The Q-II Interval
A Study of the Second Heart Sound in Normal Adults and in Systemic Hypertension

By Pravin M. Shah, M.D., and Sheldon Slodki, M.D.

In the past, phonocardiographic studies of the second heart sound have evaluated the interval between the two components. Data on normal subjects and the effects of various disease states on the splitting of the second sound have been extensively reported.1-6 The interval between the two components can be influenced by a shift of either one or both. Equal movements of both components in the same direction in a disease state may leave the interval entirely unaffected.

The Q-IIa interval (i.e., Q-aortic component of the second sound) includes the distance from Q to onset of left ventricular pressure rise plus the total duration of left ventricular systole (onset of pressure rise to aortic incisura) * (fig. 1). It has been shown that, in the absence of conduction disturbances, the interval Q to onset of left ventricular pressure rise is relatively constant. This suggested a constant relationship between Q-IIa and total left ventricular systole.

As a preliminary to our clinical study, we have plotted the averages of the Q-IIa intervals obtained in 18 normal, anesthetized dogs, against duration of left ventricular systoles, measured in the same cycles (fig. 2). A high degree of correlation is evident (r = 0.98; p < 0.005). These initial findings indicated the usefulness of a study of the Q-IIa and Q-IIp intervals.

While evaluating the possible influence of various cardiac diseases on the timing of aortic or pulmonary valve closure, it was felt imperative to establish rate-corrected, normal standards for Q-IIa and Q-IIp intervals in a large number of normal subjects, thereby providing a reference standard for the second sound in the evaluation of the disease states. The present study has been undertaken to provide these normal standards in the healthy adult subjects. These have been further utilized to assess the second heart sound in advanced systemic hypertension.

Material and Methods

Normal Subjects

One hundred and twelve healthy men were studied. These were selected from the inmates of Cermak Memorial Hospital, House of Correction, City of Chicago, and from the medical students and interns at the Mount Sinai Hospital, Chicago. Only those between the ages of 21 and 50 were included in the present study. The age distribution of the subjects studied was 21 to 30 years, 28 subjects; 31 to 40 years, 61 subjects; and 41 to 50 years, 23 subjects.

Hypertensive Subjects

Fifteen cases of severe systemic hypertension were studied, seven males and eight females. Their ages ranged from 34 to 56: two were below 40, ten between 41 and 50, and three above 50 years of age.

Fourteen patients had a diastolic blood pressure of 120 mm. Hg or greater, the highest being 180 mm. Hg. All of these patients were being followed in the Hypertensive Clinic at Mount Sinai Hospital, Chicago, for over a year, and were classed as grade III (severe essential hypertension) according to the grading system of Duncan et al.7 Only ambulatory patients, who on careful

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* It is assumed here that the aortic component of the second sound coincides with the aortic incisura.
questioning denied any history of exertional intolerance, were included. Left ventricular hypertrophy was present on clinical as well as electrocardiographic, criteria in all the patients. Fourteen of the 15 cases had a QRS interval of 0.1 sec. or less. This study was performed during the pretreatment baseline evaluation while the patients were receiving no antihypertensive and no digitalis preparations. No abnormalities of the serum electrolytes were present. One patient, with a diastolic pressure of 100 mm. Hg and a QRS interval of 0.11 sec., was included in the study because of the presence of paradoxical splitting of the second sound.

Of the 15 cases, only four had a short, mid-systolic, "ejection" type of murmur. This murmur was pronounced (grade III/VI) in one case, while in the remaining three cases it was considered to be an insignificant aortic flow murmur.

