Q-1 or C-1 Interval in the Diagnosis of Mitral Stenosis

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The delay of the first heart sound is a well-known characteristic sign of mitral stenosis. This phenomenon is due to increased left atrial pressure, delayed rise in left ventricular pressure (especially after a short diastole), and rigid mitral cusps (Wells, 1957). The delay in the first mitral sound is usually assessed by the transformation time (or Q-1 interval). But this interval has often been found to be of little diagnostic value. The Q-1 interval is not always prolonged in mitral stenosis (Chavez, Vaquero, and Mendoza, 1955; Rich, 1959; Maslyuk, 1959). No definite correlation has been established between the Q-1 interval and the mitral valve size (Donnelly, Maha, and Orgain, 1959; Hager et al., 1965), the pulmonary capillary pressure (Warembourg et al., 1964), the pulmonary artery pressure (Lukomsky, 1965), and the right ventricular pressure (Lee, Scherlis, and Singleton, 1965). There has been only a slight correlation between the Q-1 interval and both the left atrial pressure and the mitral atrio-ventricular gradient (Proctor et al., 1958). The Q-1 interval may be prolonged even in patients with pure mitral regurgitation (Sokolova, 1964).

To study the actual delay of the first mitral sound, the apex cardiogram was used, as a simple and correct method for recording the onset of the movements of the heart in systole. There is a very close correlation between the initial abrupt rise on the apex cardiogram and the initial rise of the left ventricular pressure curve (Tavel et al., 1965a). In this respect, the apex cardiogram is a direct method of recording, while the rise in ventricular pressure appears as a result of the muscular contraction and may show some lag in comparison with the apex cardiogram. On the average, the curve on the apex cardiogram starts to rise slightly before the pressure curve (Tafur, Cohen, and Levine, 1964), with an average difference of 7 ± 3 msec. (Tavel et al., 1965a).

This subject was discussed in detail in a previous work (Oreshkov, 1965). The use of the apex cardiogram makes it possible to exclude the so-called electromechanical delay, i.e. the time interval between the onset of the QRS and the onset of the ventricular contraction (Q-C interval; C from contraction—Tavel et al., 1965a) (Fig. 1). The interval between the onset of the systolic wave of the apex cardiogram and the first mitral sound represents the initial phase of ventricular contraction, before the atrio-ventricular valves close, which the author designated C-1 interval.

SUBJECTS AND METHODS

Studies were made on 57 women and 20 men with mitral valve disease; their ages ranged from 15 to 56 years. Of these 77 patients, 45 had mitral stenosis (Group I), 21 had predominant mitral stenosis (Group II), 4 had mitral stenosis and regurgitation, approximately of an equal degree (Group III), and 7 had pure or predominant mitral regurgitation (Group IV). In all patients the diagnosis was confirmed by cardiac operation (63 patients), by cardiac catheterization (13 patients), or by angiocardiography (1 patient). There were 2 control groups: 40 normal subjects, and 27 hypertensive patients.

Lead II of the electrocardiogram, a left ventricular apex cardiogram from the apex beat, and a medium frequency phonocardiogram (140 and 250 cycles per second) at the mitral area were recorded simultaneously. The tracings were made in left lateral decubitus position of the patient at the end of expiration or at the beginning of inspiration. For identification of the origin of the apex cardiogram (right or left apex beat) the configuration of the QRS complex from the point of maximal cardiac impulse was used. A pulse wave (linear) condenser microphone (Boucke-Brecht) for recording the apex cardiogram, and a piezo-electric microphone for recording the phonocardiogram were used, connected with a direct-writing multichannel recorder (Hellige, Model 9400/6). The records were taken at a paper speed of 50 mm/sec. with 20 msec. time lines. For each measurement a minimum of three consecutive
cycles was used. Where there was atrial fibrillation three (rarely less) separate cycles of approximately 800 msec. duration were chosen.

RESULTS

The results are given in Table I.

The electromechanical delay (Q-C interval) in the control groups varied between 20 and 60 msec. with an average of 28.5 msec. in the normal subjects, and in the patients with mitral valve disease between 10 and 70 msec., with an average of 29.0 msec. In the majority of all the subjects examined, this interval was found to be between 20 and 40 msec., which corresponds to the published values: mean 20 msec. (Hartman and Bullen, 1960), mean 35 msec. (Coughlan, Prieto, and Harrison, 1961), 10-40 msec. (Zuckermann, 1961), mean 23 msec. (Warembourc and Ducloux, 1963), 20-30 msec. (Laur, Voren- hoven, and Rotterdam, 1963), and mean 21 msec. (Tafur et al., 1964). In the patients with pure or predominant mitral regurgitations (Group IV) only, the mean value of the Q-C interval was considerably higher than in the other groups, which was mainly due to the unusually long interval (70 msec.) in one of the patients.

