Severe Mitral Regurgitation Secondary to Ruptured Chordae Tendineae

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RUPTURE of the chordae tendineae is an uncommon event that may lead to sudden mitral regurgitation, the severity of which generally depends on the number and location of the chordae involved. It has been reported to occur with active bacterial or rheumatic endocarditis, in association with healed rheumatic valvulitis, following healed bacterial endocarditis or trauma, or "spontaneously" in hearts without pre-existing mitral valve disease. In contrast to patients with the usual form of rheumatic mitral regurgitation, those with ruptured chordae tendineae usually die within a year, although a few cases of prolonged survival have been reported. With the availability of satisfactory surgical techniques for the treatment of mitral regurgitation, accurate clinical assessment of the lesion has obvious therapeutic implications.

The purpose of this communication is to report our experience in 15 patients with severe mitral regurgitation due to ruptured chordae tendineae. Pertinent features in the clinical evaluation that aid in the diagnosis are discussed. Particular emphasis is placed on the eight patients undergoing open-heart surgery, six of whom were studied by cardiac catheterization preoperatively. Three of these eight patients are living, and two show marked improvement 5 months and 3 years after surgery. Postoperative study of the third patient by cardiac catheterization combined with angiography has shown marked improvement in cardiac function and virtual disappearance of mitral regurgitation.

Materials and Methods

The 15 patients were obtained for study from the autopsy or operative files at the Massachusetts General Hospital covering the period between 1950 and 1964. All had received 12-lead electrocardiograms and chest films. Six underwent right and left heart catheterizations. Right heart study was performed in the routine manner. Left heart catheterization was carried out either by the Bjork, retrograde, or transseptal technics. Left ventricular angiography was done in four patients with use of a biplane Elema rapid film changer. Open-heart surgery with use of a Kay-Cross disc oxygenator and DeBakey pump was performed on eight patients.

Case Studies

Pertinent clinical and laboratory data are presented in tables 1 and 2.

Etiology

Nine patients had pre-existing rheumatic mitral valvulitis. Of these, four had evidence of healed bacterial endocarditis, two showed active bacterial endocarditis, and one, non-bacterial endocarditis. One patient had Ehlers-Danlos syndrome with multiple cardiac defects.

The etiology was considered to be "spontaneous" in six patients. Two had aortic regurgitation secondary to healed rheumatic valvulitis. One patient had achondroplasia with a coexistent atrial septal defect.

Clinical Histories

In the rheumatic group, three patients (M.K., L.D., C.B.) had symptoms referable to heart disease from 1½ to 10 years in duration. Symptoms had been present no more than 5 months in the remaining six. Eight patients experienced intractable left ventricular failure in the final days or months of their illnesses, although two had important complicating factors, such as subacute bacterial en-
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doccarditis and severe aortic regurgitation, that significantly influenced their clinical course.

Two of the six patients with "spontaneous" rupture had a clinical history extending beyond 10 months. Symptoms of myocardial insufficiency had been present in F.A. for over 6 years and in L.A. for 18 months. In the latter, aortic regurgitation was the probable cause of his early symptoms, since rupture of the chordae tendineae would best explain his abrupt deterioration 2 months prior to death.

Physical Examination

Fourteen patients had a harsh grade-IV/VI apical pansystolic murmur; in the remaining patient the murmur was grade III. It was well transmitted to the left axilla and left lung base in nine. An apical systolic thrill was felt in seven instances and a third sound heard in six. The mitral first sound was accentuated in three patients (all in the rheumatic group) and the pulmonic component of the second sound was increased in 13. Findings consistent with severe aortic regurgitation were noted in two patients (L.D., D.B.) while moderate aortic regurgitation was thought to be present in three additional patients (L.A., M.G., M.O.B.). Three patients in the rheumatic group had grade II to III apical diastolic rumbles (L.D., C.M., M.O.B.). Basal systolic ejection murmurs of grade II to III intensity were heard in eight patients with transmission into the neck in four.

