Observations on the Delayed First Heart Sound in Mitral Stenosis and Hypertension

By Arnold M. Weissler, M.D., James J. Leonard, M.D., and James V. Warren, M.D.

The measurement of the time between the onset of ventricular depolarization and the first heart sound, the Q-1 interval, has been employed in the phonocardiographic evaluation of patients with rheumatic heart disease. Prolongation of this interval has been noted frequently in patients with mitral stenosis and may be used as evidence in weighing the advisability of operation. In the present study, delayed appearance of the first heart sound was often noted in patients with hypertensive vascular disease as well. The mechanism for these changes as well as their significance in the phonocardiographic evaluation of patients with combined valvular disease and hypertension is discussed.

Delayed appearance of the first heart sound was originally described in 1911 by Weiss and Joachim as a phonocardiographic sign of mitral stenosis. With the advent of valvular surgery and the consequent need for more accurate assessment of mitral stenosis, interest in this observation has been renewed. The measurement of the Q-1 interval, that is the interval between the onset of ventricular depolarization and the first heart sound has been accordingly evaluated as an index of the severity of stenosis. Recent observations in this laboratory have suggested that, in addition, the timing of the first heart sound may be altered in patients with hypertensive vascular disease. The present study was undertaken to gain further information regarding this phenomenon.

Method

Simultaneous logarithmic phonocardiograms and electrocardiograms were recorded on a Sanborn Twinbeam Phonocardiograph at a paper speed of 75 cm. per second in 18 normal individuals, 25 patients with hypertensive vascular disease (diastolic pressure greater than 110 mm. Hg), and 10 patients with clinical and catheterization evidence of mitral stenosis. No patients with QRS intervals exceeding .10 second or with evidence of overt congestive heart failure or myocardiitis were included. All patients studied had normal sinus rhythm and all of the hypertensive patients had either electrocardiographic or radiographic evidence of cardiac enlargement. The interval between the Q wave and the first heart sound, hereafter referred to as the Q-1 interval, was measured from the onset of electric systole to the first high-amplitude high-frequency vibrations of the first heart sound (fig. 1). The onset of electric systole was usually determined from the standard limb lead best delineating a Q wave. When a Q wave was not observed, the upstroke of the R wave in lead II was employed. Five sharply inscribed complexes were measured and the average was recorded to the nearest .005 second. All measurements were made from readings taken with the standard 50 mm. bell at the apex. With the paper speeds employed in this study, independent observations of the Q-1 interval agreed within .01 second. The data were analyzed statistically according to the methods outlined by Snedecor."

Results

The results are summarized in table 1 and figure 2. The mean interval in the normal group was found to be .055 second (S. D. ± .01) with a range of .03 to .07 second. Only 1 subject in this group, an apparently healthy young adult, had a Q-1 time of .07 second. A
Q-1 interval of .07 second or greater was considered to represent abnormal delay in the appearance of the first heart sound.

The mean Q-1 interval for the patients with mitral stenosis was .08 second (S. D. ± .01) with a range of .065 to .10 second. The mean Q-1 interval for the hypertensive group was .07 second (S. D. ± .009) with a range of .05 to .085 second, the values falling between the former groups. Of particular significance was the observation that 13 of the 25 hypertensive patients had Q-1 intervals of .07 second or greater.

The mean QRS and R-R intervals for the groups are also shown. The mean QRS interval for the hypertensive group was significantly greater than the normal subjects (p < .01). The length of this interval was poorly correlated with the QRS duration (r = + .07). No significant differences in the R-R intervals were observed.

In 3 of the hypertensive patients the administration of digitalis (Deslanoside, 1.6 mg. intravenously) failed to alter the Q-1 intervals.

**DISCUSSION**

The measurement of the time interval between the onset of ventricular depolarization and the first heart sound has yielded somewhat different results in the hands of various investigators (table 2). Such variability is due in part to differences in the timing of the onset of ventricular depolarization as judged from arbitrarily chosen standard limb leads, and to the variable inclusion of the initial low-frequency vibrations of the first heart sound in the measurement. In an attempt to control these factors better in the present study, the standard limb lead demonstrating the earliest onset of ventricular depolarization rather than any single arbitrarily chosen limb lead was employed for the timing of the Q wave, and the initial high-frequency high-amplitude vibrations of the first heart sound were selected for the timing of the first heart sound. When these criteria were used, the mean Q-1 interval in the normal group was .055 second (S. D. ± .010). Although the mean is slightly higher than that reported by most previous authors, the range of values is similar to those recently reported by Kelly and Craig.

In the study of Braunwald, Fishman, and Cournand on the time relationships of ventricular pressure curves in patients with a normal cardiovascular status, the mean interval from onset of the Q wave to the beginning of left ventricular systole was .052 second (S. D. ± .0067). This time corresponds well with the present data.

In an attempt to quantitate the delayed appearance of the first heart sound in mitral stenosis, it has been suggested that prolongation of the Q-1 interval to .07 second or greater, in the absence of bundle-branch block, be considered as strong evidence in favor of significant mitral stenosis. In view of the present observations, however, we must modify our concept of the specificity of this finding, for it would appear that pathologic processes other than valvular stenosis can delay closure of the atrioventricular valves.

