Ruptured Mitral Chordae Tendineae

By PHILIP J. OSMUNDSON, M.D., JOHN A. CALLAHAN, M.D., and JESSE E. EDWARDS, M.D.

Among the lesions responsible for insufficiency of the mitral valve is rupture of the chordae tendineae. It is our purpose to describe the significant clinical and pathologic findings in 20 cases of insufficiency of the mitral valve due to rupture of the chordae tendineae. This report is based on those cases in which ruptured mitral chordae tendineae were found at necropsy at the Mayo Clinic during the years 1934 to 1958 inclusive.

The important etiologic factors for rupture of the mitral chordae tendineae are bacterial endocarditis,1-5 rheumatic valvular disease,6 7 and trauma.8 9 Cases have been encountered, however, in which conclusive evidence of these conditions has not been demonstrated. In those cases in which evidence for a specific etiologic factor is lacking, so-called spontaneous rupture10,11 must be considered. Though myocardial infarction is the prominent cause of mitral insufficiency due to ruptured papillary muscles,12 13 it has not been demonstrated to be of importance in rupture of the chordae tendineae.

Etiologic Factors of Ruptured Mitral Chordae Tendineae

Bacterial Endocarditis

Bacterial endocarditis is the major factor in the etiology of ruptured chordae tendineae. In the first case of ruptured mitral chordae tendineae reported in the medical literature (Corvisart 1812),14 necropsy findings were recognizable in retrospect as due to bacterial endocarditis. The association between bacterial endocarditis and ruptured mitral chordae tendineae was well demonstrated in reports by Clark15 and by Washbourn1 late in the nineteenth century. Ruptured chordae tendineae have been identified in cases of healed, sometimes clinically unrecognized, bacterial endocarditis3 as well as in cases of active bacterial endocarditis.

The present study includes 20 cases of ruptured mitral chordae tendineae. In 10 cases there was a history of bacterial endocarditis, active in six cases and healed in four (table 1).

Known Active Bacterial Endocarditis

The microorganisms cultured from the blood of the six patients in this group were Streptococcus mitis in two cases, Streptococcus faecalis alone in one case and associated with Aerobacter aerogenes in another case, and Escherichia coli in one case. The blood cultures were repeatedly negative in the sixth case, although active bacterial endocarditis was demonstrated at necropsy. The report of a representative case (case 4) follows.

A 37-year-old woman entered the hospital in February 1953. She had been treated first for bacterial endocarditis caused by Str. faecalis 18 months before this admission when weakness, anorexia, and persistent fever had developed. Three months after the onset she had hemiparesis on the right side. Six months later a mycotic aneurysm was excised from the right forearm, and intermittent fever persisted despite continued treatment with antibiotics. Six days before her hospitalization, diplopia and left frontal headache had developed.

On examination a high-pitched systolic murmur of moderate intensity was heard over the precordium which was maximal near the apex of the heart and transmitted posteriorly and to the left. The fingers were clubbed and the liver and spleen were palpable. Str. faecalis was cultured from the patient's blood. Treatment with penicillin combined with dihydrostreptomycin was instituted. Suddenly the patient became comatose on her twenty-fourth day in the hospital and died 48 hours later.

At necropsy the heart was found to be moder-
RUPTURED MITRAL CHORDAE TENDINEAE

Examination of the patient in October 1947, 10 months after the initial hospitalization, did not show evidence of active bacterial endocarditis. The patient's cardiac reserve was considered to be good. A loud systolic murmur and an early diastolic murmur were heard.

Later, in September 1950, when the patient was again hospitalized, he gave a history of progressive cough, dyspnea, and dependent edema of 5 months' duration. Atrial fibrillation was present. An apical thrill was noted as well as a harsh apical systolic murmur that was transmitted to the left axilla and posterior part of the thorax on the same side. The diastolic murmur was heard again. A systolic murmur was heard at the base of the heart also. Findings typical of congestive heart failure were present. The patient's condition deteriorated despite treatment and he died on the eleventh day of hospitalization.

At necropsy the heart was found to be enlarged and weighed 600 Gm. The left ventricle and the left atrium were particularly dilated. Both ventricles were hypertrophied. The anterior leaflet of the mitral valve was distorted in the portion adjacent to the posteromedial commissure. Approximately a fourth of the leaflet in this region was devoid of intact chordae tendineae (fig. 2). The leaflet was moderately thickened and arched up into the left atrial cavity. It appeared that there had been a loss of substance of this part of the leaflet. Several of the intact chordae tendineae nearby were slightly thickened. A thickened mural plaque measuring 1.6 cm. in diameter was found on the ventricular wall under the posterior mitral leaflet. This had the appearance of healed mural endocarditis. A jet lesion was found in the left atrium above and posterior to the posteromedial commissure. The other heart valves did not appear to be abnormal.