Electrocardiographic Findings

Atrial fibrillation was present in seven patients and normal sinus rhythm in eight. Left ventricular hypertrophy was seen in 13 patients, one of whom (L.A.) showed a pattern of left ventricular diastolic overload. Two also had evidence of right ventricular hypertrophy on the basis of right axis deviation. Only one patient with multiple cardiac defects showed isolated right ventricular hypertrophy.

Roentgenologic Findings

All patients had a cardiothoracic ratio in excess of 50 per cent. Three patients in the "spontaneous" group had only slight enlargement of the left atrium (fig. 1) while two (R.D. and F.A.) had moderate, and the remaining case (H.S.), marked left atrial enlargement. Every patient in the rheumatic group showed moderate to severe left atrial enlargement. Findings consistent with left ventricular hypertrophy and pulmonary congestion were present in all patients. Two cases in the rheumatic group had calcification in the region of the mitral valve.

Figure 1

Patient S.S. Postero-anterior and lateral views of the chest. No significant left atrial enlargement is present, despite demonstration of severe mitral regurgitation by angiocardiography.
### Clinical and Anatomic Data

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age</th>
<th>Sex</th>
<th>Duration of symptoms</th>
<th>Sudden deterioration</th>
<th>History of murmurs</th>
<th>Sudden onset of murmurs</th>
<th>Apical systolic murmurs, grade</th>
<th>Associated murmurs, grade and type</th>
<th>Electrocardiogram Rhythm</th>
<th>Chamber size</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.B.</td>
<td>69 m</td>
<td>2 mo.</td>
<td>0</td>
<td>10 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal systolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>G.B.</td>
<td>68 f</td>
<td>7 yr.</td>
<td>1 wk.</td>
<td>10 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal systolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>L.D.</td>
<td>33 m</td>
<td>18 mo.</td>
<td>4 mo.</td>
<td>20 yr.</td>
<td>0</td>
<td>IV</td>
<td>III, basal diastolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>M.G.</td>
<td>39 f</td>
<td>5 mo.</td>
<td>5 mo.</td>
<td>10 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal systolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>J.H.</td>
<td>58 m</td>
<td>4 da.</td>
<td>4 da.</td>
<td>20 yr.</td>
<td>0</td>
<td>IV</td>
<td>0</td>
<td>NSR</td>
<td>RAH</td>
<td>LVH</td>
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<tr>
<td>L.K.</td>
<td>37 f</td>
<td>3 hr.</td>
<td>3 hr.</td>
<td>10 yr.</td>
<td>0</td>
<td>IV</td>
<td>0</td>
<td>NSR</td>
<td>LVH</td>
<td></td>
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<tr>
<td>M.K.</td>
<td>55 m</td>
<td>10 yr.</td>
<td>6 mo.</td>
<td>30 yr.</td>
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<td>IV</td>
<td>II, basal systolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>C.M.</td>
<td>51 f</td>
<td>3 mo.</td>
<td>0</td>
<td>25 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal systolic</td>
<td>AF</td>
<td>LVH</td>
<td></td>
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<tr>
<td>M.O.B.</td>
<td>23 f</td>
<td>1 mo.</td>
<td>1 mo.</td>
<td>10 yr.</td>
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<td>IV</td>
<td>III, apical diastolic</td>
<td>NSR</td>
<td>RVH</td>
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<tr>
<td>F.A.</td>
<td>71 m</td>
<td>7 yr.</td>
<td>0</td>
<td>7 yr.</td>
<td>Yes</td>
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<td>0</td>
<td>AF</td>
<td>LVH</td>
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<tr>
<td>L.A.</td>
<td>71 m</td>
<td>4 yr.</td>
<td>2 mo.</td>
<td>4 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal diastolic</td>
<td>NSR</td>
<td>LVH</td>
<td></td>
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<tr>
<td>R.D.</td>
<td>45 m</td>
<td>15 yr.</td>
<td>0</td>
<td>15 yr.</td>
<td>0</td>
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<td>NSR</td>
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<td></td>
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<tr>
<td>S.K.</td>
<td>58 m</td>
<td>10 mo.</td>
<td>0</td>
<td>0 yr.</td>
<td>0</td>
<td>III</td>
<td>II, basal systolic</td>
<td>NSR</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>H.S.</td>
<td>60 m</td>
<td>6 mo.</td>
<td>6 mo.</td>
<td>0 yr.</td>
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<td>IV</td>
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<td>NSR</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>S.S.</td>
<td>60 m</td>
<td>2 mo.</td>
<td>1 mo.</td>
<td>0 yr.</td>
<td>0</td>
<td>IV</td>
<td>II, basal systolic</td>
<td>NSR</td>
<td>?LVH</td>
<td></td>
</tr>
</tbody>
</table>

