Role of the Phonocardiogram in Evaluation of the Severity of Mitral Stenosis and Detection of Associated Valvular Lesions

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SURGICAL TREATMENT has increased the life expectancy of patients with mitral stenosis and has favorably modified the natural course of the disease.\textsuperscript{1-5} Hence, there is a tendency to operate on patients without serious disability in order to prevent or delay the complications of systemic and pulmonary emboli, pulmonary hypertension, congestive heart failure, and atrial fibrillation. However, the long-term follow-up of patients after mitral valvotomy has established that the procedure usually benefits for a limited number of years only.\textsuperscript{6} With the passage of time, an increasing number of patients develop restenosis or other complications requiring repeated operations on the mitral valve, even when the initial procedure appeared technically adequate.\textsuperscript{7}

The difficulty in selecting the time, both for the first operation and the repeated operations, emphasizes the need for frequent studies of patients with mitral stenosis by means of objective diagnostic techniques.

Objective tests for evaluation of severity of mitral stenosis are needed for two reasons: (1) Subjective symptoms may not provide the most reliable guide to the severity of mitral stenosis. Significant obstruction of the mitral valve may cause severe hemodynamic abnormalities in patients who have no appreciable symptoms.\textsuperscript{8,9} Pulmonary edema and other serious life-threatening complications of the disease may occur suddenly and unexpectedly. (2) A thorough evaluation of the pathology and function of the mitral valve and of other cardiac structures facilitates the choice of the most appropriate surgical technique for correction of mitral stenosis. There is an increasing tendency to employ exclusively open-heart surgical technique for correction of mitral stenosis. However, successful valvotomy can often be performed more simply without use of cardiac bypass. The attractiveness of closed procedures has been enhanced by the availability of standby oxygenators.

The most precise method used to evaluate the severity of mitral stenosis and associated valvular lesions is cardiac catheterization with angiocardiography. In many patients, however, an accurate anatomic and functional diagnosis can be established without hemodynamic studies with the help of the history and physical, radiological, electrocardiographic, and phonocardiographic examinations.

The phonocardiogram (PCG) provides an objective graphic record of auscultation and accurate timing of the closure and opening of the mitral valve. In previous studies the interval from the onset of the QRS to the sound of closure of the mitral valve (Q-1), the interval from the aortic valve closure sound to the opening snap of the mitral valve (2-OS), and the difference between these two intervals (Q-1) – (2-OS) have been correlated with left atrial pressure and with mitral valve area.\textsuperscript{10-18} Some investigators found these correlations good,\textsuperscript{10-16} others found them poor.\textsuperscript{17-18} Since the overall results were inconclusive, an additional study of the role of the phonocardiogram in evaluation of the

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severity of mitral stenosis and detection of associated valvular lesions appeared justifiable.

We concluded that the phonocardiogram can be a valuable aid in differentiating patients with severe mitral stenosis from those with mild mitral stenosis. We also determined certain factors which may explain the variability of the correlations between hemodynamic and phonocardiographic measurements reported by others.

Methods

We studied 50 male and 91 female patients, from the University of Kentucky Hospital, with rheumatic heart disease in whom mitral stenosis was the sole or dominant lesion as proved by cardiac catheterization, operation, or both. The patients' ages ranged from 13 to 62 years with a mean age of 36 years. Seventy-four patients had pure mitral stenosis and 67 patients had mitral stenosis with associated valvular lesions. Cardiac catheterization and operation upon the mitral valve were performed in 89 patients, cardiac catheterization alone was performed in 23, and operation alone in 29 patients.

The amplitude of the maximal vibration of the first sound at the apex was expressed as a percentage of the amplitude of the maximal vibration of the second sound in the same cycle. The amplitude of the pulmonary valve closure sound (P2) recorded in the second intercostal space to the left of the sternum was expressed as a percentage of the corresponding amplitude of the aortic valve closure (A0). All amplitude measurements were made in 10 to 15 consecutive cardiac cycles and were then averaged.