Phonocardiograms were simultaneously obtained from two locations on the thorax (mild clavicular line, fifth left interspace; second left interspace, parasternally) with the subjects in the supine position and after sufficient time lapse to attain a steady state. Simultaneous electrocardiogram (lead II) and respiratory tracing were also recorded. The records were made in end-expiratory apnea, end-inspiratory apnea with normal respiration, and in end-expiratory apnea after mild exercise; the latter consisted of raising both legs against gravity about 10 to 15 times and was sufficient to produce a mean decrease of the R-R interval of 112.6 msec. In addition, recordings of carotid pulse and phonocardiograms from four locations on the precordium (second right interspace, second left interspace, fourth left interspace, and apex) were obtained in all subjects.

A Sanborn four-channel photographic recorder was used; dynamic microphones were placed at the above locations and were held in place by rubber straps. The recordings were made on a bromide paper at the speed of 100 mm./sec. at a nominal frequency of 200 cycles/sec. with a high-pass filter. This permitted accurate measurements up to ±3 msec. and allowed proper delineation of each component of the second sound.

The following measurements were made: R-R interval, Q-II\(_A\) (onset of QRS to the aortic component of the second sound), and Q-II\(_P\) (onset of QRS to the pulmonary component). The first three or four end-expiratory cycles were measured

Figure 1

Relationships of Q-II\(_A\) and Q-II\(_P\) intervals to ventricular systole.

Figure 2

Linear correlation between Q-II\(_A\) interval and the duration of left ventricular systole simultaneously measured in 18 dog experiments (r = 0.98; p < 0.0005).
and averaged both at rest and after exercise. In records with normal respirations, cycles at the peaks of expiration and of inspiration were respectively measured.

Statistical analysis was made on the data obtained. Least-squares regression technics were used to examine the relation of the Q-IIₐ and Q-IIₚ intervals to age, systolic pressure, and the R-R interval. Results

Normal Subjects

Aortic Component

A linear dependence of the end-expiratory Q-IIₐ interval on the square root of the R-R interval was demonstrated by statistical analysis. The correlation coefficient between the two is 0.811 (p < 0.0001). The results on 112 subjects are presented in figure 3, which shows the least-squares regression line and the 95-per cent confidence limits for individual prediction. The predicted Q-IIₐ for a given R-R interval can be expressed as:

\[
Q-\text{II}_a = 61.6 + 10.24 \sqrt{RR} \quad (1)
\]

The variance can be expressed as:

\[
\text{Variance } Q-\text{II}_a = 710.50 - 29.0270 \sqrt{RR} + 0.497498 \times RR \quad (2)
\]

The average value for one standard deviation is about 17.5. A wide range of R-R intervals (from 500 to 1,300 msec.) was considered, and this value did not show any marked variation. Therefore, a reasonable approximation to the 95-per cent confidence limits for individual estimate is the predicted value (equation 1) ±35 msec.

After exercise, the mean R-R interval was decreased by 112.6 msec. Statistical analysis showed, however, that the linear relationship of the Q-IIₐ on the \(\sqrt{RR}\) was not altered.

No significant effect of age on Q-IIₐ was noted within the range studied (21 to 50 years). Analysis of the effect of the blood pressure level in this group (systolic pressures between 105 and 140 mm. Hg) demonstrated no significant effect on the Q-IIₐ interval.

Pulmonic Component

A linear dependence of the end-expiratory Q-IIₚ on the square root of R-R interval was also noted. The correlation coefficient is 0.734 (p < 0.0001). A small, but statistically significant linear dependence on age was also demonstrated and can be expressed as:
Q-II$_p$ = 98.9 + 10.631 $\sqrt{RR} - 0.860$  

It can be readily seen that, for each additional year of age, the Q-II$_p$ interval would be expected to decrease by 0.860 msec., thus resulting in a reduction of about 25.8 msec. in 30 years. However, it is important to recognize that this dependency is based on data between the ages of 21 and 50. Extrapolation in either direction is not justified.

Since the dependence on RR, as well as on age, has been noted, the calculation of the variance of predicted values becomes somewhat complicated. However, since the contribution of age is small, a reasonable approximation to the 95-per cent confidence limits of individual prediction can be arrived at by ignoring the variance of the age effect. When this is done, it can be shown that the approximate 95-per cent confidence limits of the predicted value (equation 3) is ±50 msec.