The initial phase of ventricular contraction (C-1 interval) normally was 30-5 (20-50) msec., and in the hypertensive patients—32-8 (10-60) msec. These values are close to those found by other authors: 44 (21-82) msec. (Coughlan and Epstein, 1963); 37 (0-50) msec.—“deformation phase” (Warembourc and Ducloux, 1963); mean 35 msec. —“pre-isometric phase” (Tafur et al., 1964). The maximum prolongation of the C-1 interval was found in Group I (mean 47-4 msec.) and Group II (mean 45-2 msec.). There was a good correlation between the duration of the C-1 interval and the long diameter of the mitral orifice (Tables I and II). In the patients with pure or predominant mitral regurgitation (Group IV) this interval was normal. Sixty-two per cent of the individual values of the

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**TABLE I**

<table>
<thead>
<tr>
<th>Groups*</th>
<th>No. of cases</th>
<th>Q-C (msec.)</th>
<th>C-1 (msec.)</th>
<th>Q-1 (msec.)</th>
<th>Q-1+c (msec.)</th>
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</thead>
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<tr>
<td></td>
<td>Range Mean S.D.</td>
<td>Range Mean S.D.</td>
<td>Range Mean S.D.</td>
<td>Range Mean S.D.</td>
<td>Range Mean S.D.</td>
</tr>
<tr>
<td>A Normal subjects</td>
<td>40</td>
<td>20-50 28.5 ±8 0</td>
<td>20-50 30.5 ±7 1</td>
<td>50-90 59.0 ±8.3</td>
<td>— — —</td>
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<tr>
<td>B Hypertensive patients</td>
<td>27</td>
<td>20-60 34.5 ±9 9</td>
<td>10-60 32.8 ±10.2</td>
<td>50-100 67.3 ±12.7</td>
<td>— — —</td>
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<tr>
<td>Group I (total)</td>
<td>45</td>
<td>10-50 27.6 ±6.9</td>
<td>30-65 47.4 ±8.1</td>
<td>55-100 75.0 ±9.4</td>
<td>56.0-101.6 74.1 ±10.7</td>
</tr>
<tr>
<td>Group Ia</td>
<td>16</td>
<td>10-50 28.1 ±8.9</td>
<td>35-65 50.9 ±7.4</td>
<td>70-100 79.1 ±5.0</td>
<td>63.0-101.6 78.5 ±10.6</td>
</tr>
<tr>
<td>Group Ib</td>
<td>20</td>
<td>10-40 26.25 ±5.4</td>
<td>40-60 47.7 ±6.8</td>
<td>60-90 74.0 ±9.4</td>
<td>56.0-92.0 72.3 ±10.8</td>
</tr>
<tr>
<td>Group Ic</td>
<td>7</td>
<td>20-40 28.6 ±6.3</td>
<td>30-60 41.4 ±9.3</td>
<td>55-90 70.0 ±11.2</td>
<td>57.0-86.0 70.8 ±10.5</td>
</tr>
<tr>
<td>Group Id</td>
<td>2</td>
<td>30-35 32.5 ±3.7</td>
<td>35-40 37.5 ±3.6</td>
<td>65-75 70.0 ±7.1</td>
<td>65.0-71.0 68.0 ±4.2</td>
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<td>Group II</td>
<td>21</td>
<td>10-40 27.9 ±7.0</td>
<td>30-60 45.2 ±7.3</td>
<td>60-90 73.1 ±7.2</td>
<td>54.4-90.0 73.7 ±8.0</td>
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<tr>
<td>Group III</td>
<td>4</td>
<td>25-40 32.5 ±6.5</td>
<td>30-50 41.3 ±8.5</td>
<td>70-80 73.7 ±4.8</td>
<td>64.0-79.8 74.3 ±7.2</td>
</tr>
<tr>
<td>Group IV</td>
<td>7</td>
<td>20-70 40.0 ±16.6</td>
<td>25-40 31.4 ±4.8</td>
<td>60-100 71.4 ±14.6</td>
<td>59.2-116.0 73.7 ±19.8</td>
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</table>

* Ia, diameter of mitral orifice 2-5 mm. 
  Ib, " 5.5-10 mm. 
  Ic, " 11-15 mm. 
  Id, " >15 mm. 

+c = corrected values to a standard heart rate of 75/min. (R-R = 800 msec.).
C-1 interval in Groups I and II did not overlap those in Group IV (Fig. 2). The values of the C-1 interval in Group III lay between those of Groups I and IV.