Right and left heart catheterization was carried out in a routine manner. Pulmonary wedge pressure was used as an estimate of left atrial pressure. Left ventricular pressure was obtained by retrograde catheterization by way of the brachial artery. Cardiac output was determined by the Fick principle. The mitral valve area was calculated by the formula of Gorlin and Gorlin.\textsuperscript{19} The diagnosis of valvular insufficiency was based on selective angiography and typical pressure tracings. Exercise was performed in the supine position on a bicycle ergometer. Cardiac output during exercise was expressed as a percent of predicted normal output.\textsuperscript{20}

The area of the mitral orifice was estimated by palpation during operation by one of us (F.C.S.). This surgeon estimates that the cross-sectional area of the middle of the terminal phalanx of his right index finger is 1.2 cm\textsuperscript{2}. When the surgeon was not able to insert his index finger beyond the middle of the terminal phalanx through the valve orifice, the mitral valve area was considered to be less than 1.2 cm\textsuperscript{2} and stenosis was judged severe. Careful records of the presence or absence of calcification of the mitral valve, of clots in the left atrium, and of mitral insufficiency as well as of other associated valvular lesions were made by the surgeon.

Phonocardiograms were recorded simultaneously with electrocardiographic leads I or II on a three-channel or four-channel direct writing recorder* at a paper speed of 50 or 100 mm per second. The phonocardiograph simultaneously recorded tracings within three frequency bands: 0 to 50 (low), 20 to 200 (medium), and 100 to 800 (high) cycles per second (cps). The nominal frequencies of these bands were 25, 100, and 250 cps. The Q-1 and 2-OS intervals were measured in cardiac cycles with the R-R interval closest to 0.8 second within a range of 0.75 to 0.85 second. When needed, exercise or carotid massage was used to increase or decrease the heart rate to obtain the desired R-R interval. The Q-1 interval was measured from the onset of the QRS complex to the first high amplitude vibrations of the first sound. This vibration was usually synchronous in the tracings recorded within all three frequency ranges. In the case of slight asynchrony, the vibration recorded in the tracing within the high frequency range was chosen. The 2-OS interval was measured from the first high frequency vibration corresponding to the closure of the aortic valve to the peak of the first vibration of the opening snap. The Q-1 and 2-OS intervals were measured from the phonocardiogram recorded at the point of maximal intensity of the opening snap, usually between the third and fifth intercostal spaces midway between the left sternal border and the apex. We excluded from the study patients in whom the QRS duration exceeded 0.09 second and patients in whom the systolic blood pressure exceeded 150 mm Hg. Phonocardiograms were recorded prior to cardiac catheterization. All measurements were made by one of us (B.S.) before the results of cardiac catheterization or operation were known. Standard statistical methods for evaluation of the significance of the results were employed.

Results

Signs of Mitral Stenosis

The diastolic murmur of mitral stenosis was absent in only two of 141 patients. Both were men with associated mild aortic stenosis. One was 40 years old and the other 50. Although these two patients had no audible opening

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\textsuperscript{*}Elema-Schoenander.
snap, mitral stenosis was diagnosed prior to catheterization from the history, electrocardiogram, and calcification of the mitral valve.

The opening snap (OS) was absent in 41 patients (29%). There was a significant correlation between absence of the OS and calcification of the mitral valve ($P < 0.01$). In the group with pure mitral stenosis without calcification, the OS was absent in only six patients (10%) of whom three were children (table 1). In three adults without opening snap, the stenosis was severe, and the heart rate exceeded 120 beats per minute. Because of tachycardia the opening snap could not be differentiated with confidence from the second component of the second sound. Later, when the heart rate was slower, the opening snap was clearly discernible in two of these patients.

When the mitral valve was calcified, the opening snap was absent in 50% of the patients with pure mitral stenosis and in 73% of the patients with associated mitral insufficiency or aortic stenosis. In patients without calcification but with associated aortic stenosis or mitral insufficiency, the incidence of an absent OS was greater than in patients with pure mitral stenosis (table 1).

