Repair of Mitral Incompetence Secondary to
Ruptured Chordae Tendineae

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SUMMARY
Twenty-two patients with ruptured chordae tendineae are presented. Etiology was rheumatic heart disease in seven patients, bacterial endocarditis in four patients, both rheumatic heart disease and endocarditis in two patients, trauma in one patient, and aortic regurgitation in one patient. Exact cause of rupture was unknown in seven patients. Plastic repair of the leaflet and/or posteromedial annuloplasty was done in all the patients. In two patients the annuloplasty broke down soon after the operation and necessitated valve replacement. There was one hospital death. One patient died two years after operation probably because of cerebral embolism; a large thrombus was found in the left atrium at autopsy. All survivors have been followed for 7 months to 8 years and, except for two, are either working full-time or leading an active life.

In ruptured chordae early and five-year results after repair of the leaflet and/or posteromedial annuloplasty are very good irrespective of the left atrial enlargement and "V" wave size. Results were better in the rheumatic group than in the nonrheumatic group. In our opinion this method of treatment is superior to prosthetic valve replacement for this condition, except if the annulus is thin and attenuated.

Additional Indexing Words:
Bacterial endocarditis Mitral valve surgery Mitral insufficiency

The chordae tendineae constitute an important component of the mitral valve apparatus and play a crucial role in its mechanics. Disruption of the chordae results in a flail leaflet which everts like a hood into the left atrium causing mitral regurgitation and progressive cardiac failure. Although ruptured chordae tendineae have been known to cause mitral regurgitation for a long time, it is only in the last decade that their real importance has been stressed.1-10 Various conditions like rheumatic heart disease, bacterial endocarditis, myocardial infarction, trauma, and connective tissue disease, etc., may cause rupture of the chordae. However, there are a significant number of cases in which the cause of rupture is unknown. In its treatment, there are some who do plastic repair of the lesion,5, 6, 8, 11-14 while others prefer prosthetic valve replacement.10-17

This report describes our experience with this condition and also evaluates the results after plastic repair of the mitral valve with particular reference to long-term follow-up.

Methods
Between January 1960 and December 1970, 22 patients with severe mitral insufficiency secondary to ruptured chordae tendineae were operated upon (patients with ruptured papillary muscle are not included). Thirteen of them were males and eight were females; their ages ranged from 30 to 64 years. Functionally all patients were class III or IV (NYHA). Seven patients had a history or stigmata of rheumatic heart disease, while four...
patients gave a history of subacute bacterial endocarditis in the recent past. Two patients had a positive history for both rheumatic heart disease and bacterial endocarditis. Included in the rheumatic group were three patients who had no history of rheumatic fever or bacterial endocarditis, but had thickening of leaflets and/or calcification of the annulus. In one patient the precipitating factor for the chordal rupture was a fall from a horse. Chordal rupture was probably secondary to aortic regurgitation in one case. Exact cause of the ruptured chordae was unknown in the remaining seven patients.

The electrocardiographic findings, hemodynamic data, and operative findings are listed in table 1. Nine patients had either acute onset of heart failure or rapid deterioration in the presence of pre-existing symptoms. The chordae of the posterior leaflet were involved in 13 cases. In nine patients chordae of the anterior leaflet were ruptured. Three patients had tears or perforations of the leaflets in addition to ruptured chordae. In six out of nine patients with ruptured chordae of the anterior leaflet, the systolic murmur could be heard over the vertex of the skull in addition to the usual places.