Histologically, one of the ruptured chordae tendineae had an enlarged fragmented end with calcified and necrotic debris adjacent to the tip. The thickened anterior mitral leaflet was composed of fibrous and collagenous tissue. The plaque-like structure on the endocardium of the left ventricular wall was composed of thickened endocardium with foci of calcified debris. The adjacent fibrous tissue was vascularized.

Mitrval insufficiency occurred as a complication of bacterial endocarditis in this case and caused cardiac decompensation that resulted in death. Mitrval insufficiency resulted from rupture of the mitral chordae tendineae attached to a portion of the anterior mitral leaflet. Erosion of a portion of the mitral leaflet was an additional complication of the bacterial endocarditis.

A 42-year-old man was first admitted to the hospital in January 1947 with a history of anorexia, fatigue, and night sweats of 4 months' duration. One month prior to his admission a heart murmur was discovered for the first time. Examination disclosed fever, clubbing of the fingers, and generalized petechiae of the skin. A systolic thrill was noted at the apex of the heart. A loud, rough systolic murmur was heard at the apex and transmitted widely, particularly to the left axilla. A softer systolic murmur was heard at the base of the heart. Cultures of the patient's blood yielded Streptococcus mitis. The day after admission, hemiparesis on the left and complete left homonymous hemianopsia developed. Treatment with penicillin was begun and the response was satisfactory.

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### Table 1

**Summary of Twenty Cases of Ruptured Mitral Chordae Tendineae**

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex: age at death, yr.</th>
<th>Status of bacterial endocarditis at necropsy</th>
<th>Bacterial endocarditis</th>
<th>Contrib. of ruptured chordae to cardiac dysfunction</th>
<th>Cause of death: cardiac (C) or noncardiac (N)</th>
<th>Number and location of ruptured chordae</th>
<th>Location of jet lesions in left atrium</th>
<th>Transm. of apical aortic murmur</th>
<th>Other abnormalities of mitral valve</th>
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<tr>
<td>1</td>
<td>F 15</td>
<td>Active</td>
<td>Str. mitis</td>
<td>Mitrval valve</td>
<td>Present Contrib.</td>
<td>C</td>
<td>2 Ant. leaflet</td>
<td>Postero-lateral wall</td>
<td>Left axila; basal precordium</td>
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<tr>
<td>2</td>
<td>M 62</td>
<td>Active</td>
<td>Str. faecalis and A. aerogenes</td>
<td>Mitrval valve</td>
<td>—</td>
<td>Contrib.</td>
<td>C 2 Post. leaflet</td>
<td>Anterior septal wall</td>
<td>Left axilla</td>
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<td>3</td>
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<td>E. coli</td>
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<td>—</td>
<td>Contrib.</td>
<td>C 8 Ant. leaflet</td>
<td>Postero-lateral wall</td>
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<tr>
<td>4</td>
<td>F 37</td>
<td>Active</td>
<td>Str. faecalis</td>
<td>Mitrval valve</td>
<td>Probable Contrib.</td>
<td>N Cerebral embolus</td>
<td>2 Ant. leaflet</td>
<td>Lateral wall</td>
<td>Post. left chest</td>
</tr>
<tr>
<td>5</td>
<td>M 62</td>
<td>Active</td>
<td>Negative blood cultures</td>
<td>Mitrval valve</td>
<td>—</td>
<td>Contrib.</td>
<td>N Cerebral embolus</td>
<td>Anterior wall</td>
<td>Not recorded</td>
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<td>6</td>
<td>M 64</td>
<td>Active</td>
<td>Str. mitis</td>
<td>Aortic valve</td>
<td>Probable Contrib.</td>
<td>C 4 Ant. leaflet</td>
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<td>Posterior wall</td>
<td>Left axilla</td>
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<tr>
<td>7</td>
<td>M 46</td>
<td>Healed</td>
<td>Str. mitis</td>
<td>Mitrval valve</td>
<td>—</td>
<td>Primary</td>
<td>C 15 of ant. leaflet</td>
<td>Posterior wall</td>
<td>Left axila and post. left chest; basal precordium</td>
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<tr>
<td>8</td>
<td>M 65</td>
<td>Healed</td>
<td>Str. (anaerobic) by history</td>
<td>Mitrval valve</td>
<td>Present Contrib.</td>
<td>C 3 Ant. leaflet</td>
<td>None identified</td>
<td>Left axilla</td>
<td>Healed vegetation with distortion of ant. leaflet; thickened chordae</td>
</tr>
<tr>
<td>9</td>
<td>M 26</td>
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<td>Str. (type unknown) by history</td>
<td>Aortic valve</td>
<td>Present Contrib.</td>
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<td>3 Ant. leaflet</td>
<td>Postero-lateral wall</td>
<td>Left axila; post. left chest</td>
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<td>10</td>
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<td>Str. faecalis</td>
<td>Mitrval valve</td>
<td>Present Non-contrib.</td>
<td>N Atherosclerotic occlusion of basilar artery</td>
<td>1 Post. leaflet</td>
<td>Septal wall, ant. leaflet</td>
<td>Left axila; basal precordium</td>
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<td>Case</td>
<td>Sex</td>
<td>Diagnosis</td>
<td>Cause of Mitral Valve Damage</td>
<td>Contrib.</td>
<td>Carcinoma of Colon</td>
<td>1.5 cm. of Tissue at Anterolateral Commisuro without Intact Chordae</td>
<td>None Identified</td>
<td>Not Recorded</td>
<td>Arching of Commisural Tissue into Left Atrium</td>
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<td>Mitral Valve</td>
<td>Present</td>
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<td>1/4 of Post. Leaflet without Intact Chordae</td>
<td>Septal Wall</td>
<td>Basal Precordium</td>
<td>Large Calcified Vegetation, Post. Leaflet</td>
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*No history of bacterial endocarditis.