### Table 1

**Absence of Opening Snap**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Total patients</th>
<th>Absent OS Patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure MS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No calcification</td>
<td>62</td>
<td>6*</td>
<td>10</td>
</tr>
<tr>
<td>Calcification</td>
<td>12</td>
<td>6</td>
<td>50</td>
</tr>
<tr>
<td>MS with associated lesions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No calcification</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AI</td>
<td>17</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>AS</td>
<td>7</td>
<td>4†</td>
<td>57</td>
</tr>
<tr>
<td>MI</td>
<td>13</td>
<td>3</td>
<td>23</td>
</tr>
<tr>
<td>Calcification</td>
<td>30</td>
<td>22</td>
<td>73</td>
</tr>
<tr>
<td>Total</td>
<td>141</td>
<td>41</td>
<td>29</td>
</tr>
</tbody>
</table>

See text for explanation of table.

Abbreviations: MS = mitral stenosis; AI = aortic insufficiency; AS = aortic stenosis; and MI = mitral insufficiency.

*Three patients less than age 17 years.
†One patient aged 14 years.

### Estimate of Severity of Mitral Stenosis

The $(Q-1) - (2-OS)$ interval was termed the "PCG index of severity of mitral stenosis."$^{10}$ The PCG index ranged from $-3$ to $+6$. The more positive the index, the more severe the stenosis that might be expected. We found no significant correlation between the PCG index and the cardiac output at rest, the diastolic pressure gradient across the mitral valve at rest, and the pulmonary artery pressure (PAP) at rest. However, there was a correlation of the PCG index with the diastolic pressure gradient across mitral valve during exercise ($P < 0.05$), PAP during exercise ($P < 0.05$), and cardiac output during exercise ($P < 0.01$).

The correlation between the PCG index and the mitral valve area (MVA) calculated from catheterization data was not sufficient ($P < 0.05$) to utilize the PCG index for accurate prediction of the MVA. However, the PCG index could be used to separate patients with severe stenosis from those with mild stenosis. We divided all the patients into two groups, one with a PCG index of +2 or more, and another with a PCG index of +1 or less. A PCG estimate was defined as correct when a patient in the first group had a MVA 1.2 cm² or less, and when a patient in the second group had a MVA greater than 1.2 cm². By this definition, the PCG index underestimated the MVA if a patient in the first group had a MVA greater than 1.2 cm². By the same definition, the PCG index overestimated MVA if a patient in the second group had a MVA 1.2 cm² or less.

The results of PCG correlation with the MVA calculated from catheterization data are similar to the results of PCG correlation with the MVA determined during operation (tables 2 and 3). In patients with pure mitral stenosis, the PCG index estimated the MVA correctly in 85%, underestimated it in 5%, and overestimated it in 10% of the cases. Of 42 operated patients, the PCG estimate was correct in 36 while in six the MVA was overestimated. The PCG index did not underestimate the MVA in any of the operated patients.

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Table 2

Correlation of (Q-1)-(2-OS) Interval with Mitral Valve Area in Fifty-three Patients with Pure Mitral Stenosis

<table>
<thead>
<tr>
<th>PCG estimate of MVA</th>
<th>Cardiac catheterization</th>
<th>Operation</th>
<th>Total correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct: +2 or more, MVA 1.2 cm² or less</td>
<td>25</td>
<td>36</td>
<td>61</td>
</tr>
<tr>
<td>Correct: +1 or less, MVA more than 1.2 cm²</td>
<td>6</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Correct: total</td>
<td>31 (84%)</td>
<td>36 (86%)</td>
<td>67 (85%)</td>
</tr>
<tr>
<td>Underestimated</td>
<td>4 (10%)</td>
<td>0</td>
<td>4 (5%)</td>
</tr>
<tr>
<td>Overestimated</td>
<td>2 (6%)</td>
<td>6 (14%)</td>
<td>8 (10%)</td>
</tr>
<tr>
<td>Total</td>
<td>37</td>
<td>42</td>
<td></td>
</tr>
</tbody>
</table>

See text for explanation of table. Of 53 patients, 16 had operation without cardiac catheterization, 10 had cardiac catheterization without operation, and 27 had both cardiac catheterization and operation.