The linear dependence of Q-II$_p$ on RR was maintained in the same group of subjects following exercise.

Respiratory Variations in Splitting of the Second Sound

Technically satisfactory tracings during quiet spontaneous breathing were available in 86 normal subjects. The cycles having least splitting during expiration and those having greatest splitting during inspiration were selected for measurements. The respiratory variations in the splitting of the second sound are summarized in table 1.

It can be readily observed that the interval between the two components is smaller with advancing age during both phases of respiration. Thus, during expiration, an interval of 0.021 sec. or more (equivalent to a clinically audible splitting) was present in 15 subjects (53.5 per cent) between 21 and 30 years; in 25 subjects (52.1 per cent) between 31 and 40 years; and in only two subjects (20 per cent) between 41 and 50 years. During inspiration, a splitting of 0.021 sec. or more was noted in 23 subjects (82.1 per cent) between 21 and 30 years; in 31 subjects (64.5 per cent) between 31 and 40 years; and only in three subjects (30 per cent) between 41 and 50 years.

Of the 60 subjects in whom inspiration resulted in a wider splitting, this increase was less than 0.010 sec. in 18; between 0.011 and 0.020 sec. in 18; between 0.021 and 0.030 sec. in 19; and greater than 0.031 sec. in five. The maximum increase noted was 0.048 sec. Inspiratory delay in the pulmonic component was seen in all of these subjects. An earlier occurrence of the aortic component contributed less, but significantly, to the splitting in 36 subjects.

On analyzing the individual movements of the aortic and pulmonary components of the second sound during inspiration, in all of the 86 subjects, the aortic component was seen to vary from −15 to +10 msec. (average −1.6 msec.), and the pulmonary component from 0 to +48 msec. (average +9.85 msec.). The average shift of each component was deter-

Table 1

<table>
<thead>
<tr>
<th>Interval between two components</th>
<th>0 (indistinct)</th>
<th>less than 0.02 sec.</th>
<th>0.021 to 0.03 sec.</th>
<th>0.031 to 0.04 sec.</th>
<th>greater than 0.04 sec.</th>
<th>maximum sec.</th>
<th>mean interval msec.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no.</td>
<td>28</td>
<td>4</td>
<td>9</td>
<td>4</td>
<td>2</td>
<td>0.04</td>
<td>18.1</td>
</tr>
<tr>
<td>21-30 yr.</td>
<td>9</td>
<td>3</td>
<td>20</td>
<td>17</td>
<td>6</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>31-40 yr.</td>
<td>(32.1%) (10.7%)</td>
<td>(41.7%) (35.4%)</td>
<td>(60%) (60%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>41-50 yr.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
THE Q-II INTERVAL

Table 2

Effect of Inspiration on the Splitting of the Second Sound and on Each Component in Various Age Groups

<table>
<thead>
<tr>
<th>Age group</th>
<th>21-30 yr.</th>
<th>31-40 yr.</th>
<th>41-50 yr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no.</td>
<td>28</td>
<td>48</td>
<td>10</td>
</tr>
<tr>
<td>Avg. insp. shift in II_A (msec.)</td>
<td>-3.0</td>
<td>-1.8</td>
<td>+0.1</td>
</tr>
<tr>
<td>Range</td>
<td>-15.0 + 6.0</td>
<td>-12.0 + 10.0</td>
<td>-6.0 + 6.0</td>
</tr>
<tr>
<td>Avg. insp. shift in II_P (msec.)</td>
<td>+15.0</td>
<td>+9.0</td>
<td>+4.5</td>
</tr>
<tr>
<td>Range</td>
<td>0.0 + 40.0</td>
<td>0.0 + 48.0</td>
<td>0.0 + 14.0</td>
</tr>
<tr>
<td>Avg. insp. increase of II_A-II_P interval (splitting) (msec.)</td>
<td>+18.0</td>
<td>+10.8</td>
<td>+4.4</td>
</tr>
<tr>
<td>Range</td>
<td>0.0 - 45.0</td>
<td>0.0 - 40.0</td>
<td>0.0 - 20.0</td>
</tr>
</tbody>
</table>

mined in different age groups and has been summarized in Table 2.

