Significance of an Atrial Gallop Sound in Mitral Regurgitation

A Clue to the Diagnosis of Ruptured Chordae Tendineae

By Lawrence S. Cohen, M.D., Dean T. Mason, M.D., and Eugene Braunwald, M.D.

SUMMARY
The incidence of atrial gallop sounds was studied in 51 patients with pure mitral regurgitation of sufficient severity to require operative treatment. Seventeen patients were in sinus rhythm. Nine of them had atrial gallop sounds and mitral regurgitation secondary to ruptured chordae tendineae, while six without atrial gallop sounds had primary valvular disease. The degrees of disability or the hemodynamic findings did not differ among the patients with ruptured chordae tendineae and those with primary valvular disease and sinus rhythm. However, the duration of symptoms and the history of a heart murmur were much shorter and the left atrium tended to be smaller in patients with ruptured chordae tendineae than in those with primary valvular disease. Sinus rhythm in a symptomatic patient with severe mitral regurgitation, therefore, should suggest the diagnosis of ruptured chordae tendineae. If, in addition, an atrial gallop sound is found the diagnosis of ruptured chordae tendineae is even more likely.

ADDITIONAL INDEXING WORDS:
Sinus rhythm

Atrial gallop sound (fourth heart sound, presystolic gallop) is an unusual finding in patients with mitral regurgitation. The development of atrial fibrillation in many patients with severe mitral regurgitation precludes the occurrence of an atrial sound, and standard texts and reviews have not called attention to the presence of this sound in patients with mitral regurgitation and sinus rhythm.¹⁻⁹ Nine patients for whom the diagnosis of pure mitral regurgitation was confirmed at operation or autopsy and who exhibited an atrial gallop sound have been encountered at the National Heart Institute and ruptured chordae tendineae were responsible for the mitral regurgitation in all of them. A study of the clinical, electrocardiographic, and hemodynamic features was undertaken to identify the factors responsible for the production of an atrial gallop sound in the patients with ruptured chordae tendineae and its absence in patients with mitral regurgitation due to rheumatic involvement of the valve leaflets themselves.

Methods
Seventeen patients with pure mitral regurgitation and sinus rhythm were studied. Eleven of them were proven, at operation or at postmortem examination, to have ruptured chordae tendineae and the hemodynamic, surgical, and anatomic findings in these patients have been described recently.¹⁰ The other six patients all had mitral regurgitation due to primary valvular disease and were the only ones with this rhythm out of the 40 patients with mitral regurgitation who have undergone mitral valve replacement at this institution.

The presence or absence of an atrial gallop sound was determined by means of auscultation and confirmed by phonocardiograms. The latter were recorded at a paper speed of 75 mm per

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second on either a Sanborn four-channel Poly-Beam Recorder (Model 564) with a contact microphone (Model 350-1700-C10) and a heart sound preamplifier (Model 350-1700-B) or with a Sanborn Twin Beam apparatus, with use of a Sanborn 62-500 amplifier. The atrial gallop sound was identified as a distinct, low-pitched vibration which was preceded and followed by a steady base line, which followed the onset of the P wave by at least 70 msec, and which preceded the R wave.

Right heart catheterization was carried out in all 17 patients and transseptal left heart catheterization in 15 patients. Selective left ventricular angiocardiograms were performed in 11 patients.

Results

An atrial gallop sound was present in nine of the 11 patients with ruptured chordae tendineae (fig. 1) but in none of the six patients with mitral regurgitation due to rheumatic involvement of the valve. Although an opening snap was not heard or recorded in any of the patients, a ventricular diastolic gallop (third
heart sound) was present in each of the 17 patients.

There were six females and five males in the group with ruptured chordae tendineae and three females and three males in the group with primary mitral valvular regurgitation. The former group ranged in age from 17 to 61 years with an average age of 37 years, while the latter ranged from 10 to 37 years with an average age of 24 years.

Only one of the 11 patients with ruptured chordae tendineae had a history of acute rheumatic fever, while five of the six patients with primary mitral valvular disease had a documented episode of acute rheumatic fever. Six of the patients with ruptured chordae tendineae and atrial gallop sounds presented a history of previous bacterial endocarditis and in five of them the time interval between the episode and the hospital admission at which a fourth heart sound was detected and at which the diagnosis of ruptured chordae tendineae was established anatomically was less than 2 years. Four of the patients with primary mitral valvular disease also had a history of bacterial endocarditis and in them the episode occurred an average of 4 years prior to admission. In general, both the duration of the history of a heart murmur and of symptoms of diminished cardiac reserve were shorter among the nine patients with atrial gallop sounds and ruptured chordae tendineae than among the six patients with primary valvular disease without atrial gallop sounds (table 1).