**Ruptured Mitral Chordae Tendineae**
No History of Bacterial Endocarditis

Pathologic Evidence of Healed Bacterial Endocarditis. Ten of the 20 patients in this study did not have a history of bacterial endocarditis. Necropsy disclosed evidence considered positive for healed bacterial endocarditis in three of the 10 cases, while in an additional three cases the cardiac abnormalities, in addition to the ruptured chordae tendineae, were suggestive though not conclusive evidence of healed bacterial endocarditis. The report of a representative case (case 13) follows.

A 49-year-old man entered the hospital in May 1957. He did not have a history of rheumatic fever or any significant febrile illness. The presence of a heart murmur had been known only since the onset of the patient's symptoms 8 months before his last admission. Exertional dyspnea and dependent edema developed 8 months prior to his admission to the hospital. Orthopnea and paroxysmal nocturnal dyspnea appeared 10 days before his admission.

Physical examination showed the heart to be enlarged. A slight apical thrill was detected. A loud systolic murmur was present over the entire precordium. This was loudest in the fourth intercostal space near the midclavicular line and was transmitted posteriorly and to the left. Rales were present in the lungs; the liver was enlarged to 4 cm. below the right costal margin, and considerable pretilial edema was present. A roentgenogram of the thorax disclosed cardiac enlargement with pulmonary vascular congestion. An electrocardiogram showed a sinus rhythm with multiple premature contractions and nodal escape beats. A subsequent electrocardiogram made 2 days later showed variation between a sinus rhythm and an idioventricular rhythm. The patient did not respond despite intensive treatment for congestive heart failure and died on the fourth hospital day.

At necropsy the heart was found to be moderately enlarged and weighed 430 Gm. All chambers were dilated and the left ventricle particularly was hypertrophied. The mitral valve was the site of considerable abnormality. The posterior mitral leaflet was lacking in substance along its free edge, apparently from an old ulcerative process. The posterior leaflet projected up into the left atrial cavity. Approximately two thirds of the posterior mitral leaflet was without intact chordae tendineae. Remnants of the ruptured chordae were folded under the deformed posterior leaflet and their ends were round and smooth. The anterior mitral leaflet near the anterolateral commissure also appeared to be altered by an ulcerative process, which later healed. A small projection from the posteromedial papillary muscle represented the origin of the ruptured chordae tendineae. The intact chordae tendineae appeared normal. A jet lesion measuring approximately 2 by 3 cm. was present on the septal wall of the left atrium beginning 1 cm. above the anterior mitral leaflet. Histologically the thickened, deformed posterior mitral leaflet was composed of irregular masses of collagenous and fibrous tissue.

This case demonstrates the finding of ruptured chordae tendineae in a patient who did not have a history of bacterial endocarditis but at necropsy definite evidence of healed bacterial endocarditis was found. Mitral insufficiency resulted from rupture of the chordae tendineae and led to the death of the patient.

Without Pathologic Evidence of Healed Bacterial Endocarditis. The cause of ruptured chordae tendineae in the other four cases was not determined. At necropsy evidence of an inflammatory process which had healed prior to the death of the patient was present in each of the four cases, but the lesions were nonspecific and did not allow a precise identification of their causes. The following case is an example (case 16*).