Table 3

Correlation of (Q-1)-(2-OS) Interval with Mitral Valve Area in Twenty Patients with Predominant Mitral Stenosis and Associated Lesions*

<table>
<thead>
<tr>
<th>PCG estimate of MVA</th>
<th>Cardiac catheterization</th>
<th>Operation</th>
<th>Total correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct: total†</td>
<td>11 (61%)</td>
<td>6 (50%)</td>
<td>17 (57%)</td>
</tr>
<tr>
<td>Underestimated</td>
<td>4 (22%)</td>
<td>2 (17%)</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>Overestimated</td>
<td>3 (17%)</td>
<td>4 (33%)</td>
<td>7 (23%)</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>12</td>
<td>30</td>
</tr>
</tbody>
</table>

See text for explanation of table. Of 20 patients, two had operation without catheterization, eight had catheterization without operation, and 10 had both.

*Mitral insufficiency or aortic valve disease or both.
†See table 2.

The PCG estimate was less frequently correct in patients with associated mitral insufficiency or aortic valve disease. In this group, the PCG estimated MVA correctly in only 57% of all patients and in only six of 12 operated patients (table 3).

We also analyzed the Q-1 and the 2-OS intervals separately. To test the relation between the Q-1 interval and the MVA, the patients were divided into those with Q-1 intervals of 0.10 second or longer and those with Q-1 intervals shorter than 0.10 second. A PCG estimate was defined as correct when a patient in the first group had a MVA 1.2 cm² or less, and when a patient in the second group had a MVA greater than 1.2 cm².

To test the relation between the 2-OS interval and the MVA, patients were divided into those with 2-OS interval 0.08 second or shorter, and those with 2-OS intervals 0.09 second or longer. A PCG estimate was defined as correct when a patient in the first group had a MVA 1.2 cm² or less, and when a patient in the second group had a MVA greater than 1.2 cm².

In patients with pure mitral stenosis, the use of the PCG index and the Q-1 interval alone resulted in a greater number of correct estimates than the use of the 2-OS interval alone (table 4). The same was also true in patients with associated lesions who had cardiac catheterization.

The apparent superiority of the PCG index and the Q-1 interval over the 2-OS interval suggests that certain factors independent of the severity of mitral stenosis or of the left
atrial pressure alter the duration of the 2-OS interval more than the duration of Q-1 interval. One such factor could be the stroke volume. Weissler and associates\textsuperscript{21} demonstrated that, in patients with mitral stenosis, the duration of ejection decreases with decreasing stroke volume. At the same time, the decreased stroke volume is expected to decrease the rate of left ventricular relaxation.\textsuperscript{22} Thus, a shorter ejection time could produce an earlier closure of the aortic valve and a longer 2-OS interval. We compared the PCGs of patients with different cardiac outputs in a selected group of 16 patients with mean left atrial pressures within a narrow range of 15 to 20 mm Hg. Eleven patients had pure mitral stenosis, four had associated aortic insufficiency, and one associated mitral insufficiency. The cardiac index in this group ranged from 1.5 to 3.8 L/min/m\textsuperscript{2} the Q-1 interval from 0.08 to 0.12 second, the 2-OS interval from 0.06 to 0.10 second, and the PCG index from 0 to +6. The wide range of PCG intervals emphasizes the poor correlation between these intervals and left atrial pressure.

Figure 1 demonstrates that the 2-OS increases when cardiac output is low which suggests that the stroke volume might indeed be of importance in modifying the 2-OS interval in patients with the same left atrial pressure. While the stroke volume changes could have accounted at least partly for the variability of the 2-OS interval, there was no evidence that this factor had a predictable effect on the Q-1 interval or the PCG index. The reasons for the variability of the latter intervals in patients with the same left atrial pressure were not apparent.

We found no significant correlation of the amplitude of the first sound with MVA, left atrial pressure, diastolic pressure gradient across mitral valve, PAP, or cardiac output either at rest or during exercise. Of 38 patients in whom the amplitude of the first sound exceeded 300\% of the amplitude of the second sound, only four had mitral insufficiency. Of nine patients with first sound amplitude lower than the amplitude of the second sound, five had associated mitral insufficiency. However, these five patients represented only 14\% of all patients with mitral insufficiency. All four patients without mitral insufficiency in whom the amplitude of the first sound was lower than the amplitude of the second sound had tricuspid insufficiency.