As indicated, normal sinus rhythm was present in all 17 patients included in this report. One of the two patients with ruptured chordae tendineae who did not exhibit an atrial gallop sound had episodes of paroxysmal atrial fibrillation. Six of the 11 patients with ruptured chordae tendineae (five of nine with atrial gallop sounds) had first degree atrioventricular block, while only one patient with primary mitral valvular regurgitation had this conduction defect. Right axis deviation and electrocardiographic evidence of right ventricular hypertrophy occurred commonly in both groups. Electrocardiographic evidence of left atrial enlargement occurred more frequently in the patients with primary valvular disease than in those with ruptured chordae tendineae.

The left atrial chamber was near normal size or only slightly enlarged at operation or postmortem examination in seven of the 11 patients with ruptured chordae tendineae and was markedly enlarged in all six patients who were operated upon for primary valvular regurgitation.

The hemodynamic findings in the 17 patients are shown in table 1. There were no consistent or significant differences in the levels of intracardiac pressure or cardiac output among the patients with and those without atrial gallop sounds.

**Discussion**

Considerable controversy surrounds the mechanism of origin of diastolic heart sounds.11-14 The view that the sounds are produced by vibration of the ventricular wall during rapid filling15 has been challenged by the postulation that sudden tensing of the valve cusps and chordae tendineae is responsible for the diastolic filling gallop and possibly for the atrial gallop sound as well.6,16 Regardless of the precise mechanisms involved, there is general agreement that the atrial gallop sound consists of two components.16-20 The early, low frequency vibrations (approximately 15 cycles per second) comprising the first component, occur 70 to 140 msec after the beginning of the P wave of the electrocardiogram and simultaneously with the onset of the atrial-contraction wave in the left atrial pressure pulse. These low frequency vibrations are not audible but may be recorded with an intra-atrial phonocardiogram. The audible second component of the atrial sound (25 to 75 cycles per second) occurs 50 to 90 msec after the onset of pressure rise in the left atrium, at a time when blood is propelled by atrial contraction into the ventricle. The view that this sound always occurs during ventricular filling is supported by the finding that it occurs simultaneously with the peak of the "a" wave of the apex cardiodiagram and that it disappears when the
### Table 1

**Summary of Data on Seventeen Patients with Pure Mitral Regurgitation**

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex &amp; age (yr)</th>
<th>HRF age (yr)</th>
<th>SBE (yr PTA)</th>
<th>Duration of heart murmur/symptoms (yr PTA)</th>
<th>Atrial gallop sound</th>
<th>ECG</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P-R</td>
<td>LAE</td>
</tr>
<tr>
<td>1. R.H.</td>
<td>F 24</td>
<td>03-99-19</td>
<td>+</td>
<td>5/12</td>
<td>5/12</td>
<td>Present</td>
<td>0.20</td>
</tr>
<tr>
<td>2. L.S.</td>
<td>F 17</td>
<td>05-99-23</td>
<td>+</td>
<td>5/12</td>
<td>5/12</td>
<td>Present</td>
<td>0.18</td>
</tr>
<tr>
<td>3. A.L.</td>
<td>F 19</td>
<td>03-77-47</td>
<td>+</td>
<td>1</td>
<td>1</td>
<td>Present</td>
<td>0.22</td>
</tr>
<tr>
<td>4. C.O.</td>
<td>F 23</td>
<td>04-60-62</td>
<td>+</td>
<td>1</td>
<td>7</td>
<td>Present</td>
<td>0.16</td>
</tr>
<tr>
<td>5. B.F.</td>
<td>F 21</td>
<td>04-85-25</td>
<td>+</td>
<td>1</td>
<td>1</td>
<td>Present</td>
<td>0.18</td>
</tr>
<tr>
<td>6. J.S.</td>
<td>M 50</td>
<td>06-45-03</td>
<td>—</td>
<td>2</td>
<td>2</td>
<td>Present</td>
<td>0.16</td>
</tr>
<tr>
<td>7. J.W.</td>
<td>M 54</td>
<td>04-87-16</td>
<td>+</td>
<td>44</td>
<td>8</td>
<td>Present</td>
<td>0.24</td>
</tr>
<tr>
<td>8. A.A.</td>
<td>F 37</td>
<td>01-71-62</td>
<td>—</td>
<td>6/12</td>
<td>6/12</td>
<td>Present</td>
<td>0.16</td>
</tr>
<tr>
<td>9. B.M.</td>
<td>M 61</td>
<td>06-34-20</td>
<td>—</td>
<td>4</td>
<td>4</td>
<td>Present</td>
<td>0.22</td>
</tr>
<tr>
<td>10. A.F.</td>
<td>M 48</td>
<td>05-78-22</td>
<td>—</td>
<td>2</td>
<td>9/12</td>
<td>Absent</td>
<td>0.20</td>
</tr>
<tr>
<td>11. L.L.</td>
<td>M 52</td>
<td>05-45-40</td>
<td>+</td>
<td>24</td>
<td>2</td>
<td>Absent</td>
<td>0.28</td>
</tr>
</tbody>
</table>

