The Ballistocardiogram in Mitral Stenosis

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The ballistocardiograms of 13 of 14 patients with “pure” mitral stenosis were found to show a characteristic late diastolic and early systolic deformity. The deformity consists of a footward wave which precedes the I wave, often resulting in “doubling” of the I, or fusion with the I, producing a wide, deformed wave. Ballistocardiograms of patients with mitral insufficiency did not consistently show this deformity. Immediately after commissurotomy this abnormality was diminished, but was not completely eliminated. Some possible factors concerned in the genesis of this pattern are discussed.

Despite the extensive application of the ballistocardiographic method to the study of various disorders of the cardiovascular system, little attention has been given to the findings in the presence of rheumatic mitral stenosis. In 1941, Starr\(^1\) reported several cases of rheumatic heart disease, with emphasis placed upon the changing “size” and improved form of the record after the administration of digitalis. He did not comment on the characteristics of the pattern, but it is of interest to note that the illustrated records from these individuals did show a deformity of the I wave, at least to some extent. In 1948, when reviewing a large variety of cardiovascular disorders, Starr and Mayock found abnormal ballistocardiograms in 45 per cent of their cases of rheumatic heart disease.\(^2\)

One case of mitral stenosis and insufficiency, reported in detail, was said to show abnormally small I waves, and slurred or notched IJ segments. On the other hand, Brown and his co-workers\(^3\) were impressed by the diastolic waves in cases of mitral stenosis. Thirty per cent were said to show tall L waves, and 30 per cent prominent N waves. These workers did not broaden their comments regarding ballistic form, except as related to arrhythmias or “significant . . . myocardial damage.” The studies of Starr and of Brown and their co-workers were carried out on high-frequency bed ballistocardiographs.

Mathers and associates, utilizing a Nickerson-type low frequency bed, reported two cases of rheumatic heart disease.\(^4\) Both had abnormal records, with “bowing” of the JK segment. Newman and his co-workers, likewise using a low-frequency instrument, recorded ballistocardiograms on dogs before and after the experimental induction of mitral insufficiency. No significant change in ballistic form was noted.\(^5\)

In several publications related to the use of the direct-body ballistocardiographic pick-up, Dock and his co-workers\(^6, 7\) have commented on the presence of prominent L waves in the records from patients with rheumatic carditis and “mitral valve disease.”

It is the purpose of this report to describe findings in clinically “pure” mitral stenosis, before and after commissurotomy.

**Methods and Materials**

All studies were performed on a high-frequency (9 or 10 cycles per second when loaded with 150 pounds of dead weight) Starr-type bed ballistocardiograph. Frontal plane “vector” records were taken by means of a method previously described.\(^8\) Electrocardiograms (lead II) and belt pneumograms (strain gage) were recorded simultaneously, and in many instances these were combined with apex cardiograms and phonocardiograms. The conditions under which these ballistocardiographic studies were performed, the methods of analysis, and normal standards on which comparisons are based have been reported elsewhere.\(^9\)
Fourteen patients with clinically “pure” mitral stenosis were studied. They were made available to us through the courtesy of Drs. E. Cowles Andrus and Alfred Blalock. All of them fulfilled the criteria previously reported from The Johns Hopkins Hospital for acceptability for commissurotomy.10-12 Cases with clinically significant mitral insufficiency were excluded from this group. Their ages ranged from 22 to 54, with a mean of 35 years. Twelve of the 14 cases were females (86 per cent). Functional disability was graded on the basis of the New York Heart Association Criteria.13 One case was considered class I, the others were class II or III. All were studied immediately prior to operation, and consequently maximal clinical improvement had been obtained through the usual means prior to this study. Minor evidences of congestive failure were present in several instances (elevated systemic venous pressure, slight hepatomegaly, basilar rales), but all patients were able to lie comfortably in the recumbent position for the period of this test.