**KEY:** Murmurs graded I to VI; AF, atrial fibrillation; NSR, normal sinus rhythm; RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy; RAH, right atrial hypertrophy; scale of + to +++ employed for grading left atrial size by x-ray; m, male; f, female.
## Ruptured Chordae Tendineae

<table>
<thead>
<tr>
<th>Chest x-ray</th>
<th>Left atrium</th>
<th>Operation for valve disease</th>
<th>Major cause of death</th>
<th>Anatomic findings</th>
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<tr>
<td>++</td>
<td>I.VH</td>
<td>0</td>
<td>0</td>
<td>Bacterial endocarditis; syphilitic aortic and valvulitis; bacterial endocarditis, aortic valve; perforation, aortic and mitral valves; ruptured chordae tendineae, aortic leaflet mitral valve</td>
</tr>
<tr>
<td>++</td>
<td>LVH</td>
<td>0</td>
<td>0</td>
<td>Ruptured chordae tendineae; rheumatic aortic and mitral valvulitis; healed bacterial endocarditis, mitral valve; ruptured chordae tendineae, aortic leaflet mitral valve</td>
</tr>
<tr>
<td>++</td>
<td>LVH</td>
<td>0</td>
<td>Yes</td>
<td>Operation; rheumatic aortic and mitral valvulitis; healed bacterial endocarditis, mitral valve; ruptured chordae tendineae, aortic leaflet mitral valve</td>
</tr>
<tr>
<td>+++</td>
<td>LVH</td>
<td>0</td>
<td>Yes</td>
<td>Operation; rheumatic aortic and mitral valvulitis; ruptured chordae tendineae, aortic leaflet mitral valve</td>
</tr>
<tr>
<td>+++</td>
<td>RVH</td>
<td>0</td>
<td>0</td>
<td>Operation (suspected pulmonary embolus); rheumatic mitral valvulitis; ruptured chordae tendineae, aortic leaflet mitral valve; nonbacterial endocarditis, mitral valve</td>
</tr>
<tr>
<td>++</td>
<td>LVH</td>
<td>0</td>
<td>0</td>
<td>Myocardial infarction; healed bacterial endocarditis, mitral valve; ruptured chordae tendineae, aortic leaflet mitral valve; embolus, left anterior descending coronary artery with myocardial infarction</td>
</tr>
<tr>
<td>+++</td>
<td>LVH</td>
<td>0</td>
<td>Yes</td>
<td>—; Ruptured chordae tendineae, mural cusp mitral valve</td>
</tr>
<tr>
<td>++</td>
<td>RVH</td>
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<td>0</td>
<td>Myocardial infarction; rheumatic mitral valvulitis; active bacterial endocarditis, mitral valve; embolus, left anterior descending coronary artery with extensive infarction; ruptured chordae tendineae, aortic and mural leaflets mitral valve</td>
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<tr>
<td>++</td>
<td>RVH</td>
<td>0</td>
<td>0</td>
<td>Ruptured chordae tendineae; rheumatic aortic and mitral valvulitis; fenestration of aortic and pulmonary valves; ruptured chordae tendineae, aortic leaflet mitral valve; widely patent foramen ovale; Ehlers-Danlos syndrome; dwarfism; healed bacterial endocarditis, mitral and aortic valves</td>
</tr>
<tr>
<td>++</td>
<td>LVH</td>
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<td>0</td>
<td>Ruptured chordae tendineae, mural cusp of mitral valve</td>
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<tr>
<td>+</td>
<td>LVH</td>
<td>Yes</td>
<td>Yes</td>
<td>Operation; no autopsy; normal appearing mitral valve with ruptured chordae tendineae, aortic leaflet mitral valve</td>
</tr>
<tr>
<td>++</td>
<td>LVH</td>
<td>Yes</td>
<td>Yes</td>
<td>—; Ruptured chordae tendineae, mural cusp of normal mitral valve; atrial septal defect; achondroplasia</td>
</tr>
<tr>
<td>+</td>
<td>LVH</td>
<td>Yes</td>
<td>Yes</td>
<td>Operation; Ruptured chordae tendineae, mural leaflet of normal mitral valve</td>
</tr>
<tr>
<td>+++</td>
<td>LVH</td>
<td>Yes</td>
<td>Yes</td>
<td>—; Ruptured chordae tendineae, mural leaflet of normal-appearing mitral valve</td>
</tr>
<tr>
<td>+</td>
<td>?LVH</td>
<td>Yes</td>
<td>Yes</td>
<td>Operation; Ruptured chordae tendineae, mural leaflet of normal mitral valve</td>
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</table>