Surgery was performed with the aid of a rotating disc pump oxygenator primed with a mixture of blood and lactated Ringer's solution at a flow rate of 2.4 liters/m²/min. The heart was exposed through a right posterolateral thoracotomy and the left atrium opened through the interatrial groove. Plication of the flail segment of the leaflet alone or along with "V" excision of the flail segment was performed in 20 patients (fig. 1). Plication was done with a continuous horizontal mattress stitch reinforced with a continuous whip stitch. Thus the flail segment was everted and the next intact chordae were brought adjacent to each other (fig. 1c). In addition, tears and/or perforations were repaired in three patients. These repairs were supplemented with posteromedial annuloplasty in all the patients. In one patient anterolateral annuloplasty was also done. Because of marked tricuspid regurgitation, tricuspid annuloplasty was done in two patients besides the above procedures, while in another patient the aortic valve was replaced with a Starr-Edward prosthesis. In two patients leaflet plication was not done because the regurgitation could be controlled by annuloplasty alone. Antibiotics were administered prophylactically during the postoperative period for five days. No anticoagulants were given except in patients with prosthetic valves.

All patients who survived surgery have been followed. Direct examination was done in 15 patients. In others, information was obtained by communication with the referring physician or the patient or both. Particular note was made of any symptoms, whether the patient was able to work, presence of any apical systolic murmur and any history of embolization. Exercise tolerance test, ECG, and chest X-ray for heart size were done whenever possible. Patients who were able to work or had advanced at least one class according to NYHA classification were considered to be improved.

Results
There was one hospital death in 22 patients. This patient had a thin, attenuated annulus and the annuloplasty broke down soon after coming off bypass. Bypass was reinstated and the mitral valve replaced with a Cooley-Bloodwell valve; the patient died in the surgery. Another patient, who also had a thin and weak annulus, developed sudden, severe mitral regurgitation several hours after the surgery. She was reoperated immediately and the mitral valve was replaced with a Gott valve. She survived a stormy postoperative course, and has a permanent pacemaker for postoperative heart block. Neither patient had a history or evidence of rheumatic heart disease or bacterial endocarditis.

Twenty-one patients who left the hospital have been followed from 7 months to 8 years (an average of 40.7 months). One patient died 2 years after the operation, probably because of cerebral embolism. At autopsy, there was a large thrombus in the left atrium; the mitral valve looked satisfactory and the repair was intact. This patient had chronic atrial fibrillation. Eighteen of the 20 survivors are asymptomatic and working full-time or leading an active life. Of the remaining two, one suffered a stroke (probably embolic) and cannot work. His cardiac status however, is considerably improved after the operation. The other patient who was class IV NYHA preoperatively, is class II–III NYHA now. His heart size is smaller than preoperative level but still bigger than normal, and he has a murmur of mitral regurgitation. This patient had a 30-year history of hypertension and may have suffered at least one attack of myocardial infarction. Preoperative electrocardiogram revealed left ventricular hypertrophy and strain and intraventricular
Table 1

Preoperative Data, Operative Findings, and Follow-Up of Patients with Ruptured Chordae Tendineae

<table>
<thead>
<tr>
<th>No.</th>
<th>Name, age, and sex</th>
<th>Pertinent history</th>
<th>Duration of murmur</th>
<th>Duration of CHF</th>
<th>Functional class (NYHA)</th>
<th>ECG</th>
<th>Hemodynamic</th>
</tr>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>FAP (mmHg)</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>PCP (mmHg)</td>
</tr>
</tbody>
</table>