A 23-year-old man was examined in November 1955 for evaluation of a heart murmur first discovered 7 years previously. There was no history of rheumatic fever or trauma to the thorax. Examination of the heart in 1944 gave essentially negative results. In December 1948 an illness began that lasted 6 weeks and was characterized by fever, lassitude, and weakness. The patient was hospitalized during this illness and the roentgenogram of the thorax disclosed inflammation in one lung. The heart murmur was heard first at this time. Exertional dyspnea, severe enough to prevent participation in vigorous activities, was present from the time of this illness. Gradual diminution in the patient's tolerance for exercise developed during the next 5 years.

On first examination at the Mayo Clinic in October 1954, loud systolic murmurs accompanied by thrills were heard over both the aortic and apical areas. Little symptomatic change took place after this visit until 1 month prior to registration in November 1955. At this time the patient experienced dyspnea with minimal exertion, paroxysmal nocturnal dyspnea, vague epigastic discomfort,

*This case formed the basis of a previous publication.
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Figure 1

Ruptured chordae tendineae with remnants of ruptured chordae attached to anterior mitral leaflet. Active endocarditis was caused by Streptococcus faecalis. The chordae, involved by vegetations and rupture, are in proximity to areas of infectious activity on the leaflet of the valve (case 4).

and hemoptyis. Atrial fibrillation was present with an apical rate of 92. A coarse systolic murmur of moderate intensity was present at the second and third intercostal spaces to the right of the sternum and was accompanied by a thrill. This murmur was heard in the neck over the carotid arteries. The second sound in the aortic area was diminished and the second sound in the pulmonic area was accentuated. A loud prolonged systolic murmur of coarse quality, also accompanied by a thrill, was heard at the apex, and was transmitted posteriorly and to the left. A faint early apical diastolic murmur was present. The liver was palpably enlarged.

A roentgenogram of the thorax showed great cardiac enlargement with particular increase in size of the left atrium. The electrocardiograms in 1954 and 1955 were similar and showed changes consistent with left ventricular hypertrophy. The tracings in 1955 also showed atrial fibrillation and ventricular premature contractions.

On the basis of the physical examination and clinical tests, the patient was considered to have aortic stenosis and mitral insufficiency. Catheterization of the right and left sides of the heart was performed. The findings demonstrated mitral insufficiency without aortic stenosis.

An attempt at surgical amelioration of mitral insufficiency was undertaken. At the time of operation the heart was found to be tremendously enlarged, with a huge left atrium. Severe regurgitation of blood through the mitral valve was detectable at the time of digital exploration of the left atrium. An attempt was made to place a circumferential suture around the mitral valve ring in order to reduce the size of the orifice, but this was unsuccessful because of the tremendous dilatation of the mitral ring. The mitral insufficiency was not relieved and the patient died 3 days later.

At necropsy the heart was tremendously enlarged and weighed 1,000 Gm. All chambers were dilated, particularly the left atrium and the left ventricle. Approximately a third of the posterior leaflet lateral to its midpoint was devoid of intact chordae tendineae (fig. 3). The involved part of the leaflet arched into the left atrium giving it a hooded appearance. Seven chordae tendineae arising from the posterior leaflet were discontinuous from their origins at the posteromedial papillary muscle where only one rounded stump remained. The free ends of the chordae appeared irregular and blunt and were hanging free in the ventricular cavity. The leaflets of the mitral valve were intact and slightly thickened in their entirety. The posterior leaflet showed greater thickness near its base. The other valves were normal. A jet lesion was present in the septal wall of the left atrium involving that portion closely approximated to the aortic valve.

Histologically the free ends of the ruptured chordae tendineae had irregular roughened surfaces covered by intact endocardium. The thickening in the posterior mitral leaflet was composed of fibrous tissue that was abundantly vascularized along the atrial surface.

The cause of the ruptured mitral chordae tendineae in this case was not definitely.
known. The evidence, although not absolutely conclusive, suggested that bacterial endocarditis had been present. The lesion causing thickening of the base of the posterior mitral leaflet was consistent with a healed vegetation of bacterial endocarditis. The history of the onset of the murmur during a febrile illness further supported the opinion that the ruptured chordae tendineae were a complication of bacterial endocarditis that healed later.

The report of another representative case (case 19) follows.

A 52-year-old farmer was admitted to a hospital in November 1948 because of dyspnea and weakness of 1 year's duration. There was no history of rheumatic fever. The illness leading to hospitalization had begun suddenly in November 1947 when the patient became short of breath after working strenuously to harvest grain. He was unable to work after the onset of this illness and subsequently orthopnea and pedal edema developed accompanied by loss of weight.