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Cardiac Catheterization

Results are summarized in table 2.

Six patients (five “spontaneous,” one rheumatic) were studied by combined right and left heart catheterization. Strikingly prominent “v” waves (45 mm. Hg or greater) were seen in all patients, resembling small ventricular complexes in five (fig. 2). In the sixth patient, a good phasic representation of the left atrial pressure curve could not be obtained in the pulmonary “wedge” position with the right heart catheter. No end-diastolic pressure gradient across the mitral valve was noted in any patient. Left ventricular failure was present in five cases, and the pulmonary arteriolar resistance was elevated in four. All had a reduction in resting cardiac index. Left ventricular angiography was performed in four patients, uniformly showing severe regurgitation of contrast material into the left atrium. Systolic expansion of this chamber could be easily detected in three patients without gross left atrial enlargement. The left ventricular cavity was estimated to be enlarged in each patient without evidence of hypertrophy of the free left ventricular wall. Repetitive ventricular tachycardia precluded angiographic evaluation in two patients (L.A., M.K.).

Clinical Impression

In five patients (S.S., H.S., L.A., R.D., S.K.) undergoing operation, the diagnosis of ruptured chordae tendineae was strongly suspected preoperatively. This was due primarily to the sudden onset of severe symptoms, the abrupt appearance of a murmur, the small left atrium seen by cardiac fluoroscopy and angiography, or a combination thereof. The radiation of the murmur to the base simulating the murmur of aortic stenosis suggested involvement of the mural cusp in four of these patients (S.S., H.S., R.D., S.K.). In the remaining three patients of the operated group (M.K., M.G., L.D.), the long history of documented rheumatic heart disease seemed to afford sufficient explanation for their symptoms. The diagnosis was suspected in only one of the seven unoperated patients on the basis of the sudden increase in the

Table 2

<table>
<thead>
<tr>
<th>Radial artery</th>
<th>RV</th>
<th>PA</th>
<th>Cardiac index</th>
<th>PAR</th>
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<tbody>
<tr>
<td>Pa</td>
<td>45/0.5</td>
<td>45/0.11</td>
<td>180/60</td>
<td>100/0.14</td>
</tr>
<tr>
<td>M.K.</td>
<td>45/0.5</td>
<td>45/0.11</td>
<td>180/60</td>
<td>100/0.14</td>
</tr>
<tr>
<td>S.S.</td>
<td>45/0.5</td>
<td>45/0.11</td>
<td>180/60</td>
<td>100/0.14</td>
</tr>
<tr>
<td>H.S.</td>
<td>45/0.5</td>
<td>45/0.11</td>
<td>180/60</td>
<td>100/0.14</td>
</tr>
</tbody>
</table>

*All pressures in millimeters of mercury. L.A. left atrium; PC pulmonary capillary “wedge” pressure; LV left ventricle; PA pulmonary artery; RV right ventricle; RA right atrium; cardiac index, L/min./m2; PAR pulmonary arteriolar resistance in units; m. mean.