**Hypertensive Subjects**

**Aortic Component**

A linear dependence of the end-expiratory Q-II_A interval on the square root of the R-R interval was demonstrated by statistical analysis. The correlation coefficient between the two is 0.823 (p < 0.0005). The results of the 15 hypertensive subjects are presented in Figure 4 along with the least-squares regression line for the normal subjects. It is clear, both visually and by formal analysis, that there is no basis for assuming that the slopes of the two lines are different; however, the intercepts

![Figure 4](http://circ.ahajournals.org/)

The end-expiratory Q-II_A intervals in 15 cases with advanced hypertension are plotted against √RR interval. The dotted line is the least-squares regression line for normal subjects and the continuous line is the least-squares regression line for the hypertensive subjects.
for the two lines differ significantly \( (p < 0.0005) \). The predicted \( Q-II_A \) for a given R-R interval can be expressed as

\[
Q-II_A = 99.8 + 10.065 \sqrt{RR}
\]

A common slope for the hypertensive subjects and the normal subjects allows a direct comparison of the intercepts of the two lines. The revised equations with a common slope are

\[
Q-II_A = 95.9 + 10.21 \sqrt{RR} \quad \text{(hypertensive)}
\]

\[
Q-II_A = 62.5 + 10.21 \sqrt{RR} \quad \text{(normal)}
\]

The average value for one standard deviation is 25 msec. and this does not show marked variation \( (\text{range 23 to 28 msec.}) \) in the wide range of the R-R intervals considered \( (500 \text{ to } 1,300 \text{ msec.}) \). Therefore, a reasonable approximation to the 95-per cent confidence limits for individual estimate is the predicted value \( (\text{equation 1}) \pm 50 \text{ msec.} \)

No significant effect of age on the \( Q-II_A \) interval was noted within the group studied \( (34 \text{ to } 56 \text{ years}) \) \( (\text{fig. 4}) \).

**Pulmonary Component**

A linear dependence of the end-expiratory \( Q-II_P \) on the square root of the R-R interval was also noted. The correlation coefficient is 0.785 \( (p < 0.0005) \). No statistically significant dependence on age was demonstrated but, as the age range is narrow and the number of patients is small, such dependence might be present. The predicted \( Q-II_P \) interval for a given R-R interval can be expressed as:

\[
Q-II_P = 100.6 + 10.459 \sqrt{RR}
\]

A common slope has been calculated for the hypertensive and the normal subjects which now allows a direct comparison of the intercepts of the two lines. The revised equations with a common slope are

\[
Q-II_P = 89.11 + 10.59 \sqrt{RR} \quad \text{(hypertensive)}
\]

\[
Q-II_P = 61.45 + 10.59 \sqrt{RR} \quad \text{(normal)}
\]

The difference between the intercepts is statistically significant \( (p < 0.0005) \).

**Respiratory Variations in the Splitting of the Second Sound**

Table 3 summarizes the effects of respiration on the hypertensive subjects.

The splitting of the second sound during expiration was less than 0.03 sec. in all 15 subjects, and was so during inspiration in 11 subjects. Only one subject had a splitting greater than 0.04 sec. during inspiration. Average inspiratory increase of splitting was 7.2 msec. with a shift of the aortic component by \(-1.2 \text{ msec.} \) and of the pulmonary component by \(+6.0 \text{ msec.} \).