**Estimation of Pulmonary Artery Pressure (PAP)**

The amplitude of the pulmonary valve closure sound (P\textsubscript{2}) was expressed as a percentage of the amplitude of the aortic valve closure sound (A\textsubscript{2}). There was no correlation between the amplitude of P\textsubscript{2} and the systolic or mean PAP at rest or during exercise. In
27 patients with a mean PAP of 25 mm Hg or less, the amplitude of $P_2$ averaged 127%, and in 17 patients with a mean PAP of 50 mm Hg or more the amplitude of $P_2$ averaged 150%. This difference was not significant. However, when the patients were divided on the basis of height and weight into types of body build, the amplitude of $P_2$ was significantly higher in slender patients ($P < 0.01$) than in those of medium or heavy build. A slender body build therefore related better to a loud $P_2$ than did pulmonary hypertension.

**Significance of Apical Systolic Murmur (Table 5)**

Thirty-five of 74 patients with “pure” mitral stenosis had a systolic murmur at the apex. In 29, the murmur occupied less than half of a systole. Only one of these patients had a pansystolic murmur and this was recorded when the heart rate was rapid.

All 33 patients with mitral insufficiency had apical systolic murmurs but only four had murmurs of short duration. Whether the short murmur in “pure” mitral stenosis reflects mitral or tricuspid regurgitation too small to be detected by angiography or surgical exploration or both is not clear. Yet, it is clear that detectable mitral insufficiency is usually associated with a long apical systolic murmur. All patients with tricuspid insufficiency or aortic stenosis had apical systolic murmurs. The murmur of tricuspid insufficiency was frequently of maximum intensity at or near the apex.

**Discussion**

An apical diastolic murmur is audible and recordable in nearly all patients with mitral stenosis. The murmur was absent in two of the 160 patients of Baker and Musgrave,23 in two of the 184 patients of Malers and Samuelsson,24 and in two of the 141 patients studied by us. These results are difficult to reconcile with series in which the reported incidence of absent murmur was 10%25 or even as high as 23%.11 Factors which could contribute to the absence of the murmur of mitral stenosis have been reviewed recently.24, 26 Both patients with “silent” mitral stenosis in the present study had associated mild aortic stenosis, low cardiac output, and heavily calcified mitral valves.

The incidence of absent opening snap in our patients was comparable to that in other larger series.27, 28 Our study indicates that an absent opening snap in an adult patient with mitral stenosis is nearly always associated with calcification of the mitral valve or the presence of another valvular lesion. In the presence of a calcified mitral valve, the opening snap was absent in 67% of all patients and in 50% of patients with pure mitral stenosis. This is comparable to the incidence of 52% found by Wynn.29 However, the opening snap was absent in nearly all patients with calcified mitral valves studied by Davies and Bucky.30 The discrepancy may be due to the fact that we made no attempt to separate patients with calcification limited to the annulus from patients with calcified leaflets. The opening snap is more likely to be preserved when calcification is limited to the annulus.

In patients without calcification, the opening snap was frequently absent in the presence

**Table 5**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Total patients</th>
<th>Duration of murmur (no. of patients)</th>
<th>Total with murmurs</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pansystolic</td>
<td>More than ½ systole</td>
<td>½ systole or less</td>
</tr>
<tr>
<td>Pure MS</td>
<td>74</td>
<td>1</td>
<td>5</td>
<td>29</td>
</tr>
<tr>
<td>Assoc. MI</td>
<td>33</td>
<td>25</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Assoc. TI</td>
<td>15</td>
<td>7</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Assoc. AS</td>
<td>6</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Assoc. AI</td>
<td>13</td>
<td>—</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>141</td>
<td>35</td>
<td>16</td>
<td>44</td>
</tr>
</tbody>
</table>

For abbreviations see table 1; TI = tricuspid insufficiency.
of associated aortic stenosis or mitral insufficiency. Associated aortic insufficiency apparently did not contribute to the absence of an OS. In our patients with associated mitral insufficiency, the OS was absent less frequently than in the patients studied by Wood and by Hultgren and Leo.