**A. Nonrheumatic**

1. T.C. 61F nil 5 mo. 5 mo. III NSR 34* 22

2. A.A. 55M SBE 1 yr. 1 yr. III NSR; LVH 27/10 33/9

3. P.J. 57M nil 6 mo. 6 mo. III AF; LVH 36 40/11

4. J.A. 49M Fall from horse 8 wk. 8 wk. III NSR 28 30/7

5. K.L. 49M Hypertension 2–3 mo. 2–3 mo. IV NSR; LVH; LVS; IVCD 32 38/2

6. H.H. 64M nil 8 yr. 4 yr., worse for 6 mo. III NSR 50 60/20

7. E.W. 61M nil 14 yr. 2½ yr.; worse for 1½ yr. III–IV NSR; LVH 38 30

8. M.R. 49M nil 7 mo. 7 mo. III NSR; LVH 41 –

9. V.M. 36F SBE 17 yr. 3 yr. III–IV NSR; LVH – –

10. M.J. 38M SBE 5 mo. 5 mo. III NSR; LVH 44 20

11. G.C. 30M SBE 14 yr. 1 yr. III NSR; LVH 42 27

12. W.W. 46M nil 8 yr. 8 yr. III AF; LVH 58 30–34

13. P.P. 47F nil 29 yr. 4 yr. III AF – –
### A. Nonrheumatic

<table>
<thead>
<tr>
<th>LAP (mmHg)</th>
<th>&quot;V&quot; wave</th>
<th>LVEDP (mmHg)</th>
<th>PVR (dasc&quot;)</th>
<th>LA vol. (ml.)</th>
<th>CI</th>
<th>Operative findings</th>
<th>Postoperative course and follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>—</td>
<td>25</td>
<td>—</td>
<td>150</td>
<td>101</td>
<td>3.9</td>
<td>LA normal size; annulus thin &amp; friable; ruptured chordae post. leaflet</td>
<td>Annuloplasty breakdown several hours after operation; mitral valve replaced with Gott valve; 3 yr. postop. &amp; doing all right; had one embolic episode; 3/4 apical systolic murmur</td>
</tr>
<tr>
<td>—</td>
<td>22</td>
<td>3-12</td>
<td>29</td>
<td>144</td>
<td>2.9</td>
<td>Small LA; ruptured chordae &amp; perforation post. leaflet</td>
<td>1 1/2 yr. postop.; working full-time; 1/6 apical systolic murmur</td>
</tr>
<tr>
<td>—</td>
<td>24</td>
<td>0-9</td>
<td>182</td>
<td>314</td>
<td>3.4</td>
<td>Small LA; ruptured chordae post. leaflet</td>
<td>3 1/2 yr. postop.; suffered a stroke &amp; can't work; 2/6 apical systolic murmur</td>
</tr>
<tr>
<td>—</td>
<td>23</td>
<td>—</td>
<td>276</td>
<td>176</td>
<td>2.5</td>
<td>Slightly enlarged LA; ruptured chordae post. leaflet</td>
<td>2 yr. postop.; working full-time; no apical systolic murmur</td>
</tr>
<tr>
<td>—</td>
<td>36</td>
<td>20</td>
<td>—</td>
<td>208</td>
<td>2.4</td>
<td>Slightly enlarged LA; ruptured chordae ant. leaflet</td>
<td>2 yr. postop.; 3/6 apical systolic murmur radiating to axilla; heart size smaller than preop. level but bigger than normal; symptomatic and can't work</td>
</tr>
<tr>
<td>—</td>
<td>40</td>
<td>22</td>
<td>570</td>
<td>170</td>
<td>1.5</td>
<td>Normal-sized LA; ruptured chordae ant. leaflet; annulus very thin &amp; friable</td>
<td>Annuloplasty breakdown on the table; mitral valve replaced with Cooley-Bloodwell valve; could not be taken off bypass</td>
</tr>
<tr>
<td>—</td>
<td>—</td>
<td>20</td>
<td>184</td>
<td>—</td>
<td>1.9</td>
<td>LA enlarged; ruptured chordae post. leaflet</td>
<td>2 yr. postop.; leading active life; 3/6 apical systolic murmur</td>
</tr>
<tr>
<td>29</td>
<td>65</td>
<td>—</td>
<td>330</td>
<td>295</td>
<td>1.4</td>
<td>Enlarged LA; ruptured chordae post. leaflet</td>
<td>1 yr. postop.; working full-time; no murmur</td>
</tr>
<tr>
<td>22</td>
<td>25</td>
<td>10</td>
<td>—</td>
<td>—</td>
<td>2.6</td>
<td>LA not markedly enlarged; ruptured chordae ant. leaflet</td>
<td>6 yr. postop.; working full-time; 2/6 apical systolic murmur</td>
</tr>
<tr>
<td>20</td>
<td>46</td>
<td>15</td>
<td>—</td>
<td>—</td>
<td>1.7</td>