Findings on physical examination were those of relatively pure mitral stenosis. Three of the 14 had evidence by x-ray of enlargement of the left ventricle, while in the remaining cases this chamber was considered normal. The left atrium and/or pulmonary artery was prominent in all cases. Pulmonary artery pressure, determined by cardiac catheterization or direct needle-puncture at the time of thoracotomy, was elevated in all instances in which the procedure was successful (11 of 14 cases). In five cases the electrocardiogram was interpreted as showing right ventricular “strain.” In the remaining cases the electrocardiogram was considered normal.

For comparison, eight cases of clinically determined mitral insufficiency (with or without stenosis) were selected from the Cardiac Clinic. Age and general clinical disability were comparable. The diagnosis of mitral insufficiency was based on the presence of a loud apical systolic murmur, with or without a thrill, and left ventricular enlargement. Patients with evidence of other valvular lesions were excluded.

In order to secure a comparable control group, the mean values for 18 normal females in the fourth decade were utilized. The group with mitral stenosis is not strictly comparable to the normal group, in that 2 of the 14 patients were males. In addition, ages are not entirely comparable, since although the average age of the group with mitral stenosis was 35 years, there were three individuals over 40, and three under 30 years.

Utilizing the electrocardiogram for timing, and measuring all intervals and durations of waves to the closest 0.01 second, the abnormal deflection and normal systolic waves were tabulated in each case.

Results

A. Ballistocardiographic Form in Mitral Stenosis

In accord with previous ballistocardiographic experiences, moderate variation in ballistic form was found from case to case. Despite the overall variability, one anomaly was strikingly consistent. In 13 of the 14 cases of mitral stenosis, a deformity of the late diastolic and early systolic portions of the complex was found. This is shown in the examples diagrammed in figure 1, and illustrated in figure 2. This abnormality consists of a presystolic headward wave, often broad and rounded, sometimes sharply inscribed and highly variable, which is followed by a much more consistent footward deflection which precedes the I, and follows the electrocardiographic Q by 0.10 second (±0.03 second). This footward wave, which is later than the normal G, deforms the H wave, decreasing its amplitude or totally eliminating it. The I wave is characteristically distorted, and its inscription delayed. The distortion of the I ranges from clearly defined “doubling,” that is, with the interfering anomaly and normal I separately inscribed, to fusion of these two footward waves, with a wide slurred “I” resulting. There are gradations of all degrees between these extremes.

In this series of 14 cases of mitral stenosis the J wave was usually normal. In two instances, the total ballistocardiographic pattern was grossly deformed, and in these the J wave was variously and inconsistently distorted. Six cases showed rounding and slurring of the expiratory J, although inspiratory form was normal. In the remaining six cases the J was normal in both phases of respiration. The K was likewise usually normal, except for the two totally deformed records, although an occasional expiratory complex was characterized by distortion of K. In only one instance was an unusually prominent L wave seen.

Mid- and late diastolic deflections were neither consistent nor striking, except for the presystolic deformity previously described.

The frontal plane “vector” studies were of
interest, since the distribution of the IJ loop differed from the normal. In normal adults the I is oriented footward and slightly to the right (as seen from the patient’s head in dorsal recumbency), while the J is headward and slightly to the left. In contrast to this pattern, the IJ axis in mitral stenosis is rotated slightly counterclockwise, that is, with the I oriented to the left and footward, the J to the right and headward. Fifty per cent of the records in this series revealed such a counterclockwise pattern, only one was clockwise, and the remainder were oriented along the head-foot axis.

Vector dissociation was that in several cases the deformity was much more striking, 60 degrees and 90 degrees clockwise from the head-foot axis, than in the conventional record, although clearly seen in the latter.