The duration of the Q-1 and 2-OS intervals in patients with mitral stenosis is a function of the duration of the cardiac cycle. Accordingly, whenever these intervals are measured, the heart rate is usually taken into consideration. It has been customary to report an average of intervals measured in several cycles or to use for measurements cycles of about 0.8-second duration, corresponding to heart rates of 75 beats per minute. However, even workers using the same duration of cardiac cycle report variable ranges of Q-1 intervals in patients with mitral stenosis. We tabulated some of the measurements of Q-1 and 2-OS intervals made in studies of apparently similar groups of patients. Nearly all reported Q-1 intervals fell into two large general categories: long and short (table 6). The longer intervals have a minimum duration of 0.07 to 0.08 second, a maximum of 0.12 to 0.14 second, and an average of 0.10 second. The shorter intervals have a minimum duration of 0.04 to 0.06 second, a maximum of 0.09 to 0.11 second, and an average of about 0.08 second. Our intervals are in the category of longer values and are in agreement with values reported by Leo and Hultgren, and Steinzeig and associates.

A wide range of Q-1 intervals in patients with mitral stenosis is found by all investigators. It is attributed not only to variations in left atrial pressure but also to variability of the electromechanical interval and the velocity of the left ventricular pressure rise. However, these factors do not explain the large differences in absolute values of the Q-1 interval found by various investigators.

Table 6
Duration of Q-1 and 2-OS Intervals in Selected Studies

<table>
<thead>
<tr>
<th>Author and patients</th>
<th>Lesion</th>
<th>Patients</th>
<th>Q-1 (in 0.01 sec)</th>
<th>2-OS (in 0.01 sec)</th>
<th>Cycle (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leo and Hultgren</td>
<td>Pure MS</td>
<td>20</td>
<td>8-12</td>
<td>10.4</td>
<td>6-11</td>
</tr>
<tr>
<td>Hultgren and Leo</td>
<td>MS + MI</td>
<td>20</td>
<td>8-11.5</td>
<td>9.6</td>
<td>5.9-10.6*</td>
</tr>
<tr>
<td>Steinzeig et al.</td>
<td>Pure MS</td>
<td>16</td>
<td>8-13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lee et al.</td>
<td>Pure or dominant MS</td>
<td>75</td>
<td>7-12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Di Bartolo et al.</td>
<td>Pure or dominant MS</td>
<td>33</td>
<td>5-13.5</td>
<td>8.8</td>
<td>3-15</td>
</tr>
<tr>
<td>Granath</td>
<td>Pure or dominant MS</td>
<td>59</td>
<td>4-11</td>
<td>8.1</td>
<td>4-11</td>
</tr>
<tr>
<td>Kelly</td>
<td>Pure MS</td>
<td>75</td>
<td>4-10</td>
<td>9</td>
<td>4-8†</td>
</tr>
<tr>
<td>Weissler et al.</td>
<td>Pure or dominant MS</td>
<td>10</td>
<td>6.5-10</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Proctor et al.</td>
<td>Pure or dominant MS</td>
<td>49</td>
<td>5-9</td>
<td>7</td>
<td>5-12</td>
</tr>
<tr>
<td>Mounsey</td>
<td>Dominant MS</td>
<td>28</td>
<td>3-14</td>
<td>7</td>
<td>Not stated</td>
</tr>
<tr>
<td>Bayer et al.</td>
<td>Pure or dominant MS</td>
<td>100</td>
<td>3-10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>This study</td>
<td>Pure or dominant MS</td>
<td>73</td>
<td>8-14</td>
<td>10.6</td>
<td>4.5-11</td>
</tr>
</tbody>
</table>

*In 10 patients.
†In 14 patients.
‡Determined by intracardiac phonocardiography.
The duration of Q-1 interval will be decreased if the interval is measured in a lead where the initial part of QRS is isoelectric. However, it seems improbable that the incidence of error due to this factor should vary significantly among different groups of patients.

Differences in identification of the first sound are probably of greatest importance. Recently Tavel and associates\textsuperscript{34} stated that the first heart sound was easy to identify. We agree with this statement although sometimes the first deflection of high amplitude used for the measurement of Q-1 interval is slightly asynchronous in tracings simultaneously recorded within different frequency ranges. In our study, the first deflection of high amplitude in the tracing recorded within the high frequency range was used for the measurement of the Q-1 interval. If we had used the corresponding deflection in a tracing recorded within lower frequency ranges, the Q-1 interval might have been slightly shorter. However, the asynchrony of the initial deflection of high amplitude was present in only a few patients. Therefore, it appears unlikely that differences in the characteristics of the recording systems employed in different studies could account for large differences in the timing of the first sound.