<td>LA not markedly enlarged; ruptured chordae ant. leaflets with tear of both ant. &amp; post. leaflets</td>
<td>8 yr. postop.; working full-time; no murmur</td>
</tr>
<tr>
<td>60/10</td>
<td>50</td>
<td>0-10</td>
<td>330</td>
<td>—</td>
<td>2.2</td>
<td>LA not markedly enlarged; ruptured chordae ant. leaflet</td>
<td>8 yr. postop.; working full-time; 3/6 apical systolic murmur</td>
</tr>
<tr>
<td>—</td>
<td>48</td>
<td>22</td>
<td>600</td>
<td>—</td>
<td>1.8</td>
<td>LA not enlarged; ruptured chordae post. leaflet, also a tear in same leaflet; annulus was thin &amp; friable; also had AS &amp; AI</td>
<td>1 1/3 yr. postop.; asymptomatic &amp; working full-time; no murmur and in NSR</td>
</tr>
<tr>
<td>30/8</td>
<td>—</td>
<td>6</td>
<td>203</td>
<td>—</td>
<td>—</td>
<td>LA enlarged; ruptured chordae post. leaflet</td>
<td>7 mo. postop.; 4/6 apical systolic murmur radiating to axilla &amp; neck; working 5 hr/day</td>
</tr>
<tr>
<td>No.</td>
<td>Name, age, and sex</td>
<td>Pertinent history</td>
<td>Duration of murmur</td>
<td>Duration of CHF</td>
<td>Functional class (NYHA)</td>
<td>ECG</td>
<td>PAP (mmHg)</td>
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</tr>
<tr>
<td>1.</td>
<td>T.M. RF 50F</td>
<td>RF</td>
<td>36 yr.</td>
<td>6 yr.</td>
<td>IV AF; LVH</td>
<td></td>
<td>23</td>
</tr>
<tr>
<td>2.</td>
<td>W.A. RF 50F</td>
<td>Since childhood</td>
<td>6 yr.</td>
<td></td>
<td>III–IV AF</td>
<td></td>
<td>20 17/5</td>
</tr>
<tr>
<td>3.</td>
<td>R.P. nil 54M</td>
<td></td>
<td>24 yr.</td>
<td>2–3 mo.</td>
<td>III–IV AF; LV; ischemia</td>
<td></td>
<td>43</td>
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<tr>
<td>4.</td>
<td>S.M. nil 35F</td>
<td></td>
<td>2½ yr.</td>
<td>2½ yr.</td>
<td>IV AF</td>
<td></td>
<td>78/41</td>
</tr>
<tr>
<td>5.</td>
<td>B.I. Hypertension 50M</td>
<td></td>
<td>40 yr.</td>
<td>2 yr.</td>
<td>III NSR; LVH</td>
<td></td>
<td>36</td>
</tr>
<tr>
<td>6.</td>
<td>C.H. RF; SBE 50M</td>
<td></td>
<td>43 yr.</td>
<td>8 yr.</td>
<td>IV AF; LVH; LVs</td>
<td></td>
<td>40</td>
</tr>
<tr>
<td>7.</td>
<td>J.A. RF 52F</td>
<td></td>
<td>41 yr.</td>
<td>5 yr., worse</td>
<td>III AF; Biventricular hypertension</td>
<td></td>
<td>42</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>for 6 mo.</td>
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<tr>
<td>8.</td>
<td>W.M. RF 39F</td>
<td></td>
<td>21 yr.</td>
<td>1 yr.</td>
<td>IV AF; LVH; LVs</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>9.</td>
<td>B.G. RF; SBE 55F</td>
<td></td>
<td>45 yr.</td>
<td>9 mo.</td>
<td>III AF; LVH</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**B. Rheumatic**

- **Table 1** (continued)

| Abbreviations: CHF = congestive heart failure; MI = myocardial infarction; SBE = subacute bacterial endocarditis; RF = rheumatic fever; AF = atrial fibrillation; NSR = normal sinus rhythm; LVH = left ventricular hypertrophy; LVS = left ventricular strain; LA = left atrium; PA = pulmonary artery; PC = pulmonary capillary; NYHA = New York Heart Association; PVR = pulmonary vascular resistance; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; Post. = posterior; Ant. = anterior; Postop. = postoperative; TI = tricuspid insufficiency; AI = aortic insufficiency; AS = aortic stenosis; IVCD = intraventricular conduction defect; dsc⁻² = dynes second centimeter⁻².

