The Apical First Heart Sound as an Aid in the Diagnosis of Atrial Septal Defect

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An accentuated first heart sound at the mitral area is often noted in cases of uncomplicated atrial septal defect.\(^1\) Leatham and Gray\(^2\) showed that it is actually the second component of the first heart sound that is accentuated and stated that this occurs in the presence of large left-to-right shunts. We have found this phenomenon to be part of the typical phonocardiographic picture of atrial septal defect with large left-to-right shunt but have also noted it in three cases in whom the other phonocardiographic and clinical characteristics of atrial septal defect were not clearly present. On right heart catheterization, these latter three cases were shown to have atrial septal defects, the shunts being small or moderate in magnitude. In addition, the analysis of a large series of routine phonocardiograms indicated that this peculiar characteristic of the first heart sound could be a useful diagnostic clue, since it occurred only rarely in conditions other than those associated with an uncomplicated left-to-right shunt at the atrial level.

Material and Methods

In all, the phonocardiograms of 187 subjects were reviewed (table 1). All phonocardiograms were obtained during the past year. A multichannel photographic recording system\(^8\) was used at a paper speed of 75 mm. per second. The microphone was the BM No. 21300-4.\(^6\) The records were taken routinely at four sites: the aortic area (AA), the pulmonary area (PA), the left sternal edge at the third or fourth intercostal space (LSE), and the mitral area (MA). An electrocardiogram and sometimes a carotid or jugular venous pulse were recorded simultaneously with the phonocardiogram. At least two sound frequency bands were recorded at each of the four positions: low frequency (LF, 12 to 100 cycles per second) and high frequency (HF, 400 to 2000 cycles per second).

All records were taken with the patient in the recumbent position and breathing normally. In the few cases where the respiratory sounds were loud enough to interfere with the recorded heart sounds the records were taken with the breath held in expiration.

Careful measurements were made in each case of the interval between the Q wave onset in the electrocardiogram and the onset of the first heart sound (S\(_1\)), or the onset of each of its two components when S\(_1\) was split. The first component of a split S\(_1\) is considered to be the mitral valve component (M\(_1\))\(^3-5\) and characteristically begins 0.05 to 0.07 second after Q-wave onset. The onset of M\(_1\) may, however, be delayed in cases of mitral stenosis\(^5-8\) or systemic hypertension.\(^5\) The second component of S\(_1\) is considered to be the tricuspid component (T\(_1\)) and was identified for the purposes of this study as the second component of a split S\(_1\) occurring 0.08 to 0.1 second after Q-wave onset\(^4\) (fig. 1). Sounds occurring 0.12 to 0.17 second after Q-wave onset were regarded as early ejection sounds\(^9\) (fig. 2). Sounds beginning later than 0.17 second after Q-wave onset were considered to be systolic clicks of extraeardiae origin.

Results

In general the splitting of the first heart sound in the phonocardiogram was detected better in LF than in HF records, and especially during expiration.

In 89 cases the apical first heart sound was either of low intensity or was not split. This included one suspect case of atrial septal defect. Suspicion was on the basis of a systolic ejection murmur in the pulmonary area, a widely split second heart sound, a prominent pulmonary artery segment on x-ray, and an electrocardiographic pattern of incomplete right bundle-branch-system block. Unfortunately, hemodynamic studies were not performed.
APICAL FIRST HEART SOUND

In 57 patients $M_1$ was louder than $T_1$ at the apex. There were no suspected or proved cases of atrial septal defect in this group. In 23 other cases the two components tended to be equal in intensity. One of these 23 cases was a suspect atrial septal defect because of a systolic ejection murmur along the left sternal edge, wide splitting of the second heart sound, and a right bundle-branch-system block, but again hemodynamic studies could not be performed. Two others in this group of 23 had the clinical features of atrial septal defect; this diagnosis was proved by right heart catheterization. The diagnosis in one of these cases was further confirmed during surgery and at postmortem examination (fig. 3).

There were only 18 cases in the entire series in whom $T_1$ was louder than $M_1$ at the apex. These included 10 cases of atrial septal defect, proved at catheterization; and five others in whom the diagnosis was strong on clinical grounds. The etiologic diagnoses in the remaining three cases were arteriosclerotic heart disease, syphilitic heart disease, and primary pulmonary hypertension. The overall results are summarized in table 1.