Ruptured mitral chordae tendineae

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex &amp; age (yr)</th>
<th>HRF age (yr)</th>
<th>SBE (yr PTA)</th>
<th>Duration of heart murmur/symptoms (yr PTA)</th>
<th>Atrial gallop sound</th>
<th>ECG</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P-R</td>
<td>LAE</td>
</tr>
<tr>
<td>1. K.A.</td>
<td>M 27</td>
<td>05-22-14</td>
<td>+</td>
<td>9</td>
<td>3</td>
<td>Absent</td>
<td>0.20</td>
</tr>
<tr>
<td>2. M.S.</td>
<td>F 34</td>
<td>05-36-18</td>
<td>+</td>
<td>10</td>
<td>3</td>
<td>Absent</td>
<td>0.14</td>
</tr>
<tr>
<td>3. B.D.</td>
<td>F 14</td>
<td>05-20-12</td>
<td>+</td>
<td>9</td>
<td>7</td>
<td>Absent</td>
<td>0.16</td>
</tr>
<tr>
<td>4. R.B.</td>
<td>M 37</td>
<td>05-08-73</td>
<td>—</td>
<td>2</td>
<td>2</td>
<td>Absent</td>
<td>0.17</td>
</tr>
<tr>
<td>5. Z.F.</td>
<td>F 23</td>
<td>06-19-99</td>
<td>+</td>
<td>U</td>
<td>3</td>
<td>Absent</td>
<td>0.16</td>
</tr>
<tr>
<td>6. M.S.</td>
<td>M 10</td>
<td>06-09-94</td>
<td>+</td>
<td>3</td>
<td>3</td>
<td>Absent</td>
<td>0.21</td>
</tr>
</tbody>
</table>

**Rheumatic mitral valvular regurgitation**

**Abbreviations:**
- HRF = history of rheumatic fever
- SBE = subacute bacterial endocarditis
- yr PTA = years previous to hospital admission
- ECG = electrocardiogram
- P-R = P-R interval in seconds
- LAE = left atrial enlargement
- PA = pulmonary artery
- LA = left atrium
- LV = left ventricle
- S/D (mean) = systolic/diastolic (mean) pressures
- PVR = pulmonary vascular resistance in dynes seconds centimeter⁻⁵
- CI = cardiac index
atrium contracts against a closed mitral valve in the presence of complete heart block.

Wood\textsuperscript{21} has indicated that atrial fibrillation is usually present in patients with pure mitral regurgitation who are symptomatic and who seek medical attention. This statement is confirmed by our experience in that, of the 40 patients with primary valvular disease in whom mitral valve replacements have been carried out at this institution, only six were in sinus rhythm while 34 were in atrial fibrillation. In contrast, all 11 patients with ruptured chordae tendineae were in sinus rhythm even though their clinical disability was at least as great as that present in the patients with primary mitral valvular disease. The frequency of atrial fibrillation in primary valvular regurgitation may be the result of damage to the atrium with the episode of active rheumatic fever, it may reflect the relatively long time that regurgitation exists before the patient develops serious disability, and it may also be related to the degree of left atrial enlargement. Seven of the 11 patients with ruptured chordae tendineae had a murmur for less than 2 years, while all of the patients with valvular mitral regurgitation had murmurs for more than 2 years. These findings suggest that severe mitral regurgitation produced by ruptured chordae tendineae is characterized by a more rapid clinical course and comes to the attention of the physician relatively soon after its onset in comparison to the more common form of regurgitation produced by primary valvular involvement. Thus, symptoms of cardiac decompensation usually appear in the patients with ruptured chordae tendineae before the factors responsible for atrial fibrillation develop. Conversely, only six of 40 patients with rheumatic mitral regurgitation deemed ill enough to warrant surgical treatment were in normal sinus rhythm, suggesting that the progression of symptoms in these individuals is slower and that serious disability may not occur until relatively late in the course of the illness.