Physical examination at the time of admission to the clinic disclosed a blood pressure of 105 mm. Hg systolic and 85 mm. Hg diastolic. A loud harsh apical systolic murmur and a presystolic murmur were heard. A systolic murmur also was heard at the base of the heart with transmission to the vessels in the neck. Venous distention indicated an elevated venous pressure. Pulmonary rales, hepatomegaly, and pedal edema were present. A roentgenogram of the thorax disclosed cardiac enlargement and pulmonary edema.

The patient responded to the prescribed medical program at first, but cough, fever, and hemoptysis developed on the fifteenth hospital day. His condition deteriorated and he died on the twenty-second hospital day.

At necropsy the heart was found to be enlarged and weighed 555 Gm. The left atrium was dilated. Both ventricles were dilated and hypertrophied. The posterior mitral leaflet projected up into the left atrial cavity giving it a hooded appearance (fig. 4). Approximately four fifths of the posterior leaflet was without intact chordae tendineae. Only three chordae tendineae remained intact, and these were in the region near the posteromedial commissure. The other cardiac valves appeared normal. Portions of both the anterolateral and the posteromedial papillary muscles were atrophied. Minimal thickening of the posterior mitral leaflet was present. Neither the remnants of the ruptured chordae tendineae nor the remaining intact chordae tendineae appeared thickened or fused, or the site of vegetation. The endocardium of the septal wall of the left atrium above the anterior mitral leaflet was irregularly roughened and thickened, having the appearance of a jet lesion. A thrombus was present in the right atrial appendage.

Microscopically the ends of the ruptured chordae tendineae were covered by intact endothelium. Moderate fibrosis was present between the muscle fibers and the adjacent papillary muscle. The junction of the papillary muscle with one of the ruptured chordae tendineae was vascularized in one area near the endocardial surface. The microscopic appearance of the left atrial wall in the region of the septum supported the opinion gained from gross examination that this was a jet lesion.

Evidence of a healed inflammatory process in the heart of this patient was minimal. The lesions remaining as residuals of the inflammatory process that had healed prior to death of the patient were not specific enough to allow an etiologic diagnosis. The sudden appearance of cardiac decompensation 1 year prior to death suggested that the chordae tendineae had ruptured at that time.

Other Etiologic Factors. Rheumatic heart disease was a factor of etiologic importance in the 20 cases comprising this study primarily in predisposing to bacterial endocarditis. In each of the five cases with evidence at necropsy of quiescent rheumatic heart disease there also was conclusive evidence of superimposed bacterial endocarditis, active in one case and healed in four cases. In two additional cases necropsy findings were considered suggestive of inactive rheumatic endocarditis and valvulitis. In both cases, evidence of active bacterial endocarditis was found.

No evidence of direct trauma to the thorax of an unusual nature was found in any of the 20 cases. In all cases the findings indicated that the abnormality of the chordae tendineae was acquired and not congenital.

Pathologic Anatomy in Ruptured Mitral Chordae Tendineae

In this series of 20 cases, chordae tendineae arising from the anterolateral papillary muscle were ruptured in nine cases; chordae arising from the posteromedial muscle were ruptured in eight cases; and chordae arising from both groups were ruptured in three cases.

The site of insertion of the chordae tendineae on the valve leaflet is of importance in
RUPTURED MITRAL CHORDAE TENDINEAE

Figure 3
Ruptured chordae tendineae of the posterior mitral leaflet with arching of this portion (R) into the left atrium. An arrow demonstrates the path of the regurgitant blood from the posterior leaflet to the septal wall where a jet lesion is located. The close relationship of the jet lesion and the aortic valve (A.V.) is demonstrated. The septal wall and aortic valve in sagittal section are shown. An artifact is beneath the base of arrow (case 16).

Figure 4
Ruptured chordae tendineae of the posterior leaflet with only remnants of the chordae present on the leaflet. The hooded appearance of the posterior leaflet is demonstrated (case 19).

cases of ruptured chordae tendineae. In the present study, chordae inserting on the anterior leaflet were ruptured in nine cases, those inserting on the posterior leaflet in eight cases, those inserting on both leaflets in one case, those inserting in the region of the anterolateral commissure in one case, and those inserting in the region of the posteromedial commissure in one case.

Ruptured chordae numbered from two to nearly all those inserting on one leaflet. There was a general correlation between the number of ruptured chordae and the degree of mitral insufficiency as estimated at the time of necropsy.

