EFFECT OF HYPERTENSION ON THE P WAVE OF THE ELECTROCARDIOGRAM

BY

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The P wave of the electrocardiogram has been investigated in great detail in normal subjects (Shipley and Hallaran, 1936; Graybiel et al., 1944; Stewart and Manning, 1944; Thomas and Dejong, 1954) and in various types of cardiac disease (Wood and Selzer, 1939; Thomas and Dejong, 1954; Martins de Oliveira and Zimmerman, 1959). There have been few studies of the P wave in hypertension, however, and because of this the present work was undertaken.

SUBJECTS AND METHODS

The record cards of the Cardiac Department were examined until 25 patients with hypertension had been found for each of the following ranges of diastolic blood pressures, 100–109, 110–119, 120–129, and over 130 mm. Hg. In addition, 25 subjects with a normal pressure, 10 patients with malignant hypertension, and 15 with hypertensive heart failure were studied. Patients with coincidental congenital, rheumatic, or ischemic heart disease were excluded. The cardiograms of these 150 patients were then examined. A preliminary survey showed that the P wave tended to be broadest and tallest in lead II and therefore this lead was selected for detailed study. The voltage, duration, time between onset and peak (peak delay), and P–R interval were measured. The shape of the P wave in standard lead II and chest leads V4–6 was noted and placed into one of three categories, peaked, rounded, or notched.

All the cardiograms were taken on a Cambridge Electrite machine with a standardization of 1 mV per cm. and a paper speed of 25 mm. per second.

RESULTS

The P wave measurements are shown in the scattergram (Fig. 1). Certain points of interest emerge and these are shown in Table I.

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Duration of P (sec.)</th>
<th>Voltage of P (mV)</th>
<th>Mean peak delay (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>&gt;0·01(%)</td>
<td>&lt;0·08(%)</td>
</tr>
<tr>
<td>Normal pressure</td>
<td>25</td>
<td>0·08</td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>Diastolic pressure</td>
<td>50</td>
<td>0·10</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>&lt;120 mm. Hg</td>
<td></td>
<td>0·10</td>
<td>28</td>
<td>6</td>
</tr>
<tr>
<td>Diastolic pressure</td>
<td>50</td>
<td>0·10</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>&gt;120 mm. Hg</td>
<td>10</td>
<td>0·10</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Malignant hypertension</td>
<td>10</td>
<td>0·10</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Hypertensive failure</td>
<td>15</td>
<td>0·10</td>
<td>20</td>
<td>0</td>
</tr>
</tbody>
</table>

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The scattergram and Table I show that although the mean values for voltage, duration, and peak delay of the P wave in hypertension only slightly exceed those for normal subjects, the distribution of the individual values is altered so that more tend to lie in the upper ranges of normal. There appeared to be no relation between the length of the P–R interval and the height of the diastolic blood pressure. The mean P–R interval for the whole series was 0·16 sec.

Shape of the P Wave. The only significant finding was an increased incidence of notched P
waves in the left ventricular chest leads when the diastolic pressure exceeded 120 mm., and when malignant hypertension and hypertensive heart failure were present (Table II). In the last two groups the incidence of notching was also increased in lead II.

The type of notching seen in hypertensive patients was similar to that seen in mitral stenosis and was due to the dominance of the left atrial component (Fig. 2).

**Fig. 2.—Cardiogram showing the wide bifid P waves commonly seen in hypertension (lead II).**

**Factors Affecting Incidence of Notched P Waves.**

1. **Heart size.** Of the 50 patients with diastolic pressures above 120 mm., 35 had been examined radiologically: 24 had a cardiothoracic ratio exceeding 0.5, and 50 per cent of these had notched P waves in lead II compared with 22 per cent of those with normal ratios. However, in the præcordial leads the incidence of notched P waves was greater in the group with smaller hearts (50% as compared to 30%).

2. **Duration of hypertension.** No correlation was found between the incidence of notched P waves and the duration of hypertension.

3. **T wave inversion in V6.** When the blood pressure is raised an inverted T wave in V6 associated with a deep S wave in V2 and a tall R wave in V5 is accepted as an indication of left ventricular hypertrophy (Sokolow and Lyon, 1949). When this cardiographic sign was present the incidence of notched P waves in lead II was 40 per cent compared to 20 per cent when the T wave was upright. The incidence of notched P waves in the left ventricular chest leads, however, was not increased when the T wave in lead V6 was inverted.

4. **Effect of treatment.** The cardiograms of 25 patients with an initial diastolic pressure above 120 mm. were examined before and after treatment which varied in duration from six months to three years. No significant change was noted in the shape, duration, or height of the P wave as the result of treatment.