One patient, who had a diastolic pressure of 100 mm. Hg and a grade-III systolic murmur, demonstrated paradoxical splitting, the expiratory splitting being 0.03 sec. This patient had a QRS interval of 0.11 sec. resulting from an incomplete left bundle-branch block.

**Discussion**

Graphic studies of the second sound in normal adults are relatively few. Leatham and Towers \( (1951) \), in their study, report on 20 healthy subjects including 10 children and 10

| Table 3 |

*Splitting of the Second Heart Sound in Hypertensive Patients during Various Phases of Respiration*

<table>
<thead>
<tr>
<th>Phase of respiration</th>
<th>Expiration</th>
<th>Inspiration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interval between two components</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 (indistinct)</td>
<td>7 cases</td>
<td>4 cases</td>
</tr>
<tr>
<td>less than 0.02 sec.</td>
<td>5 cases</td>
<td>4 cases</td>
</tr>
<tr>
<td>0.021 to 0.03 sec.</td>
<td>3 cases</td>
<td>3 cases</td>
</tr>
<tr>
<td>0.031 to 0.04 sec.</td>
<td>0 cases</td>
<td>0 cases</td>
</tr>
<tr>
<td>greater than 0.04 sec.</td>
<td>0 cases</td>
<td>1 case</td>
</tr>
<tr>
<td>Maximum splitting (sec.)</td>
<td>0.03</td>
<td>0.042</td>
</tr>
<tr>
<td>Mean splitting (msec.)</td>
<td>11.4</td>
<td>18.6</td>
</tr>
<tr>
<td>Average increase of splitting</td>
<td>7.2 msec.</td>
<td></td>
</tr>
<tr>
<td>Average shift of ( II_A )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average shift of ( II_P )</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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young adults. They described splitting of the second sound in all; however, in some this occurred only on inspiration. The average inspiratory splitting was 0.05 sec. (0.02 to 0.08 sec.). Boyer and Chisholm \(^8\) initiated studies on the individual components of the second sound using the first sound as the reference point, and described a dual mechanism for the inspiratory increase of splitting. Shafter \(^9\) (1960) repeated similar studies in 15 normal subjects (six children, eight young adults) and remarked that, in children, the aortic component moved little with respiration as compared to the pulmonary. On the other hand, Castle and Jones \(^10\) (1961) studied the mechanism of respiratory variations in the splitting of the second sound in 68 normal children, and described that the aortic component moved earlier on inspiration by an average of 7 msec. while the pulmonary component was delayed by an average of 13 msec. Aygen and Braunwald \(^11\) studied 51 normal subjects between the ages of 4 and 49 (average 14.2) using Q-II intervals. They made no comment on the effect of age on the degree of splitting.

The results of the present study confirm a linear dependence of the Q-II\(_A\) and Q-II\(_P\) intervals on the square root of R-R interval, and the correlation was seen to persist after the heart rate was altered by exercise. Equations for prediction of the end-expiratory Q-II\(_A\) and Q-II\(_P\) intervals from a given R-R interval have been described. The standard deviation of the predicted Q-II\(_A\) is 17.5, and of the predicted Q-II\(_P\) is 25. These somewhat large standard deviations may partly be due to an inability to determine the exact onset of the QRS complex. This may well be obviated in the future by means of two simultaneous electrocardiographic leads or a vectorcardiogram. This degree of variation does not permit one to predict small deviations in the Q-II intervals in individual subjects; however, the present method provides a useful reference point for study of a group of individuals with similar hemodynamic abnormalities. Consistent and statistically significant deviations from the normal group would permit a more accurate analysis of the influence of a disease state, or of known hemodynamic abnormalities, upon individual components of the second heart sound.

Furthermore, since the Q-II\(_A\) and Q-II\(_P\) intervals in the absence of intraventricular conduction disturbances bear a significant correlation to the duration of left and right ventricular systoles, respectively, these intervals can be used for their study, especially since the influence of various diseases on the duration of ventricular systole has not been widely studied. It is not often practicable to undertake hemodynamic studies (such as cardiac catheterization) in patients with coronary heart disease, heart failure, or with other contraindications. The Q-II intervals can be used as simple determinable indices of the duration of systole for each ventricle.