**B. Quantitative Data in Mitral Stenosis**

The complete quantitative data are summarized in table 1. The abnormal early footward deflection, which precedes the I wave, occurs an average of 0.097 second after the Q wave. If the one case with mitral stenosis in which this deformity did not occur is excluded, the mean interval from Q to this abnormal wave becomes 0.103 second. For contrast, this interval is compared statistically with the Q-G interval of normal controls (although this is not meant to imply that the origin of these deflections is similar). The mean Q-G in the controls is 0.044 second and the upper limit for this figure is 0.06 second. One record illustrates complexes in which “doubling” of the I wave (second diagram), and “fusion” of the I with the early deflection (third diagram) occur. Note the abnormal footward deflection occurring 0.010 second after the electrocardiographic Q wave, and 0.06 second after the normal ballistocardiographic G wave. The I wave appears late (0.195 sec. after the Q) and its duration is quite prolonged. (See text for details.)
the mitral stenotic group was found to have an interval of 0.06 second while all others exceeded this figure. Measurements of the Q-G and Q-I were performed with the assumption that the first headward deflection after the "interfering" footward wave is the H, and the subsequent footward deflection the I. figures are used, the mean Q-H interval is 0.153, which is 0.041 second longer than the mean Q-H interval in the control group. The mean Q-I is 0.195 second, or 0.020 second longer than in the normal females. These differences are statistically significant.

Of the remaining figures, only one was found

![Fig. 2. Representative samples of the ballistocardiograms of patients with mitral stenosis. The arrows identify the abnormal footward deflections distorting the early portion of the systolic complexes. A shows an extreme example of the phasic shift in deformity of the ballistic complex with respiration. In B this phasic influence is not apparent, although consistently doubled and deformed I waves are present. In C is shown the record from a patient with atrial fibrillation in which the characteristic deformity is present.]

Since this portion of the ballistocardiogram was distorted, the designation H and I may not be strictly applicable. Since 13 of 14 records were found to show the characteristic deformity, mean values were obtained for the total group, and also for the 13 patients in whom this finding was present. If the latter to show a significant difference between the mitral group and the controls. This figure is the I wave duration. In measuring this interval, both the early interfering wave and the normal I wave were included, since they were often fused or so nearly fused that the "H" wave failed to reach the baseline. There is highly
significant difference between the normal (0.055 second) and the mitral stenotic I duration (0.1231 second).

In order to attempt correlation between clinical functional status, pulmonary arterial pressure, and the ballistocardiographic form, these findings were charted. Clinical disability is graded into classes 1 to 4 (New York State Heart Association); the pulmonary arterial pressures are the average of several readings in millimeters Hg; the electrocardiogram is charted as showing a normal pattern or right ventricular strain. The ballistocardiographic pattern is classified 1 to 4, with class 1 representing an intermittently deformed early systolic component, class 2 a consistently present deformity, with separately inscribed waves ("doubled" I), class 3, "fused" I, that is, the fusion of the interfering deflection and the I, and class 4, gross abnormality, with deformity not only of early systolic but also of the J and K.

As is apparent from table 2, there is a general, but not entirely consistent, tendency for those individuals with increasing degrees of clinical disability and higher pulmonary artery pressure to show more marked ballistocardiographic abnormality.

Of the small group of eight patients with clinical mitral insufficiency, only one had a record which could have been considered compatible with the mitral stenotic pattern. Four cases showed an intermittently "doubled" I wave, but without the widening and delayed inscription present in mitral stenosis. The mean Q-I interval in the patients with mitral insufficiency is 0.173 second, which is not significantly different from that in the controls. The I is widened, being 0.076 second, but is significantly less than the 0.123 second duration found in the ballistocardiogram in mitral stenosis.