The 10 proved cases of atrial septal defect could be divided into two groups. The first consisted of seven patients with all the clinical manifestations of atrial septal defect (fig. 4). Cardiac catheterization in each case revealed a left-to-right shunt at the atrial level, such that between 60 and 68 per cent of total pulmonary flow was shunt flow. In four of these cases the diagnosis was confirmed during surgical correction. In the second group of three patients the only noteworthy phonocardiographic finding was $T_1$ greater than $M_1$ at the mitral area. This served to raise the possibility of atrial septal defect. The clinical and laboratory findings in this group of patients are further described.

**Case Reports**

**Case 1**

N. M. was a 25-year-old asymptomatic Negro woman. The heart appeared slightly enlarged clinically but there were no abnormal impulses. On auscultation the first heart sound appeared to be of normal intensity and the second heart sound was physiologically split. In the recumbent position a midsystolic click could be heard. At the apex there was a low-pitched third heart sound. A soft ejection systolic murmur was heard at the pulmonary area. The phonocardiogram revealed, in addition, a split first heart sound at the mitral area with the second component louder than the first (fig. 5). X-ray and fluoroscopy revealed no chamber enlargement, and the electrocardiogram was within normal limits. Right heart catheterization revealed a left-to-right shunt at the atrial level and approximately 44 per cent of total pulmonary flow was calculated to be due to shunt flow. All pressures were normal.

**Case 2**

H. S. was a 51-year-old Negro woman admitted with a history of progressive exertional dyspnea and chest pain over the past 15 years.

The heart was not enlarged clinically. The first heart sound was of normal intensity and the second was physiologically split. A loud ejection systolic murmur was heard at the pulmonary area and along the left sternal border. The phonocardiogram revealed, in addition, that $T_1$ was

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**Table 1**

**Summary of Phonocardiographic Findings (Listed by Number of Cases)**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Apical S₂, soft or single</th>
<th>$M_1 &gt; T_1$</th>
<th>$T_1 = M_1$</th>
<th>$T_1 &gt; M_1$</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>6</td>
<td>12</td>
<td>4</td>
<td></td>
<td>22</td>
</tr>
<tr>
<td>Congenital heart disease (Excluding ASD)</td>
<td>27</td>
<td>9</td>
<td>6</td>
<td></td>
<td>42</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suspected</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Proved</td>
<td></td>
<td>2</td>
<td>10</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>44</td>
<td>29</td>
<td>3</td>
<td></td>
<td>76</td>
</tr>
<tr>
<td>Miscellaneous conditions</td>
<td>11</td>
<td>7</td>
<td>7</td>
<td>3</td>
<td>28</td>
</tr>
<tr>
<td>Total</td>
<td>89</td>
<td>57</td>
<td>23</td>
<td>18</td>
<td>187</td>
</tr>
</tbody>
</table>
greater than M₁ at the apex. A midsystolic click at the mitral area was also recorded (fig. 6). At fluoroscopy a prominent hilar dance was noted but there was no localized chamber enlargement. The electrocardiogram showed nonspecific ST-T abnormalities. At right heart catheterization, the catheter was passed into the left atrium via an interatrial communication. Left and right atrial pressure pulses differed in contour but were similar in mean level. A left-to-right shunt calculated at 50 per cent of total pulmonary flow was found at the atrial level.

Case 3

L. P. was a 20-year-old white woman complaining of transient precordial pain, slight dyspnea on exertion, and palpitations over the preceding 4 months.

The heart was not enlarged clinically. The first and second heart sounds appeared normally split and there was a grade-II ejection systolic murmur along the left sternal border. Phonocardiography revealed, in addition, an accentuated first heart sound due to a tricuspid component louder than the mitral at the mitral area (fig. 7). The electrocardiogram, as well as cardiac x-rays and fluoroscopy, was within normal limits. At catheterization the left atrium and ventricle were entered via an interatrial communication. A left-to-right shunt making up about 40 per cent of total pulmonary flow was found at the atrial level.

Discussion

Splitting of the first heart sound was first described by Potain in the nineteenth century. He advanced no physiologic explanation for this finding although he believed that the first heart sound was produced by closure of the atroventricular valves.

In 1925, Katz demonstrated experimentally in dogs that there is slight asynchronism in contraction and ejection of the two ventricles. It has since become generally accepted that the mitral valve closes before the tricuspid valve in man and that these two events are the main components of the physiologically split first heart sound.

Normally M₁ is louder than T₁ at the apex. This is believed due to the relatively more powerful contraction of the left ventricle and thus more forceful closure of the mitral valve. At the tricuspid area T₁ is often louder than M₁; this is attributed to the relative proximity of the tricuspid valve or right ventricle to the chest wall at this point (fig. 1).