We considered the possibility that, in some cases, we have mistakenly identified the sound of tricuspid valve closure as the first sound. In patients with mitral stenosis, the sound of tricuspid valve closure may occasionally precede the sound of mitral valve closure.\textsuperscript{35, 36} However, in all patients without tricuspid stenosis studied by Hultgren\textsuperscript{35} and Dalla-Volta and associates\textsuperscript{36} the Q-T\textsubscript{1} interval was shorter than our shortest Q-1 interval. Moreover, the sound of tricuspid valve closure is of low amplitude, and when recordable, has its maximum amplitude at the left sternal border rather than at the apex.\textsuperscript{36}

The range of the reported 2-OS intervals is more uniform than the range of Q-1 intervals and the average values are nearly the same in all studies (table 6). This suggests that, in patients with mitral stenosis, the sounds of aortic valve closure and of mitral valve opening are easier to identify than the onset of the first sound. Unfortunately, the 2-OS interval served us less well for identification of “tight” mitral stenosis than the Q-1 interval or the PCG index.

In patients without associated lesions, the PCG index nearly always correctly predicted “tight” mitral stenosis. The MVA was 1.2 cm\textsuperscript{2} or less in 36 of 39 patients with a PCG index ranging from +2 to +6. In patients with associated valvular lesions, a PCG index of more than +2 was less reliable. The MVA was 1.2 cm\textsuperscript{2} or less in only 10 of 16 patients with PCG indices of this order.

Proctor and co-workers\textsuperscript{18} found a poor correlation between PCG index and MVA. In their patients with pure mitral stenosis, the correlation appeared similar to that in our patients with associated lesions. The duration of the 2-OS intervals was approximately the same in their study as in ours. However, the Q-1 interval in their study ranged from 0.05 to 0.09 second and in our study from 0.08 to 0.14 second. This may explain the different correlations in these two studies.

Our results are in good agreement with those of Craig\textsuperscript{13} who found that the MVA was always less than 1.2 cm\textsuperscript{2} when the PCG index was +2 or greater. A good correlation between the PCG index and the MVA was also reported by Wells.\textsuperscript{10} In his study, a MVA of 1 cm\textsuperscript{2} or less was found in patients whose PCG indices ranged from −1 to +5. These findings are in partial disagreement with our results since in half of our patients with PCG indices ranging from −1 to +1, the MVA was greater than 1.2 cm\textsuperscript{2}. We have no obvious explanation for this discrepancy in patients with a PCG index of −1 to +1. However, we have indirect evidence that patients with a PCG index within this range frequently have a MVA exceeding 1.2 cm\textsuperscript{2}. We found a PCG index of this magnitude in several patients after valvotomy where the surgeon could insert freely two fingers through the mitral valve. Hultgren and Hubis\textsuperscript{37} studied phonocardiograms of patients with a Starr-Edwards ball-valve prosthesis and concluded that the closing click (CC) and the opening
click (OC) of this valve correspond to mitral closure and the mitral opening snap in their relation to the cardiac cycle. The estimated effective area of the Starr-Edwards mitral valve is about 2.5 cm.² The interval (Q-CC) − (2-OC) ranged from −6 to +1.5 in patients studied by Hultgren and Hubis³⁷ and from −5 to +1 in patients with prostheses studied by us.

The more positive the PCG index, the greater the likelihood of a small MVA. However, one should not expect a sharp cutoff separating all patients with “tight” stenosis from patients with lesser grades of stenosis. We found that a PCG index of +2 best separated patients with MVAs less or greater than 1.2 cm.² This means that only a small number of patients with a PCG index of +2 or greater had a MVA greater than 1.2 cm.² However, a number of patients with a PCG index less than +2 had a MVA less than 1.2 cm.²

The usefulness of the PCG index is not limited to the preoperative evaluation of patients with mitral stenosis. The opening snap remains after valvotomy in most patients. Measurement of the PCG index helps to assess the results of operation (fig. 2) and may contribute to the detection of restenosis.