Location of Ruptured Chordae Tendineae

In patients with bacterial endocarditis two important factors apparently influence the location of the ruptured chordae. In cases in which the infection involves the mitral valve it appears that the involved chordae are located adjacent to sites of most active infection. This situation prevailed in 11 of 13 cases in the present series that were considered to have definite evidence of bacterial endocarditis, active or healed, during the patient’s life or at necropsy. The mechanism that determined this location appeared to be extension of the infectious process from the leaflet to the adjacent chordae, as stated by Saphir, and by Libman and Friedberg (fig. 1).

The other important factor determining location of the ruptured chordae prevails in a special situation in which bacterial endocarditis involves the aortic valve causing aortic insufficiency. In such a case the regurgitant stream of blood often strikes the ventricular surface of the anterior mitral leaflet and the chordae attaching to the midportion of the anterior mitral leaflet. It is probable that the infectious process extends from the aortic valve to the chordae of the anterior mitral leaflet by this means, with subsequent rupture of the involved chordae. Two cases in this group of 20 demonstrated this situation. In both cases there were residual defects of the aortic cusps resulting from bacterial endocarditis (fig. 5).

Jet Lesions

Endocardial roughening and thickening characteristic of so-called jet lesions were found on the left atrial wall in 16 of the 20 cases. These lesions usually were found on the atrial wall opposite the part of the leaflet...
that was the site of the ruptured chordae. Other factors that contributed to the location of the jet lesions were abnormalities of the leaflets such as vegetations, fixed deformities resulting from old vegetations, and loss of valvular substance from ulcerative processes. The jet lesions usually were situated in the lower part of the left atrium (fig. 6).

The manner in which blood regurgitates through the mitral valve in cases of ruptured chordae tendineae can be deduced from certain anatomic findings. In cases in which the ruptured chordae had been present for some time, the involved mitral leaflet could be observed at necropsy to be arched up into the left atrial cavity leaving the leaflet fixed in a characteristic hooded appearance (fig. 4). In cases in which rupture of the chordae probably occurred shortly before death, this finding was not observed. It is postulated that during systole the ruptured chordae allow the involved mitral leaflet to extend beyond its normal position of coaptation with the opposite leaflet and beyond the level of the involved part of the same leaflet. The regurgitant blood striking the leaflet is deflected rather sharply across the left atrium where it strikes the atrial wall or atrial surface of the intact mitral leaflet.

In two of the four cases in which no jet lesions could be definitely identified, some chordae had ruptured that were normally attached in the vicinity of the commissures. It has been postulated that the reason for the infrequent occurrences of left atrial jet lesions in association with rheumatic mitral insufficiency is that the energy of the regurgitant stream is dissipated by the direction the regurgitant stream takes into the left atrium. This also may explain the absence of left atrial jet lesions in these two cases of ruptured chordae to the commissural regions in which, probably, the regurgitant stream was directed more centrally into the left atrial cavity.

Findings in Study of Clinical Records

Murmurs

The theory has been advanced that the location of jet lesions may explain some of the characteristics of heart murmurs.17,18 The location of the ruptured chordae (the particular portion of the leaflet that allows regurgitation) and the evidence of jet lesions in the left atrium form the anatomic bases for an explanation of some characteristics of the murmurs.

The murmur most frequently recorded in this series of cases was an apical systolic murmur, which was present in every case. The adjectives used to describe the murmurs varied considerably but included the terms 'harsh,' ‘high pitched,’ ‘whistling,’ 'blowing,' and 'coarse,' as well as various estimations of the intensity. An associated systolic thrill at the apex was recorded in five cases. In the cases in which no lesions of other valves existed, a diastolic murmur was recorded in three instances. This was specifically described in one patient as a presystolic component of the systolic murmur.

In the group as a whole there was no uniformity in the transmission of the systolic murmurs. The areas to which transmission occurred were those in which the murmur of mitral insufficiency is typically transmitted, namely, the left axilla, the left posterior portion of the thorax, and the basal area of the precordium. There appeared to be a definite correlation between the location of the ruptured chordae and the transmission of murmurs in some cases.

Definite left atrial jet lesions were recognized in all eight cases involving chordae of the posterior leaflet. In one case the jet lesion was located on the anterior mitral leaflet opposite the site of ruptured chordae. In seven cases the lesions were located on the septal wall of the left atrium. In one case of rupture of chordae to the posterior mitral leaflet the jet lesion was located in the septal wall of the left atrium adjacent to the aortic valve (fig. 3). The apical systolic murmur was transmitted to the aortic area and to the vessels of the neck; it simulated the murmur of aortic stenosis. The proximity of the jet lesion to the aortic valve forms the anatomic basis for the theory of the production of the aortic systolic murmur in this case.