- *Bar indicates mean pressure.

Conduction defect. Cardiac catheterization one and three-fourths years after surgery, showed that the mitral regurgitation was decreased from 80 ml/beat (53%) to 13 ml/beat (14%); the left ventricular end-diastolic pressure remained unchanged (20 mm Hg), but ejection fraction dropped from 40% to 26%. This patient was confined to chair before surgery but is able to do household chores now. The improvement, however, has been limited because of impaired left ventricular function probably on the basis of hypertensive heart disease and mitral regurgitation.

There were three instances of embolism. One occurred in the patient with a prosthetic valve while on anticoagulants. Out of 19 patients discharged from the hospital after repair of the mitral valve and not on anticoagulants, two (approximately 10%) developed embolism, resulting in one death. Both patients had chronic atrial fibrillation. Fourteen patients (including one with a
prosthetic valve) have an apical systolic murmur. The murmur is grade 1/6 in two patients, 2/6 in six, 3/6 in five, and 4/6 in one patient. Six patients have no murmurs of mitral insufficiency. All patients with systolic murmurs are doing very well functionally except one (K.L.).

Comparing the results in the two groups, of the nine patients in the rheumatic group, eight have had a good result from plastic repair, and one suffered a fatal cerebral embolism two years postoperatively. Of the survivors, two have no murmur. In the nonrheumatic group, only nine of the 13 patients have had a good result from reconstructive surgery. Four of them have no murmur. Two had an immediate breakdown requiring prosthetic replace-

ment (of these, one died), one suffered a cerebrovascular accident which prevents working, and one is unable to return to full-time work because of hypertensive heart disease and some residual mitral regurgitation.

Discussion

Opinions differ regarding the surgical treatment of the ruptured chordae tendineae. Some recommend routine prosthetic valve replacement\(^{15-17}\) of the mitral valve in all such cases, while others do this only in ruptured chordae of the anterior leaflet.\(^{18}\) Still others do plastic repair of the leaflets and anuloplasty for this condition.\(^5\,6,\,8,\,11\,14\)

The major alternative to plastic repair is valve replacement. The data available dealing with long-term results after mitral valve

<table>
<thead>
<tr>
<th>Data</th>
<th>Operative findings</th>
<th>Postoperative course and follow-up</th>
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</thead>
<tbody>
<tr>
<td>LAP (mmHg)</td>
<td>“V” wave</td>
<td>LVEDP (mmHg)</td>
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<td>35</td>
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<td>350</td>
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<td>350</td>
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<tr>
<td>28/4</td>
<td>25-60</td>
<td>820</td>
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</tbody>
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replacement have mainly concerned patients suffering from rheumatic valvular heart disease (data not exclusively for ruptured chordae), and therefore are not necessarily comparable to ours. However, some comparisons are germane.

From the Mayo Clinic is reported a five-year survival rate of approximately 50% following mitral valve replacement (hospital mortality included). Starr et al. report similar data for patients operated in 1960 and 1961, with an incidence of thromboembolism of about 37%. Because of the problems associated with prosthetic valves, we have always preferred to repair the mitral valve whenever possible and use the prosthesis as a last resort. Low operative mortality (approximately 4.5%), excellent long-term functional results (up to eight years), and low embolic rate without anticoagulants (approximately 10%) in this report amply justify this approach. Because emboli occurred only in patients with

\[ V \]
RUPTURED CHORDAE TENDINEAE

atrial fibrillation, this incidence might be further reduced by anticoagulating this group.