At least a part of the explanation for the occurrence of an atrial gallop sound in patients with ruptured chordae tendineae may be found in the shorter time course of their disease. In patients with chronic rheumatic mitral regurgitation the left atrium is stretched, slowly but persistently. It is likely that the audible component of the atrial gallop sound is dependent upon a vigorous atrial contraction and the chronically diseased, thin-walled, dilated atrium may not be capable of generating a contraction of sufficient force to produce a fourth heart sound. In contrast, the atria of patients with ruptured chordae tendineae may be only slightly dilated, but their walls often are markedly thickened,\textsuperscript{30} and their contractions may be vigorous enough to produce atrial gallop sounds. The finding of a normal or nearly normal-sized left atrium in seven of the 11 patients with ruptured chordae tendineae (five of the nine with atrial gallop sounds) and in none of the six patients with rheumatic valvular regurgitation and sinus rhythm is consistent with this hypothesis. One of the two patients with ruptured chordae tendineae who did not demonstrate an atrial gallop sound had episodes of atrial fibrillation, suggesting that the contractile properties of his left atrium might already have become impaired. This suggestion is supported by the finding that the atrial contribution to left ventricular filling could not be identified on his apex cardiogram when he was in normal sinus rhythm (fig. 2). Interestingly, this patient (L.L.) had known of a heart murmur for 24 years, a time far exceeding that of all but one of the other patients with ruptured chordae tendineae.

The finding of first degree atrioventricular block was more frequent in the patients with ruptured chordae tendineae. The reason for its more frequent appearance in these patients is not clear, but this electrocardiographic finding has been associated with the finding of an atrial gallop sound\textsuperscript{22} since the prolongation of atrioventricular conduction allows sufficient time for the occurrence of a ventricular filling sound prior to the onset of ventricular contraction.

In summary, it appears likely that the rapid development and shorter duration of mitral regurgitation in patients with ruptured chordae
tendineae is primarily responsible for the finding of an atrial gallop sound in these patients and not in the patients with primary valvular disease. Regardless of the mechanisms responsible for the findings reported, it is clear that the presence of sinus rhythm in a patient with pure mitral regurgitation whose disability is serious enough to require operative treatment should suggest the diagnosis of ruptured chordae tendineae. Thus, of a total of 51 such patients, 34 were in atrial fibrillation and all of these had primary valvular disease. Seventeen were in sinus rhythm and only six of these had primary valvular disease while 11 had ruptured chordae tendineae. The presence of an atrial gallop sound appears to be even better evidence for the presence of ruptured chordae tendineae, since nine of the 11 patients with this finding had ruptured chordae tendineae, while atrial gallop sounds were not heard in any of the six patients with primary valvular disease and sinus rhythm.

Addendum

Since the report was submitted, two clinical studies on patients with ruptured mitral chordae tendineae have been published. Although not specifically commented upon, all five patients reported by Childress et al., were in sinus rhythm and the one phonocardiogram illustrated demonstrates a fourth heart sound. In the study by Raftery et al., 22 of the 23 patients with ruptured chordae tendineae or diseased papillary muscles were in sinus rhythm. One of the three phonocardiograms

Figure 2

Phonocardiogram, apex cardiogram, and electrocardiogram of patient 11 (L.L.) with ruptured chordae tendineae and episodes of atrial fibrillation. Tracings obtained while patient was in sinus rhythm. No atrial contribution to ventricular filling can be identified on the apex cardiogram. Arrows mark the usual timing of an atrial filling wave.
illustrated also demonstrated a fourth heart sound.

Acknowledgment

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References

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