**Effect of Age**

In end-expiratory apnea, no significant effect of age could be demonstrated on the aortic component; however, the Q-II\(_P\) interval was shown to be statistically related to age. Within the age groups examined (21 to 50 years), the pulmonary component occurred earlier for every additional year of age by a mean of 0.860 msec. The influence of age was confirmed in the studies of 86 subjects during normal breathing, since splitting of the second sound greater than 0.02 was significantly rare in both phases of respiration in the age group 41 to 50 years as compared to younger subjects. This further emphasizes the need to consider the factor of age in the evaluation of the second sound in disease states.

**Effect of Respiration**

A significant increase of the splitting during inspiration was noted in 60 subjects (70 per cent). This was contributed to by an earlier occurrence of the aortic component in 36 subjects. A delay in the pulmonic component was demonstrable in all 60 cases. The over-all contribution by a delay of the pulmonary component to the increase in splitting was 85.6 per cent. The earlier occurrence of the aortic component accounted for 14.3 per cent of the increased interval. The mechanism of these
two phenomena is thought to result from unbalanced filling and altered output of the two ventricles during inspiration related to increased right atrial filling and decreased pulmonary venous return. In Aygen and Braunwald’s series,11 86 per cent of the increased splitting was accounted for by prolongation of right ventricular systole and 14 per cent by abbreviation of left ventricular systole. In 19 cases of the present series, however, some delay in the aortic component was noted during inspiration. This uncommon phenomenon was also noted, but not explained, by Aygen and Braunwald11 in some of their cases. The average delay in our 19 cases was 5.0 msec. and was not related to age.

A further analysis of the influence of age on the degree of respiratory increases in splitting and on the degree of shift of the individual components demonstrated some intriguing relationships. Inspiratory increase in the splitting of the second sound was significantly less in subsequent groups of increased age. This resulted both from a decrease in mobility of the aortic component and a lesser delay of the pulmonary component. These differences with age become more obvious by comparing the results of others in younger subjects with the present material in adults. Thus, Shafter9 in his group of normal children and young adults reported an earlier occurrence of the aortic component as averaging 12.6 msec. and a delayed pulmonary component of 16.3 msec. Aygen and Braunwald,11 in patients with a mean age of 14 years, reported that the aortic component moved 5.2 msec., and the pulmonary, 32.6 msec. The present material represents data in adults: age groups 1 (21 to 30), 2 (31 to 40), and 3 (41 to 50), show a shift in the aortic component of -3.0 (group 1), -1.8 (group 2), and +0.1 msec. (group 3), respectively; the shift of the pulmonic component was +15 (group 1), +9.0 (group 2), and +4.5 msec. (group 3), respectively. These data disclose a distinct trend toward a diminished hemodynamic responsiveness of either ventricle to the influence of respiration with advancing age. The explanation may well lie in a smaller increase of venous return and less pooling in the lungs, as a result of normal inspiration, in older age groups.

This influence of advancing age on the end-expiratory position of the pulmonary component of the second sound and on the respiratory variations of the splitting is apparently a hitherto unappreciated phenomenon.

With advancing age, the left ventricle gains increasing dominance over the right as revealed electrocardiographically by a progressive leftward shift of the axis and by a diminution of the waves due to right ventricular depolarization. This suggests that, with increasing age, the left ventricular mass acquires greater influence on total contractile dynamics in comparison to the right. The role of the interventricular septum dominated by the left ventricle in the dynamics of right ventricular systole may become more prominent. Such a phenomenon could explain an apparent progressive loss of right ventricular autonomy with age. The two ventricles would then tend to become more synchronous with age, and the duration of their systole would probably be dictated by the left ventricle, in normal circumstances. Such a postulate can explain the observed progressive decrease in the splitting of the second sound with age chiefly caused by the shift of the pulmonary component.