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**TABLE II**

**INCIDENCE OF NOTCHED P WAVES IN VARIOUS GRADES OF HYPERTENSION**

<table>
<thead>
<tr>
<th>Group</th>
<th>Percentage incidence of notched P waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lead II</td>
</tr>
<tr>
<td>Normal pressure</td>
<td>28</td>
</tr>
<tr>
<td>Diastolic pressure &lt; 120 mm. Hg</td>
<td>22</td>
</tr>
<tr>
<td>Diastolic pressure &gt; 120 mm. Hg</td>
<td>30</td>
</tr>
<tr>
<td>Malignant hypertension</td>
<td>80</td>
</tr>
<tr>
<td>Hypertensive failure</td>
<td>47</td>
</tr>
</tbody>
</table>
TABLE III
INCIDENCE OF NOTCHED P WAVES AND OF P WAVE DURATIONS EXCEEDING 0·10 SEC. IN HEALTHY ADULTS

<table>
<thead>
<tr>
<th>Authors</th>
<th>No. of cases</th>
<th>P wave duration &gt;0·10 sec. (%)</th>
<th>Percentage incidence of notching in lead II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Graybiel et al. (1944)</td>
<td>1000</td>
<td>4·4</td>
<td>28</td>
</tr>
<tr>
<td>Stewart and Manning (1944)</td>
<td>500</td>
<td>7·4</td>
<td>5</td>
</tr>
<tr>
<td>Shipley and Hallaran (1936)</td>
<td>200</td>
<td>8</td>
<td>30</td>
</tr>
<tr>
<td>Thomas and Dejong (1954)</td>
<td>100</td>
<td>0</td>
<td>15</td>
</tr>
</tbody>
</table>

DISCUSSION

The present study indicates that there are no special features of the P wave that will indicate the presence of hypertensive heart disease in any individual patient. When hypertensive patients are considered as a group, however, it becomes apparent that the duration of their P waves tends to lie nearer to the upper limit of normal than that of normal subjects. Thus the mean P wave duration of the 125 hypertensive patients studied was 0·10 sec. (normal 0·086 sec., Graybiel et al., 1944; 0·09 sec., Stewart and Manning, 1944). In 20·8 per cent the duration exceeded 0·10 sec., compared to 4–8 per cent of normals (Table III).

This difference is highly significant statistically (p < 0·001). Moreover, in only 4·8 per cent was the P wave duration less than 0·08 sec., compared to 12·3 per cent in normals (Graybiel et al., 1944). This difference is also significant (p < 0·01). Similar results were obtained by Wood and Selzer (1939) but Thomas and Dejong (1954) could find no P waves greater than 0·10 sec. in duration in 49 hypertensive patients not in cardiac failure.

Wood and Selzer found that 64 per cent of their 14 patients with hypertensive failure had P waves exceeding 0·01 sec. duration and they noted that these widened P waves were usually bifid and of low voltage (0·1 mV). In the present series the findings were rather different and only 20 per cent of the P waves were of longer duration than 0·10 sec., and these had normal voltage. In lead II, 47 per cent of the P waves were notched while in the left ventricular chest leads the corresponding figure was 64 per cent. The numbers of patients in Wood and Selzer's series and in the present study were small, however, and the differences between the two are not statistically significant. In malignant hypertension the percentage of abnormally wide P waves (40%) was greater than in any other group in the series.

When the diastolic pressure exceeded 120 mm. Hg, dominance of the left atrial component of the P wave occurred with increased frequency and resulted in a notched wave very similar to that occurring in association with mitral stenosis. These notched P waves occurred particularly frequently in the left ventricular chest leads and were present in 50 per cent of patients with diastolic pressures above 120 mm. Hg. The incidence of notched P waves was particularly high in malignant hypertension and they were seen in lead II in 80 per cent of the patients and in the left ventricular chest leads in 60 per cent.

Even when notching of the P wave was not seen, the summit of the P wave was frequently delayed. When the diastolic pressure was below 120 mm. the summit of the P wave occurred less than 0·04 sec. after the commencement of the P wave in 44 per cent of the records examined but when the diastolic pressure exceeded 120 mm. the corresponding figure was only 2 per cent. This delay in reaching the summit of the P wave can be explained on the basis of left atrial hypertrophy.

No correlation could be found between the incidence of notched or widened P waves and the duration of hypertension. Moreover, treatment of the hypertension for periods up to three years had no effect on the shape or duration of the P wave.

Martins de Oliveira and Zimmerman (1959) in a study of the P wave in various conditions considered that among the indications of overloading of the left atrium an increase in the width of the P wave and the appearance of notching were important signs. Sodi-Pallares and Calder...
(1956) also accept this view and consider that a change in the morphology of P is often the earliest electrocardiographic sign of essential hypertension. The findings of the present study support this statement and show that notched P waves wider than 0.1 sec. are of common occurrence in hypertension. They have been observed in patients who are symptom free and without radiological or other cardiographic change. They probably indicate left atrial hypertrophy and it would be of interest to correlate the width and shape of the P wave with the left ventricular end-diastolic pressure.

**SUMMARY**

The P wave of the electrocardiogram has been studied in 125 patients with diastolic blood pressures exceeding 100 mm. Hg. Of these patients 100 had benign hypertension, 10 malignant hypertension, and 15 hypertensive heart failure.

When compared with normal controls the hypertensive patients showed a statistically significant increased incidence of P waves of longer duration than 0.10 sec.

An increased incidence of notched P waves was found in the left ventricular chest leads in patients with diastolic pressures above 120 mm. Hg and in patients with malignant hypertension and hypertensive heart failure. In the last two conditions there was also an increased incidence of notched P waves in standard lead II. The type of notching resembled that seen in mitral stenosis.

The summit of the P wave tended to occur later in hypertensive patients than in normal subjects particularly when the diastolic pressure exceeded 120 mm. Hg.

No correlation was found between the width and shape of the P wave and the duration of hypertension. Hypotensive therapy for up to three years caused no alteration in those P waves that were originally widened or notched.

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**REFERENCES**


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