In the present study, six patients were operated more than five years ago (three of them eight years ago). Five patients had ruptured chordae anterior leaflet. Four were repaired by "V" excision of the flail segment and one by plication. All of them are alive and markedly improved; five of them are functional class I NYHA and one is class II. Two have no systolic murmurs. Two patients (G.C., M.J.) were catheterized 12 months and 7 months after operation; both showed significant decrease in mean pulmonary artery and pulmonary capillary pressure and increase in the cardiac index. The percentage of regurgitation decreased from 81% to 13% in patient G.C., and from 77% to 0% in patient M.J. Although the number of patients is small, it is obvious that mitral reconstruction for ruptured chordae is capable of giving excellent long-term results.

Fourteen patients have apical pansystolic murmurs; functionally, all of them are improved except one. Anderson et al., who studied the clinical and hemodynamic status of 11 patients 4-41 months after mitral annuloplasty for mitral regurgitation, found that the increase in intensity of systolic murmur was a less prominent feature of improvement in these patients. In this series, incidence of postoperative systolic murmur was higher in patients with ruptured chordae anterior leaflet (seven out of nine) compared to posterior leaflet (six out of 13); there was not much difference between the two groups as far as functional results were concerned. The methods of repair were similar in the two groups.

A posteromedial annuloplasty was done in all patients after repair of the leaflet. We believe this is an important step because the repair reduces the atrioventricular area and thus maximizes the available leaflet area. Various types of valvuloplastic procedures have been reported. We prefer to plicate the flail segment with or without excision of a "V"-shaped segment from the flail leaflet. This valvuloplasty is simple and leaves behind very little foreign material. We have little experience with artificial chordae, but agree with the objection to their use that after correction of the mitral regurgitation the left ventricle will shrink in size, possibly making the new chordae redundant and producing recurrent insufficiency. Certainly an estimate of necessary chordal length in an empty heart at surgery is at best difficult and subject to serious errors. The breakdown of annuloplasty occurred only in the group without rheumatic fever (see below). The patients with rheumatic fever usually have leaflets that, although pliable, are several times thicker than normal. Conceivably the same process extends into the annulus resulting in a tougher structure for holding sutures.

There were two instances of breakdown of annuloplasty. One occurred soon after coming off the bypass, and the other happened several hours after the surgery. The annulus in both cases was attenuated and friable. The leaflets as well as the chordae were thin, unscarred without any evidence of inflammation. Microscopically large quantities of ground glass hyaline substance and/or basophilic material were seen in the leaflet. It is possible that both patients had some sort of connective tissue disorder with poor suture-holding power. These cases may have some resemblance to the myxomatous valvar transformation reported by Read et al.; out of ten patients who underwent valve replacement, three developed dehiscence of the valve ring, resulting in one death and three reoperations. Since the basic problem here is one of poor attenuated friable tissue, neither of the alternatives—plastic repair or valve replacement—is attractive. In such cases, although both repairs are prone to breakdown, distribution of the stress with multiple sutures over a wider area with the artificial valve makes valve replacement look preferable.

It has been reported that patients with small left atrium and large "V" waves are most likely to benefit from surgery. This, however, should not be taken to mean that all patients with a large atrium and small "V" waves are poor surgical candidates, and

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therefore, that surgery is contraindicated. Excellent results have been obtained in the present series after plastic repair of mitral insufficiency due to ruptured chordae tendineae irrespective of the atrial enlargement and "V" wave size. On the other hand, patient K.L. is interesting because although he had an acute onset, slightly enlarged left atrium, and big "V" waves, his long-term result after repair was unsatisfactory because of poor left ventricular myocardial function and residual mitral regurgitation.

References


Correction
Kirklin JW: Circulation 43: 321, 1970. The title should be:
Pulmonary Arterial Banding in Babies with Large Ventricular Septal Defects
Repair of Mitral Incompetence Secondary to Ruptured Chordae Tendineae
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