**Effect of Advanced Hypertension**

The influence of arterial hypertension on the corresponding semilunar valve closure is not established. There is some evidence that pulmonary hypertension, at least secondary to shunts, results in a narrower splitting of the second sound.3, 4, 6, 12 This presumably results from an earlier closure of the pulmonary valve and an earlier pulmonary component. Some observers believe this to be true for pulmonary hypertension from any cause.6, 12 On the basis of these observations one would expect an earlier closure of the aortic valve in systemic hypertension resulting in a widely split second sound. On the other hand, Gray has reported one instance of paradoxical splitting with hypertension.18 Since this has not been more
frequently reported, it does not appear to be a common phenomenon.

The influence of systemic hypertension on the duration of ventricular systole in man is not precisely known. Earlier acute experiments (Wiggers) with aortic occlusion suggested that the duration of left ventricular systole was slightly shortened. Recent experimental studies by Wallace et al. on the effect of increased aortic resistance without change in heart rate or stroke output showed that the duration of total left ventricular systole was essentially unaffected. The results of these studies cannot, however, be applied without reservation to a chronic situation in patients with considerable left ventricular hypertrophy.

The present group of advanced hypertensive subjects with left ventricular hypertrophy in the absence of heart failure demonstrated a statistically significant delay in the aortic as well as the pulmonary component of the second heart sound.

It can be seen that the predicted Q-IIₐ interval in hypertensive subjects is greater than in the normal subjects by 33.4 msec.

Similarly, the predicted Q-IIₚ in hypertensive subjects is greater by 27.66 msec. as compared to the normal subjects.

Since the delays in the aortic and the pulmonary components are similar, the interval between them would not be expected to differ substantially from the normal. The results reported in table 3 show that, for the group as a whole, the splitting of the second sound during both phases of respiration was similar to that of normal subjects of comparable ages (above 34 years). As the degree of splitting is unaffected in most of the hypertensive subjects, a study of the interval between the two components would not have disclosed in itself the delay of either component.

Prolongation of Q-IIₐ and Q-IIₚ intervals would seem to indicate a prolongation of both left and right ventricular systoles in advanced hypertension.

The mechanism of similar prolongation of systole in the two ventricles is not clear. In the absence of drug therapy at the time of study, the possible influence of autonomic ganglion-blocking agents, reserpine, or digitalis can be excluded. Prolongation of left ventricular systole is probably related to a hypertrophied myocardium forcing blood against an increased resistance. Wiggers reported a prolongation of the duration of ejection in mechanically induced aortic obstruction. Braunwald et al. in well-controlled experiments, observed no change in the duration of ejection with increasing mean aortic pressure. However, with a marked elevation of pressure, a lengthening of the duration of ejection did occur. More recent experiments (Wallace et al.) indicated that elevating mean aortic pressure shortened ejection time, prolonged the isovolumetric contraction phase, and either had no effect or decreased slightly the duration of total systole. Weissler et al. studied left ventricular ejection time from carotid tracings in 11 hypertensive patients. They observed that ejection time as correlated to stroke volume tended to be shorter in uncomplicated hypertension. As the heart rates and cardiac indices of the various patients were similar to those of the normal group, these findings would indicate that severe hypertension in man shortens ejection time. On the basis of these observations it would appear that the observed prolongation of total systole probably results from lengthening of the isovolumic contraction period. A longer isovolumic contraction period could be attributed either to a slower rate of pressure rise (dp/dt) or to the higher diastolic pressure in the aorta (which has to be attained before ventricular ejection begins), or both. Thirteen of the hypertensive subjects were above 40 years of age. On the basis of the effects of advancing age on ventricular dynamics in normal adults, they would be expected to show synchronous ventricular dynamics and lack of independent variability of right ventricular response. Thus, probably the grossly hypertrophied interventricular septum is responsible for prolongation of right ventricular systole. A further study in hypertensive subjects without left ventricular hypertrophy.
seems indicated in order to substantiate this role of myocardial hypertrophy.