Two explanations have been offered for the relatively accentuated apical T₁ in atrial septal defect: 1. The large left-to-right shunt, bringing about an increase of tricuspid valve flow over mitral valve flow, keeps the tricuspid valve cusps in the position found in rapid right ventricular filling throughout diastole. Thus the velocity of tricuspid valve closure with the onset of systole is rapid and results in an unusually loud sound relative to that of mitral valve closure. M₁, on the other hand,
is relatively soft because the period of rapid left ventricular filling is over before end-diastole and the mitral valve cusps float together prior to systolic closure. 2. The apex area in this condition may overlie the right ventricle; hence, T₁ is better transmitted to this area than is M₁. This study permits no judgment as to the relative merits of these two hypotheses.

There are few other known causes for T₁ being louder than M₁ at the apex, and these are very uncommon. A delayed and accentuated T₁ would be an expected feature of tricuspid stenosis but this is rare as an isolated lesion. Delay and accentuation of T₁ have also been reported in cases of myxoma of the right atrium.¹⁵

The present phonocardiographic analysis shows that T₁ louder than M₁ at the apex is a rare finding (three in 168 cases), in a miscellaneous group of individuals from which suspected or proved cases of atrial septal defect have been separated. Further, there was not one proved or suspected case of atrial septal defect in the group of 57 cases in which M₁ was louder than T₁ at the apex. It is not surprising that there was one suspected atrial septal defect in the 89 cases with a single S₁, and one suspected and two proved cases of the 23 in the T₁ = M₁ group. "Borderline" cases are to be expected and impose a limitation on the usefulness of measurements such as this, where there must be a continuum between the definitely normal and the definitely abnormal.

In 15 of the 19 proved or suspected cases of atrial septal defect, T₁ was louder than M₁ at the apex. The diagnosis of uncomplicated atrial septal defect or at least of left-to-right shunt at the atrial level can be made with confidence when the clinical and phonocardiographic features are characteristic and T₁ louder than M₁ was part of a typical phonocardiographic picture in five of the seven suspected cases and in seven of 12 proved cases of atrial septal defect. More significant is the fact that in an additional three proved cases, T₁ louder than M₁ at the apex was the striking feature in the phonocardiogram, whereas

**Figure 3**

Phonocardiogram of a proved case of atrial septal defect. At PA, an ejection systolic murmur (SM) and a moderately wide splitting of S₂ (0.04 second in expiration) are seen. The pulmonic component (P) is louder than the aortic (A). At MA, S₁ is split with a variable relationship between the intensity of M₁ and T₁.

**Figure 4**

Typical case of atrial septal defect (proved at catheterization and surgery). Electrocardiogram has a 60-cycle interference. At PA (above) an ejection systolic murmur and moderately wide splitting of S₂ (0.05 second) are seen. At MA, (below), T₁ is louder than M₁.

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the rest of the clinical and phonocardiographic picture was uncharacteristic.

In contrast to the other possible causes mentioned above, leading to $T_1$ being louder than $M_1$ at the apex, uncomplicated atrial septal defect is one of the most common congenital heart defects and is seen and has been surgically corrected in patients of all age groups from the pediatric to the geriatric.

The phonocardiographic findings in atrial septal defect, though often characteristic, are variable, and numerous auscultatory phenomena have been described in association with this lesion, including abnormal splitting of the first and second heart sounds, ejection sounds, opening snaps and third and fourth heart sounds. Ejection and regurgitant-type systolic murmurs and early and mid-diastolic murmurs have also been described. It is this variability of findings that gives added significance to the character of the split first heart sound at the apex. The two most established and clinically valuable features of atrial septal defect (wide and fixed splitting of the second heart sound and incomplete right bundle-branch-system block) are not present.
in all cases. The phonocardiographic finding of an apical first heart sound with T1 greater than M1 is uncommon enough so that its presence should alert one to the possibility of an atrial septal defect even in the absence of other typical clinical and phonocardiographic findings.

Summary

The phonocardiograms of 187 patients were reviewed. In 89 the apical first heart sound was soft or single; in 57 M1 was louder than T1 and in 23 the two components were equal. In only 18 cases was T1 louder than M1 at the mitral area, and of this group five were suspected of having atrial septal defect and 10 were proved cases. In three of the proved cases, this was the only significant finding, the other usual features being absent. Only two proved cases of atrial septal defect in this series did not have T1 louder than M1 at the apex; both had T1 equal to M1. One case suspected of having atrial septal defect also had T1 equal to M1 and in another suspected case the first heart sound was single. This unusual characteristic of the first heart sound may be useful as an indication favoring the diagnosis of uncomplicated atrial septal defect.

Acknowledgment

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