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Figure 5

Case 6. Active endocarditis due to Streptococcus mitis with involvement of the aortic valve, with aortic insufficiency and secondary spread of the infectious process to the chordae of the anterior mitral leaflet. Vegetations on the chordae are demonstrated. Ruptured chordae, though not clearly evident, were located in the region of this infectious activity. (The arrow points in the presumed direction of the regurgitant stream originating at the aortic valve.)

An additional patient in the present study (case 19) had auscultatory findings similar to those in the case described (case 16). These findings were a basal systolic murmur with transmission to the cervical vessels in addition to the apical systolic murmur. The aortic valve was normal at necropsy in this case also, and the jet lesions on the septal wall were anatomically close to the aortic valve.

Two other cases of mitral insufficiency due to ruptured chordae tendineae simulating aortic stenosis have been described. 18, 20 In both cases, however, the chordae to the anterior mitral leaflet were ruptured rather than the chordae to the posterior mitral leaflet. In one the jet lesions were partially adjacent to the aortic valve, but in the other the jet lesions were on the posterior atrial wall.

In the present study there were four other cases in which ruptured chordae of the posterior leaflet were found and basal systolic murmurs were recorded. Thus, in six of the eight cases with ruptured chordae of the posterior leaflet, transmission of an apical systolic murmur to the basal area of the precordial was present. In these eight cases the apical systolic murmur was transmitted also to the left axilla in three, to the posterior left part of the thorax in two, and to both of these areas in one case. An anatomic basis for the transmission of the murmurs posteriorly to the left in these cases was not apparent. Jet lesions were identified in the left atrium in seven of the nine cases in which the anterior leaflet was the site of the ruptured chordae tendineae. The location in the atrial wall ranged from the posteromedial atrial wall to the lateral wall, being generally opposite the site of incompetency of the anterior leaflet.

The systolic murmurs were heard in other areas in addition to the apex in seven of the nine cases. The apical systolic murmur was transmitted to the left axilla and posterior portion of the left side of the thorax in two, to the left axilla alone in four, and to the posterior left portion of the thorax alone in one. Basal systolic murmurs were recorded in four cases in this subgroup of nine cases, but in two cases aortic stenosis was verified at necropsy, while in two cases the aortic valve was not stenotic.
The findings relating particularly to the transmission of the apical systolic murmur to the basal precordial areas in cases with rupture of chordae to the posterior leaflet, and those relating to the transmission of the murmurs posteriorly to the left in cases with rupture of chordae to the anterior leaflet, offer support to the theory that the transmission of murmurs is related in part to the direction of regurgitant flow and possibly to the impact of blood on the left atrial wall. The lack of complete correlation between the sites of rupture and the location of the jet lesions to the areas in which the murmurs were recorded in these cases suggests that there are other factors that were not apparent.

**Time of Rupture of Chordae Tendineae**

The sudden appearance of a previously unrecognized precordial systolic murmur has been considered to be of significance in the diagnosis of ruptured mitral chordae tendineae.7 This situation, however, was not sufficiently well defined in any case in the present study to provide evidence for ruptured chordae as the cause of the mitral insufficiency. It was impossible to determine the time of appearance of the murmurs except in three instances in which a murmur was known to have begun during active bacterial endocarditis.

In those cases in which heart murmurs were known to be present prior to bacterial endocarditis, there was no good evidence for definite or significant change of the murmurs during the bacterial endocarditis.

**Cardiac Decompensation**

The sudden onset of cardiac decompensation or a sudden worsening of the state of cardiac compensation has been noted previously in patients with rupture of the mitral chordae tendineae.10,11 It has been postulated that this event may be temporally related to the rupture. This phenomenon was present in two cases in the present study (cases 19 and 20).

In these two cases the rapid and sudden appearance of symptoms and signs of cardiac decompensation appeared 12 and 11 months prior to death, respectively. In one of them (case 19) an apical systolic heart murmur was noted prior to this sudden appearance of failure. The characteristics of the murmurs were not significantly changed during the course of this patient’s illness.

The clinical features of these two cases bear certain resemblances to those of the cases of spontaneous rupture of the mitral chordae tendineae reported by Horton-Smith,10 Frothingham and Hass,11 and one of the cases reported by Bailey and Hickam.7

Signs and symptoms of cardiac decompensation were present in 16 of 20 cases in this study. In four cases cardiac decompensation was minimal, and in each of these only one or two ruptured chordae were found. In the patients in whom ruptured chordae were the primary factors in the cardiac disability there was a good correlation between the severity of the heart disease and the number of ruptured chordae.