Our study failed to disclose a significant correlation between the PCG index and left atrial pressure or other hemodynamic measurements at rest. However, there was a correlation of the PCG index with the left atrial pressure, the PAP pressure and the cardiac output during exercise. The reasons for better correlation with hemodynamic measurements during exercise than at rest are not obvious.

The intensity of the first sound bore no relation to the severity of mitral stenosis. This agrees with the conclusion that Wood reached.² Wood felt that a very loud first sound rules out mitral insufficiency. In some of our patients with associated mitral insufficiency, the amplitude of the first sound was very high.

The lack of correlation between the amplitude of P₂ and the PAP is not surprising. In our study the amplitude of P₂ was significantly greater in slender patients. Undoubtedly, a number of other factors, not evaluated in this study, could alter significantly the amplitude of P₂.

The incidence of apical systolic murmur in our patients with mitral stenosis is similar to that reported by Wood² and by Mounsey and Brigden.³⁸ Our study shows that the likelihood of mitral insufficiency increases with increasing duration of the apical systolic murmur. All patients with a pansystolic murmur studied by Mounsey and Brigden had associated mitral insufficiency. All but one patient with combined mitral stenosis and insufficiency studied by Hultgren and Leo³¹ had an apical pansystolic murmur. Some of our patients with mitral insufficiency had short systolic murmurs, but in these patients insufficiency was slight. Our study emphasizes that the pansystolic murmur of tricuspid insufficiency is frequently well transmitted to the apex.

**Summary**

The phonocardiogram was correlated with hemodynamic measurements and with the results of surgical exploration in 141 patients with pure or dominant mitral stenosis. The study revealed the following results:

1. A diastolic murmur at the apex was absent in only two of 141 patients.

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**Figure 1**

Correlation of 2-OS interval with cardiac index (C.I.) in 16 patients with left atrial pressure ranging from 15 to 20 mm Hg. Patients with pure mitral stenosis are identified by dots and patients with associated lesions by crosses. See text.

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2. The opening snap (OS) was absent in 29% of all patients and in 10% of patients with pure mitral stenosis and noncalcified mitral valve. The most important single factor contributing to the absence of an OS was calcification of the mitral valve. Other factors included young age and associated aortic stenosis or mitral insufficiency.

3. There was no significant correlation between the \((Q-1)-(2-OS)\) interval (PCG index) and the diastolic pressure gradient across mitral valve at rest. The correlation between the PCG index and the calculated mitral valve area (MVA), and the diastolic pressure gradient across the mitral valve during exercise was significant \((P < 0.05)\).

4. The PCG index correctly predicted whether the MVA was smaller or greater than 1.2 cm\(^2\) in 85% of patients with pure mitral stenosis but in only 57% of patients with associated lesions. The MVA was less than 1.2 cm\(^2\) in 36 of 39 patients with pure mitral stenosis in whom the PCG index ranged from +2 to +6. Of 42 operated patients with pure mitral stenosis, the PCG estimate was correct in 36. The PCG index did not underestimate MVA in any of the operated patients.

5. In a group of patients with the same left atrial (LA) pressure, the 2-OS interval was longer in patients with low cardiac output. The relation between the 2-OS interval and the cardiac output could explain in part the lack of correlation between LA pressure and 2-OS interval and the smaller number of correct estimates of MVA made by the 2-OS interval as compared to the Q-1 interval and the PCG index.
6. There was no significant correlation between the amplitude of the first sound at the apex and the severity of mitral stenosis. Several patients with associated mitral insufficiency had a first sound of high amplitude.

7. There was no significant correlation between the amplitude of the sound of pulmonary valve closure (P2) and the pulmonary artery pressure. P2 amplitude was significantly greater in slender patients.

8. An apical systolic murmur was recorded in 48% of patients with pure mitral stenosis. The longer the murmur, the greater was the likelihood of associated mitral insufficiency. A pansystolic murmur at the apex usually indicated an associated mitral or tricuspid insufficiency.

References


50 Years Ago—Lutembacher’s Syndrome

Role of the Phonocardiogram in Evaluation of the Severity of Mitral Stenosis and Detection of Associated Valvular Lesions

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