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intensity of the apical systolic murmur with appearance of severe left ventricular failure. One patient in this group was thought to have massive pulmonary embolism. Since the patient appeared terminal, the pulmonary artery was explored without the finding of an embolus; the patient failed to survive the procedure. The remaining patients had a long history of heart disease which, alone or because of association with some other process, did not alert the clinician to the correct diagnosis.

Findings and Results of Operation

Four of the eight patients undergoing operation showed rupture of the chordae tendineae anchoring the aortic leaflet of the mitral valve; the remaining patients had rupture of the chordae supporting the mural cusp. Surgical treatment consisted of placement of numerous artificial chordae (3-0 Mersilene) in seven patients and valve replacement with a Starr-Edwards prosthesis in one. Five patients failed to survive operation or the immediate postoperative period. In four of these the presence of a significant degree of aortic regurgitation necessitated intermittent cross-clamping of the aorta; none could be resuscitated following surgery. The fifth patient first had a bicuspidization procedure for his aortic incompetence; approaching his lesions in this sequence allowed repair of the mitral valve in a reasonably dry field. Postoperatively, the patient did well for 12 hours but then developed progressive, intractable pulmonary edema culminating in death 4 days later.

After resuscitation in the three surviving patients (H.S., M.K., R.D.), the mitral valve was judged to be competent in all by palpation. Postoperatively these patients did well. The apical systolic murmur disappeared and cardiac compensation improved, although slowly in patient M.K.

The most satisfying result to date has been in patient H.S. (placement of artificial chordae) who is working full time as a machinist, completely free from symptoms 3 years after operation. He still has a faint grade-I apical systolic murmur, but his heart has returned to normal size radiographically (fig. 3), and left ventricular hypertrophy has disappeared from his electrocardiogram. Postoperative catheter and angiographic studies have shown marked diminution in the height of the "v" wave from 75 to 19 mm. Hg, a significant de-

Figure 2

Patient L.A. Simultaneous pulmonary capillary "wedge" and left ventricular end-diastolic pressure tracings. There is a slight delay in inscription of wedge pressure which represents the transmission time from left atrium to the right heart catheter tip. Strikingly prominent "v" waves are seen without end-diastolic gradient across the mitral valve. Left ventricular failure is present.

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crease in pulmonary arteriolar resistance, and improvement in resting cardiac index (fig. 4). Only a very small amount of mitral regurgitation was present angiographically, and the left atrium was reduced to almost normal size (fig. 5). Patient M.K. (placement of artificial chordae) improved slowly for 6 weeks after operation but then felt something “give” in his chest. Mild congestive heart failure recurred with a grade-II apical systolic murmur. He has been maintained on a strict cardiac program in the year since operation, and, although still symptomatic, is considerably improved. Patient R.D. (Starr-Edwards valve prosthesis) is asymptomatic 5 months after valve replacement. He is being maintained on anticoagulants, moderate sodium restriction, and digitalis.

Postmortem Examination in Unoperated Patients

Of the seven patients in this group, rupture of the chordae tendineae may be ascribed a major cause of death in four. Active bacterial endocarditis was the primary cause of death in two other instances, producing rupture of the aortic and mitral valves in one patient and myocardial infarction from coronary embolism.

![Figure 3](image)

**Figure 3**

Patient H.S. Preoperative chest film on left shows cardiac enlargement particularly in the region of the left ventricle. Film on right taken 7 months postoperatively shows return of the heart to normal size.

![Figure 4](image)

**Figure 4**

Patient H.S. Left atrial pressures before and after operation demonstrating marked diminution in height of “v” wave with return of mean pressure to normal following operation.
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in the other. The final patient died abruptly following an acute myocardial infarction from a coronary embolus arising from healed vegetations of bacterial endocarditis treated 20 months previously.

Six of these seven patients had evidence of healed rheumatic valvulitis. In addition to rheumatic involvement, the patient with Ehlers-Danlos syndrome and dwarfism had multiple intracardiac defects. These included a patent foramen ovale, fenestration of aortic and pulmonary valves, and healed bacterial endocarditis of the aortic and mitral valves. In one patient (F.A.) no other disease was associated with the ruptured chordae.