**Table 2.**--Tabulation of clinical features and degree of abnormality of the ballistocardiogram. (See text for explanation of ballistocardiogram grading system.) Note the general trend to increasing abnormality with increasing clinical disability and rising pulmonary artery pressure.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Functional Clinical Disability (Class 1-4)</th>
<th>Pulmonary Artery Pressure</th>
<th>Electrocardiographic Abnormality Right Ventricular Strain</th>
<th>Ballistocardiographic Abnormality (Class 1-4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N. B.</td>
<td>Class 1</td>
<td>?</td>
<td>absent</td>
<td>Class 1</td>
</tr>
<tr>
<td>M. B.</td>
<td>Class 2</td>
<td>?</td>
<td>absent</td>
<td>Class 1</td>
</tr>
<tr>
<td>A. B.</td>
<td>Class 2</td>
<td>?</td>
<td>absent</td>
<td>Class 1</td>
</tr>
<tr>
<td>E. J.</td>
<td>Class 2</td>
<td>49/27</td>
<td>absent</td>
<td>Normal</td>
</tr>
<tr>
<td>R. J.</td>
<td>Class 2</td>
<td>51/22</td>
<td>absent</td>
<td>Class 2</td>
</tr>
<tr>
<td>M. C.</td>
<td>Class 2</td>
<td>54/24</td>
<td>present</td>
<td>Class 2</td>
</tr>
<tr>
<td>J. W.</td>
<td>Class 2</td>
<td>55/37</td>
<td>?</td>
<td>Class 4</td>
</tr>
<tr>
<td>P. B.</td>
<td>Class 2</td>
<td>60/30</td>
<td>absent</td>
<td>Class 3</td>
</tr>
<tr>
<td>F. G.</td>
<td>Class 2</td>
<td>85/51</td>
<td>present</td>
<td>Class 4</td>
</tr>
<tr>
<td>M. H.</td>
<td>Class 3</td>
<td>40/22</td>
<td>absent</td>
<td>Class 2</td>
</tr>
<tr>
<td>G. B.</td>
<td>Class 3</td>
<td>50/43</td>
<td>absent</td>
<td>Class 4</td>
</tr>
<tr>
<td>L. E.</td>
<td>Class 3</td>
<td>82/47</td>
<td>present</td>
<td>Class 3</td>
</tr>
<tr>
<td>J. A.</td>
<td>Class 3</td>
<td>91/36</td>
<td>present</td>
<td>Class 3</td>
</tr>
<tr>
<td>E. C.</td>
<td>Class 3</td>
<td>138/57</td>
<td>present</td>
<td>Class 3</td>
</tr>
</tbody>
</table>

Postoperative ballistocardiograms were obtained in six cases, but unfortunately all were performed shortly after operation, and it is probable that at least six months will be required before optimal improvement is attained, following commissurotomy. All six cases showed satisfactory results immediately after operation. In general, the postoperative ballistocardiographic changes were not striking. Four cases showed some improvement, and in five the ballistic amplitude increased. The early deformity persisted to some degree in all cases although it was distinctly less prominent in
four. Figure 3 illustrates the most striking case, with an abnormal preoperative record. and an almost normal one following operation. The early systolic deformity is far less evident.

DISCUSSION

In the ballistocardiograms of many normal individuals there is a footward wave which precedes the I wave. This normal early deflection has been designated the "G" wave, and occurs simultaneous with or within 0.06 second after the electrocardiographic Q wave (0.06 second represents the upper limit for the mean Q-G interval, not for isolated complexes). This deflection shows phasic variation with respiration.

The ballistocardiogram in 13 of 14 cases of mitral stenosis has been shown to be characterized by an abnormal early systolic wave. In contrast to the normal G, this footward deflection occurs late (mean of 0.103 second after the Q) and is associated with a late I wave (mean Q-I 0.195 second).

The deformity seems to represent an abnormal footward impact, which occurs either in late diastole or early systole, distorting the early systolic portion of the ballistic complex, and producing a "doubled" I wave. When the deformity is further advanced, fusion of this deflection with the I occurs, producing a wide rounded I (mean I duration 0.129 second). This has not been observed in normal individuals. It has been seen in other disorders, especially constrictive pericarditis and aortic or iliac thrombosis.