In a series of 15 patients with mitral stenosis (diameter of the mitral orifice from 2 to 13 mm.), who were examined before valvotomy and two to three weeks later, the C-1 interval shortened from mean 51.3 (40–65) msec. to mean 38.3 (30–60) msec.

The transformation time (Q-1 interval) varied less in the separate groups. The values in the patients with mitral valve disease exceeded the normal ones, but they did not differ essentially with respect to the kind of mitral valve disease. Thus, the Q-1 interval averaged 75.0 msec. in Group I, 73.1 msec. in Group II, 73.7 msec. in Group III, and 71.4 msec. in Group IV. The correlation between the Q-1 interval and the diameter of the mitral orifice was less conspicuous than that between the C-1 interval and the mitral area (Tables I and II). All the individual values of the Q-1 interval in Groups I and II overlapped those in Group IV. Even when the terminal Q-1 variant of Group IV was excluded, only 10.6 per cent of the individual values in Groups I and II did not overlap those in Group IV (Fig. 3). Similar results were obtained by Proctor et al. (1958).

As is known, the duration of the Q-1 interval in mitral stenosis is inversely proportional to the length of the preceding cardiac cycle (Wells, 1957; Steinzeug et al., 1957; Di Perri and Fabrizi, 1958; Tavel, Feigenbaum, and Campbell, 1965b). Because of this, the Q-1 intervals measured were corrected to a standard heart rate of 75/min. (R-R = 800 msec.) using the Ježek (1961) formula. The mean corrected values did not differ essentially from those that were not corrected, but the corrected Q-1 intervals in Groups I and II and in Group IV overlapped each other less (Fig. 4) in comparison with those not corrected.

The statistical study also proved that the diagnostic reliability of the C-1 interval was higher in comparison with that of the Q-1 interval (Table II). It should also be added that the Q-1 interval in the hypertensive patients was considerably longer.

### Table II

<table>
<thead>
<tr>
<th>Groups*</th>
<th>C-1</th>
<th>Q-1</th>
<th>Q-1e</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t</td>
<td>p</td>
<td>t</td>
</tr>
</tbody>
</table>

* For explanation of Groups see text, and footnote to Table I.

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**Fig. 2.** Individual values of the C-1 interval in normal subjects (NS), hypertensive patients (HP), and patients with mitral valve disease. The mean values are shown by a dotted line. See text.
than in the normal series, which is well known (Weissler, Leonard, and Warren, 1958; Dack et al., 1960).

**DISCUSSION**

Since the prolongation of the transformation time (Q-1 interval) in pure or predominant mitral stenosis is due to a longer initial phase of ventricular contraction (C-1 interval), it is logical that this latter interval should be used in the diagnosis of mitral stenosis. The diagnostic significance of the Q-1 interval diminishes because of the electromechanical delay, which does not vary in relation to the kind of the mitral valve disease. Thus, the electromechanical delay may, to a certain extent, straighten out the characteristic differences expected in the duration of transformation time in mitral stenosis and regurgitation. For instance, there are cases of mitral stenosis with very short Q-C interval—10 msec., and very long C-1 interval—60 msec.; the resulting Q-1 interval is at the upper normal limit—70 msec. (Proctor et al., 1958) (Fig. 5A). And, conversely, in some cases of mitral

![Image](http://heart.bmj.com/BrHeartJ:1stpub10.1136/hrt.29.5.778)
regurgitation there may be an unusually long Q–C interval, for instance 70 msec., and a normal C-1 interval—30 msec.; the resulting Q-1 interval is 100 msec., which is characteristic of severe stenosis (Chavez et al., 1955) (Fig. 5B). Warembourg and Ducloux (1963) first reported a longer deformation time of the ventricle (C-1 interval) in cases with mitral stenosis (mean 68.8 msec.). But these authors considered both the C-1 and the Q-1 intervals to be of equal diagnostic significance, and they did not find any correlation between the duration of the C-1 interval and the area of the mitral orifice.

Although the Q-1 interval as a whole is dependent on the heart rate, especially in mitral stenosis, the correction of the C-1 interval, because of its shortness, may be avoided without considerably diminishing its diagnostic value. In cases of atrial fibrillation the error would be minimal if cardiac cycles of approximately 800 msec. duration were used.

SUMMARY

The initial phase of the ventricular contraction (C-1 interval), measured from the onset of the systolic wave in the apex cardiogram to the onset of the main vibrations of the first mitral sound in the phonocardiogram, is a more reliable index in the diagnosis of mitral stenosis than the transformation time (Q-1 interval). The C-1 interval shows the actual delay of the first mitral sound. The diagnostic value of the Q-1 interval is diminished by the electromechanical delay, which may change the results.

REFERENCES