The present data do not elucidate the mechanism for the delayed appearance of the first heart sound in the hypertensive patient.
Prolongation of the Q-1 interval might result from a slowing of either electric or mechanical events between the onset of depolarization and the appearance of the first heart sound. With respect to the former, prolongation of the depolarization process as evidenced by a significantly increased QRS interval was observed in the hypertensive group. However, only a poor correlation existed between the duration of depolarization and the Q-1 interval, indicating that other factors of a mechanical nature must also play a role. The lack of clinical evidence for congestive heart failure in the patients studied as well as the failure of digitalis to alter the Q-1 interval in 3 of the hypertensive subjects would tend to dismiss myocardial failure as an important factor. Another plausible explanation for the delayed first heart sound is a diminution in the rate of pressure rise during early ventricular systole. Although such a mechanism has been observed in patients with myocarditis, there is presently no evidence of similar changes in patients with hypertension or ventricular hypertrophy.

The delayed appearance of the first heart sound in hypertensive patients helps in part to explain the frequent occurrence of an atrial gallop sound in these individuals. Present evidence is consistent with the thesis that the gallop is produced by a separation of the early atrial components from the later major vibrations of the first heart sound. The delayed appearance of the major vibrations of the first sound results in further separation of the 2 components, facilitating their audibility as individual sounds. The atrial gallop phenomenon is frequently present in the absence of a prolonged Q-1 interval, suggesting that the mechanisms for these 2 phenomena are not interdependent.

In patients with rheumatic heart disease, the determination of the Q-1 interval alone or in combination with the measurement of the time between the second heart sound and the opening snap (2-OS time) offers a semi-quantitative means of estimating the degree of mitral valvular stenosis. Abnormal prolongation of the Q-1 interval, a very short 2-OS time, and a formula developed by Wells relating the 2 measurements have been employed in various laboratories as criteria for significant, i.e., operable, mitral stenosis. As a consequence of the present observations and recent studies demonstrating that hypertension can increase the 2-OS time as well, the phonocardiographer is confronted with considerable difficulty in evaluating patients with combined valvular disease and hypertension. It would appear that the presence of hypertension imparts the impression in the Q-1 interval, of a more severe stenosis, while in the 2-OS time, of a lesser degree of valvular stenosis than is actually present. It might be possible that employing the Well’s formula

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**Table 2.—Reported Observations on the Normal Q-1 Interval in Man* **

<table>
<thead>
<tr>
<th></th>
<th>Seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weiss and Joachim</td>
<td>.05 — .07</td>
</tr>
<tr>
<td>Gerhartz</td>
<td>.061</td>
</tr>
<tr>
<td>Bull</td>
<td>.04</td>
</tr>
<tr>
<td>Fahr</td>
<td>.02 — .03</td>
</tr>
<tr>
<td>Lewis</td>
<td>.011 — .039</td>
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<tr>
<td>Battaerd</td>
<td>.02 — .065</td>
</tr>
<tr>
<td>Kanner</td>
<td>.03</td>
</tr>
<tr>
<td>Leblanc</td>
<td>.02 — .04</td>
</tr>
<tr>
<td>Kelly</td>
<td>.04</td>
</tr>
</tbody>
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*Adapted from Orias and Braun-Menéndez.*

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**Fig. 2.** Range of individual Q-1 interval measurements (seconds) in normal subjects and patients with hypertension and mitral stenosis.
for assessment of the degree of stenosis, (Q-1 minus 2-OS), the deviations induced by hypertension would be mathematically cancelled. However, since the mechanism for prolongation of the Q-1 interval and that for increasing the 2-OS time are most probably different, it cannot be assumed that the effects of hypertension would be cancelled from a physiologic standpoint. In view of these observations one must exert considerable caution in the phonocardiographic evaluation of the patient with mitral stenosis and co-existent hypertension.

**SUMMARY**

The Q-1 interval is a measure of the time between onset of ventricular depolarization and the first heart sound. Prolongation of this interval has been noted frequently in patients with mitral stenosis.

Prolongation of the Q-1 interval was a frequent observation in 25 patients with severe hypertensive vascular disease, the range of values for this group falling midway between normal individuals and patients with mitral stenosis. While the mechanism for the delayed appearance of the first heart sound is not wholly elucidated, present evidence favors combined electric and mechanical factors. The importance of these observations in the phonocardiographic evaluation of patients with combined valvular disease and hypertension is stressed.

**SUMMARIO IN INTERLINGUA**

Le intervallo Q-1 representa un mesura del tempore inter le initiation del displorisation ventricular e le prime sono cardiae. Prolongation de iste intervallo ha frequentemente essite notate in patientes con stenosis mitral.

Prolongation del intervallo Q-1 eseva un phenomeno frequente in 25 patientes con sever morbo vascular hypertensive. Le scala del valores in iste gruppo eseva intermediari inter illo in individuos normal e illo in patientes con stenosis mitral. Ben que le mechanismo responsabile pro le retardo del prime sono cardiae non es completamente elucidate, le datos nune disponibile supporta le notion que il se tracta del effetto de un combination de factores electric e mechanic.

Es signalate le importantia de iste observationes pro le evaluation phonocardiographic de patientes con morbo valvular e hypertension in combination.

**REFERENCES**


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_Circulation._ 1958;18:165-168
doi: 10.1161/01.CIR.18.2.165
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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