The presence of a "splintered" or "doubled" I wave has been noted by several observers with the vertical ballistocardiograph. The second wave has been called I, by Krahl, who demonstrated that this impact varies in position during the respiratory cycle, occurring early in expiration, late in inspiration. A similar, though less definite respiratory influence is present in some cases of mitral stenosis. Starr has stated that his studies with the vertical ballistocardiographic technic demonstrated no significant differences in the timing or the duration of waves from the results with the horizontal instrument. Krahl suggests that the early wave, which occurs with the
vertical ballistocardiograph in normal persons, may be the result of deceleration of the blood ejected by the right ventricle, as it meets the pulmonary arch or peripheral pulmonary resistance. If such a factor were responsible, then by analogy one could postulate that the profound increase in pulmonary artery resistance and reduced elasticity in mitral stenosis accentuate this deceleration impact to a far more striking degree, and that this factor contributes to the development of the above described anomaly even on the horizontal bed.

Kuo and associates reported three cases in which “double peaked” systolic complexes were present. One of these was an individual with mitral stenosis. All showed two headward waves preceding the J wave, which would result in “doubling” of the I. These workers demonstrated the presence of ventricular asynchrony in all three cases, and suggested that this deformity occurs when pulmonary ejection precedes aortic, and is maximal in the presence of right ventricular hypertrophy. There is right ventricular hypertrophy in a large percentage of cases of mitral stenosis, but the significance of the asynchrony factor remains to be determined. Kuo and co-workers observed “two upward deflections” in two cases of mitral insufficiency as well.

The possible contribution of an intracardiac impact as an alternative or additional factor must be considered when attempting an explanation of the deformity found in mitral stenosis. The high pressure in the pulmonary circuit and left atrium and the normal or low left ventricular pressure suggest, on theoretical grounds at least, that an abnormal late diastolic—early systolic impact could develop during ventricular filling. It is clear that atrial systole is not a necessary requirement, since the deformity described occurs in the presence of atrial fibrillation as well as with normal sinus rhythm.

Thus the explanation for the delayed, widened, and deformed early systolic ballistocardiographic complex is not immediately forthcoming. Whether the increased pulmonary resistance, ventricular asynchrony, or atrioventricular pressure gradient contribute significantly, either individually or collectively, cannot be determined from the available data. Other factors may well be responsible.

It is clear, however, that in 13 of 14 cases of relatively pure mitral stenosis, a distinct, though not specific, deformity is present. Whether the presence of this pattern will be of value in differentiating individuals with stenosis from those with significant mitral insufficiency remains to be determined. This small series would suggest that if the fully developed pattern (class 3 or 4) described previously is present, the lesion is predominately stenotic, but larger groups of patients with both lesions are required before real significance can be attached to this finding.

Immediate postoperative results suggest that the deformity is lessened, though not eliminated by commissurotomy. Prolonged observation of the patients constituting this series will be necessary before final conclusions may be drawn with respect to changes in the ballistocardiogram following successful commissurotomy. It will also be of interest to determine in what degree long-term clinical improvement is reflected by ballistocardiographic improvement.

**Summary**

1. The ballistocardiographic findings in 14 individuals with “pure” mitral stenosis have been reported. Thirteen of the 14 were shown to have a consistent deformity of the early systolic portion of the ballistic complex.

2. Six cases were studied following satisfactory commissurotomy. The abnormal early systolic pattern was lessened, but not eliminated, in four.

3. The possible contributions of increased pulmonary vascular resistance, right ventricular hypertrophy, ventricular asynchrony, and abnormal atrioventricular pressure gradient are discussed.

4. The presence of a relatively consistent ballistic deformity in mitral stenosis is at the present time a physiologic, ballistocardiographic challenge, more than a finding with definite clinical usefulness.

**Sumario Español**

Los balistocardiogramas de 13 de 14 pacientes con estenosis mitral pura mostraron...
una deformidad característica tarde en diástole y temprano en sistole. La deformidad consiste en una ondulación caudal que precede la ondulación I, a menudo resultando en una doble I, o fusión con la I, produciendo una ondulación deformada. Ballistocardiogramas de pacientes con insuficiencia mitral no mostraron esta deformidad consistentemente. Inmediatamente después de comisurotomía esté abnormalidad disminuyó, pero no fue completamente eliminada. Algunos factores que posiblemente conciernen con el origen de esta abnormalidad se discuten.

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