Discussion

Severe mitral regurgitation was associated with rupture of chordae tendineae in all 15 patients of the present series and was a major contributor to death in nine of the 12 patients who died. Active bacterial endocarditis, the most common cause of rupture in the pre-antibiotic era, was present in only two of the nine patients with rheumatic mitral valvulitis, although four of these showed evidence of healed endocarditis and one had nonbacterial endocarditis. The high incidence of endocarditis, either active or healed, suggests that this disease and its sequelae continue to be of major etiologic importance. The role of rheumatic valvulitis appears to be permissive by providing a nidus for infection although rheumatic scarring is probably of primary importance in the occasional patient. Ehlers-Danlos syndrome, a disease with alterations in collagen formation and various congenital defects, has been causally related to valvular heart disease in at least one instance.\textsuperscript{17, 18} In our patient with this disease, the presence of rheumatic mitral valvulitis and healed endocarditis provides adequate explanation for rupture without invoking another etiology. A surprisingly large number of patients, six, had no demonstrable etiology.

The most helpful feature of the clinical history was the sudden onset of cardiac decompensation or an abrupt worsening of previous symptoms, particularly when these proved resistant to a strict cardiac program. The sudden onset of a loud apical systolic murmur is probably the most reliable sign of chordae rupture but this could be documented in only three of our patients. The apical murmur was remarkably harsh in quality and radiated to the left axilla and lung base in the patients with involvement of the aortic leaflet. Those patients with a flail mural cusp uniformly showed transmission of the murmur to the base (but not to the neck) with disappearance of the late systolic component, thereby suggesting associated aortic stenosis. Such “pseudoaortic stenosis” has been attributed to the superomedial direction of the regurgitant

\textit{Figure 5}

\textit{Patient H.S. Transseptal left ventriculography showing massive mitral regurgitation preoperatively with enlarged left atrial and left ventricular cavities. Postoperatively no significant reflux can be seen through the mitral valve; the left ventricular cavity is normal in size and contracts well.}
jet through the mural cusp region by Os- 
mundson, Callahan, and Edwards although a 
recent report suggests that similar ausculta-
tory findings may result from involvement of 
the aortic leaflet as well. An extremely help-
ful diagnostic feature in 

four patients was the finding of a small left 
atrium with systolic expansion fluorosco-

copically in the presence of obviously severe mitral 
regurgitation. Menges, Ankeney, and Heller-
stein have emphasized the presence of gross 
left atrial enlargement in their series of pa-

tients with ruptured chordae tendineae. The 
discrepancy between their cases and our four 
patients may be explained in large measure by 
the duration of the valvular incompetence. 
All of their patients had symptoms and signs 
for at least a year; similar patients of ours 
also showed impressive left atrial enlarge-
ment. The patients whose mitral regurgitation 
had been present for only a few months had 
relatively small left atria with the exception of 
patient H.S. who developed considerable 
left atrial enlargement over a 6-month pe-


riod. We would therefore suggest that the 
duration of severe mitral regurgitation is an 
important determinant in the production of 
left atrial enlargement and that the degree of 
such enlargement is most helpful diagnosti-

cally when a relatively small left atrium is as-


associated with gross mitral regurgitation.

Cardiac catheterization was of greatest di-
agnostic aid when combined with left ven-
tricular angiography, which allowed assess-
ment of degree and location of reflux, left 
atrial size, and presence or absence of left 
atrial systolic expansion. The demonstra-


tion of a small left atrium in the presence of severe 
regurgitation strongly suggested the correct 
diagnosis in three of the four patients in 
whom it could be done. All patients showed 
striking “v” waves in the left atrial pressure 
record without an end-diastolic gradient 
across the mitral valve, a finding consistent 
with severe, pure mitral regurgitation. Car-
diac catheterization was particularly useful in 
those four patients whose physical findings 
suggested associated aortic stenosis. Since the 
surgical approach to combined aortic and mi-
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in the initial postoperative period had varying degrees of aortic regurgitation requiring cross-clamping of the aorta for long periods to obtain a dry operative field. The resulting anoxia may well have produced irretrievable damage in an already severely stressed myocardium despite apparently adequate mitral valve repair.