**Discussion**

Bacterial endocarditis was the factor of major etiologic importance in the cases in this study. Of the 20 cases presented, bacterial endocarditis was known to be present during life in 10. In these cases necropsy showed the infection to be active in six and healed in four. In the other 10 cases there was no history of bacterial endocarditis, but in three of these there were findings at necropsy that were interpreted as those of healed bacterial endocarditis. In three additional cases necropsy findings were less definite but nevertheless suggestive of healed bacterial endocarditis. In the remaining four cases residual abnormalities from inflammatory processes in the heart were demonstrated but these changes were not specific. Mitral insufficiency in patients who have had bacterial endocarditis may be the result of one or of several abnormalities in addition to rupture of the chordae tendineae. These include destruction of the leaflet tissue, fixation of the posterior leaflet to the left ventricular wall by organized vegetations, and distortion of the leaflets by large vegetations and enlargement of the mitral orifice.17 In the 20 cases of this study, rupture of the chordae tendineae was the primary factor in mitral insufficiency in five.

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The role of rheumatic endocarditis in the production of rupture of mitral chordae tendineae is not completely known. In some cases, rheumatic endocarditis predisposes to bacterial endocarditis even to the location of bacterial vegetations on the valve-chordae complex. Cases were encountered in the present series in which the inflammatory changes observed were suggestive of healed rheumatic endocarditis, and convincing evidence, either clinical or pathologic, could not be found for superimposed bacterial endocarditis. It is perhaps possible also that chordae tendineae, involved by rheumatic endocarditis, could rupture spontaneously.

In the present series, the microorganisms responsible for bacterial endocarditis were largely streptococci of different varieties. The antibacterial measures now available effect a cure in a great many cases owing to streptococci. In such cases little evidence might remain at necropsy of previous bacterial endocarditis. It seems possible that bacterial endocarditis, whether clinically recognized or not, could be responsible for rupture of mitral chordae tendineae and still not show pathognomonic evidence of its existence.

Two cases in the present study (cases 19 and 20) and three reported previously, suggest that rupture of chordae tendineae did not occur during active inflammation. The two patients in this series apparently were in good health when dyspnea developed rather suddenly after a period of unusually heavy exertion. Dyspnea initiated serious cardiac decompensation and these two patients died 11 and 12 months later, respectively. It is postulated that the rupture of the chordae tendineae occurred near the time of onset of dyspnea.

Rupture of mitral chordae tendineae might be responsible for the production of new murmurs. In the cases in this study, recognition of the beginning of cardiac murmurs was not a useful clinical event. The principal difficulty in making the diagnosis of ruptured mitral chordae tendineae on a clinical basis would seem to be in differentiating it from other causes of mitral insufficiency. Ruptured mitral chordae tendineae as a cause of mitral insufficiency might be suspected, but probably a definite diagnosis would be difficult or impossible clinically. The appearance of mitral insufficiency in association with bacterial endocarditis or with a febrile illness which was not definitely identified, or in which the transmission of the murmur seemed to be in keeping with the anatomic varieties discussed previously, should be considered suggestive. Recent surgical advances in the treatment of mitral insufficiency have led to increased interest in the entire problem. A technic for surgical correction of mitral insufficiency due to ruptured mitral chordae has been described by McGoon. Fortunately it is not vital to make this difficult specific diagnosis before exposing the mitral valve at the time of open-heart operation.

Summary

The present study presents pertinent clinical and pathologic findings in 20 cases of ruptured mitral chordae tendineae encountered at the Mayo Clinic between 1934 and 1958 inclusive. Mitral insufficiency results from the rupture of chordae tendineae, the severity being related to the number of chordae ruptured. The resulting heart disease may be severe and may progress to cardiac decompensation and death. Bacterial endocarditis was the major etiologic factor in rupture of the chordae tendineae in this study.

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The area of the transverse section of the pulmonary artery being in one part, before it divaricates into branches, of the same dimension with the orifice of the aorta, the velocity of the blood in that part may be accounted the same as in the orifice of the aorta. But though the quantities and velocities of the blood, in passing out of both ventricles, be the same, yet it does not thence follow, that their expulsive forces must be both the same: for if the blood in passing into the pulmonary artery, finds less resistance from the preceding blood, than the blood does in entering into the aorta, then a less force will expel it out of the right ventricle with equal velocity; and accordingly, as there is not so much force required to drive the blood thro’ the lungs, as thro’ the rest of the whole body, so we may observe, that the substance of the muscle of the right ventricle has not near the thickness of that of the left.—Stephen Hales, B.D., F.R.S., Haemastatics, Vol. II, London, 1733.
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PHILIP J. OSMUNDSON, JOHN A. CALLAHAN and JESSE E. EDWARDS

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