Respiratory variations in the splitting of the second sound were seen in 11 subjects consisting of some delay of the pulmonary component with or without earlier occurrence of the aortic component. One patient showed paradoxical splitting with an interval between the two components of 0.03 sec. during expiration, which became smaller during inspiration. This patient had a diastolic pressure of 100 mm Hg, which is the lowest in the present series. The patient, however, had the most prominent systolic “ejection” murmur (grade III/VI). Subvalvular muscular obstruction resulting from the development of a hypertrophic muscular ridge, as well as incomplete left bundle-branch block, could have contributed to the paradoxical splitting. Hemodynamic studies were, however, not performed in this case. Considering the rarity of paradoxical splitting in hypertension, as judged by the paucity of reported cases, it is likely that only complicated cases tend to exhibit this phenomenon.

Summary

A study of the second heart sound in 112 normal adults ranging in age from 21 to 50 years is presented, along with results, in 15 patients with advanced systemic hypertension.

A detailed analysis of the Q-II\textsubscript{A} and Q-II\textsubscript{P} intervals, as related to heart rate, respiration, age, and blood pressure had been performed. A linear dependence on the \( V \) RR interval was noted in both groups.

With advancing age, a statistically significant decrease in the Q-II\textsubscript{P} was noted in the normal subjects.

A decreased mobility of the Q-II\textsubscript{A} and Q-II\textsubscript{P} intervals with respiration was described.

In hypertensive subjects, a significant prolongation of both the Q-II\textsubscript{A} and Q-II\textsubscript{P} intervals without variation in the II\textsubscript{A,P} interval (splitting) as compared to the normal subjects was found. The effect of respiration on the splitting of the second sound was also found to be comparable to the normal for the same age group.

The relationship of changes in ventricular dynamics to systemic hypertension are discussed on the basis of these findings.

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Invention of the Stethoscope

The story of the invention of the stethoscope by René Théophile Hyacinthe Laennec (1781-1826) has often been told: in 1816 he noticed boys in a Court of the Louvre at play with the ear applied to long pieces of wood listening to the transmitted sound of a pin scratch at the opposite end. He immediately put this hint into practice, as he says, by applying a rolled up quire of paper to the chest of a stout girl with symptoms of heart disease, and was electrified by finding that the heart sounds were more audible than to the direct ear. He keenly practised this method at the Necker Hospital, where, however, Granville, as an onlooker on 16 September 1816, states that the original birth of mediate auscultation actually occurred on the chest of a male patient. Laennec then devised a wooden cylinder 1½ inches in diameter, a foot long, and perforated longitudinally by a bore three lines wide; this he regarded as too simple to require a name other than "the cylinder" or "bâton"; but eventually, as somewhat barbarous names, such as sonometra, pectorilogue, thoraciscope, cornet de papier, and cornet médical appeared on the horizon he suggested, if it must have a name, stethoscope. He gave an account of his new method before the Académie des Sciences on 28 February 1818, and in May of the same year lectured before the Medical Faculty of Paris. With his knowledge of morbid anatomy he correlated the local lesions with the corresponding physical signs, like his teacher Corvisart, thus advancing the anatomico-clinical method and the special pathology of the organs, in the extraordinary short time of three years, so that his classical work *Auscultation médiate* appeared about 15 August 1819. While correcting the proofs he had been busily making stethoscopes so that every buyer of his book might be properly equipped; in fact it is probable that at the time of his death every existing stethoscope was the work of his hand (Thayer).—Sir Humphry Davy Rolleston. *The Harveian Oration*. Great Britain, Cambridge University Press, 1928, p. 75.
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