Summary

Fifteen cases of severe mitral regurgitation secondary to ruptured chordae tendineae have been reviewed, eight of whom underwent open-heart surgery to correct their lesions. Three patients are living at this time with two being markedly improved. Cardiac catheterization was performed in six prior to surgery.

Progressive deterioration within the year following the onset of difficulty or sudden worsening of previous cardiac symptoms characterized the usual clinical course. The apical systolic murmur, always loud, was conspicuously harsh and radiated to the base of the heart, simulating aortic stenosis when the mural cusp was ruptured. Although strongly suggesting the diagnosis, the sudden onset of an apical systolic murmur could be documented in only three instances. The presence of a small left atrium radiographically with tall left atrial “v” waves and marked reflux of contrast material into a paradoxically pulsating left atrium also pointed to the correct diagnosis.

Various technics of surgical repair and problems influencing survival of patients in the present series are discussed.

Acknowledgment

We wish to express our thanks to Dr. Allan L. Friedlich and Dr. Guillermo C. Sanchez for allowing us to include their patients in this report.

Addendum

Since this article was submitted for publication, five additional cases with ruptured mitral chordae tendineae have been encountered. The diagnosis was made preoperatively in three patients on the basis of demonstrated severe mitral regurgitation into a small left atrium. In four patients the aortic leaflet of the mitral valve was flail; each one of the cases had severe aortic regurgitation. The fifth patient had involvement of the mural cusp. All underwent valve replacement with a Starr-Edward prostheses. Three patients are markedly improved 2 to 6 months following surgery. A fourth patient has shown only modest improvement after insertion of two prosthetic valves. One patient died 4 weeks after operation secondary to rupture of his mitral ring.

References

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Famous General Practitioners—Robert Koch

Robert Koch, born in 1843, was one of thirteen children of a mining engineer. He studied medicine at Göttingen where he came under the influence of Henle, who taught that: “before microscopic forms can be regarded as the cause of contagion in man they must be found constantly in the contagious material, they must be isolated from it and their strength tested.” Henle little knew what fruit his words would bring forth by the aid of one of his listeners.

Koch qualified in 1866 at the age of twenty-three. He was very ambitious but he wished to marry, so he took a post as medical officer to a hospital for defective children at Langenhagen where he also did some general practice. Two years later he began practice at Niemagk and a year later went on to Rakwitz. . . . In 1872 he passed the examination for the State Medical Service and was posted as district medical officer in a remote little town in Wollstein called Bomst, where he was also allowed to do private general practice. Though he had little time to himself he spent that little in the investigation of splenic fever (anthrax) which was very prevalent among the sheep and cattle in that district. Koch knew that Pollender had seen minute living forms in the blood of cattle suffering from splenic fever, and he was aware that Davaine had transmitted the disease by injecting the blood of a diseased animal into the body of another animal, even when none of the microscopic forms had been observed in the blood which was injected, and he strove to reconcile these apparently contradictory facts. He possessed a microscope (said to have been given him by his devoted wife) and he began to observe and experiment. He found a way to cultivating anthrax bacillus outside the body, detected the spores, grew the bacillus from the spores, watched their growth in hang-drop preparations, proving by experimental demonstration that the anthrax bacillus was the cause of splenic fever. In 1876 he wrote to Professor Ferdinand Cohn (of Breslau University) asking for permission to demonstrate to him the experiments which had led to this discovery. The demonstration was successful and conclusive, and Cohn was the first to realize and to assert that a new era of microbiology had begun and that a new scientific star had appeared. . . . This most difficult initial stage of this research was carried out with the most discouraging conditions, far away from any recognized medical centre, by a man who was at the same time carrying on a busy general practice.—ZACHARY COPE, KT. Some Famous General Practitioners and other Medical Historical Essays. London, Pitman Medical Publishing Co., Ltd., 1961, p. 19.
Severe Mitral Regurgitation Secondary to Ruptured Chordae Tendineae
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