgitation and only mild-to-moderate left ventricular myocardial disease. That a long history of congestive heart failure is correlated with a poor operative result is not surprising, considering that long-standing congestive failure may be associated with irreversible myocardial changes. Similarly, the presence of a markedly dilated left atrium in patients whose primary disease lies below the mitral atrioventricular ring would signify advanced and perhaps irreversible disease. On the other hand, a tall, peaked regurgitant wave in the left atrial tracing would imply not only a large regurgitant flow but also a relative preservation of normal left atrial size and compliance, and hence a relatively recent onset of mitral insufficiency.

It should be stressed that these principles do not apply to patients with rheumatic valvular disease as they do to the patients discussed in this paper. Thus, in rheumatic mitral insufficiency, enlargement of the left atrium may be due not only to the hemodynamic changes resulting from the valvular lesion but also to direct involvement of the atrial myocardium by the rheumatic process. Left atrial enlargement does not then have the same significance and does not correlate to the same degree with the severity of the lesion producing regurgitation as it does in the nonrheumatic patient. For the same reason, a relatively small left atrial v wave does not have the same unfavorable prognostic import in rheumatic mitral insufficiency as it does in nonrheumatic insufficiency.

Left ventricular angiography has often been relied upon to assess the severity of mitral regurgitation and the integrity of myocardial function in patients considered for surgery. There is no doubt that this is useful, and we now do this in addition to cardiac catheterization in most patients of the type presented in this report. However, the risks of left heart angiography are substantially greater than those of right heart catheterization. In addition the data obtained by routine cineangiograms are qualitative and often subject to varying interpretation. Quantitative determination of left ventricular volumes and ejection fraction should be more helpful but is not easily applied in most laboratories, and this is also true of other quantitative methods for assessing mitral regurgitation. For these reasons the simple objective features discussed, particularly the left atrial v-wave amplitude, seem worth emphasizing as guides to the selection of patients for surgery.

In conclusion, any patient with disabling cardiac symptoms and a murmur suggestive of mitral insufficiency should be considered a potential candidate for mitral valve surgery, even if the primary cardiovascular disease seems to be hypertension, arteriosclerosis, or a cardiomyopathy. If the symptoms are of less than 2 years' duration, the left atrium is not grossly enlarged, and pulmonary artery wedge v waves exceeding 40 mm Hg are found at right heart catheterization, surgery can be recommended in most instances. Left heart catheterization with left ventricular cineangiography will provide further information regarding the degree of insufficiency and left ventricular myocardial dysfunction and should be done in selected patients in whom the indications for surgery are unclear.

Acknowledgment

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