ELECTROCARDIOGRAPHIC DIAGNOSIS OF LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSION

BY

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It is known that the electrocardiogram may remain normal in the presence of even a moderate to severe degree of hypertension (Dawber et al., 1952; Evans, 1957; and Simpson, 1960). The purpose of this study is to determine how frequently electrocardiographic evidence of left ventricular hypertrophy occurs in hypertensive patients and to note the most useful cardiographic findings indicative of left ventricular hypertrophy. At the outset, therefore, it is important to define normal blood pressure and this is exceedingly difficult. Bøe et al. (1957) in their study of the population of Bergen found they could establish a blood pressure pattern for a city population but their distribution curve did not enable them to determine where normal blood pressure levels according to age ceased and pathological hypertension began. This was also the experience of Master et al. (1950) in an earlier study of an industrial population. For our purposes we have chosen 160 and 95 mm. Hg systolic and diastolic pressures respectively as the upper limit of normality but, as there is no sharp division between clearly normal and clearly abnormal levels of blood pressure, this figure must be arbitrary.

MATERIAL AND METHODS

A group of 118 men and 215 women over the age of 40 years with a blood pressure over 160/95 mm. Hg was studied. The pressures were recorded with the patients at rest and the lowest of multiple readings was used, the diastolic pressure being taken at the junction between phases III and IV, i.e. the point at which the sounds abruptly fade (Pickering, 1955). None of the patients were in cardiac failure and none had any cardiac disease that might confuse the electrocardiographic pattern of left ventricular hypertrophy. Overweight patients (more than 10 per cent of expected weight for their sex, age, and height) were excluded from the study because of the equivocal value of their blood pressure readings. The patients were subdivided according to the height of the blood pressure as shown in Table I, those with a blood pressure over 160/95 up to 190/105 forming Group A, those with one over 190/105 up to 220/130 forming Group B, and those with one over 220/130 mm. Group C. When either the systolic or diastolic pressure alone exceeded the limit for the group, the diastolic reading was used as the deciding figure.

Conventional twelve-lead electrocardiograms were recorded. These included three limb leads, three augmented unipolar leads, and six unipolar precordial leads (Criteria Committee of the New York Heart Association, 1955; Goldberger, 1942). The criteria of left ventricular hypertrophy used were based on those described by Sokolow and Lyon (1949). The following abnormalities were recorded, the first three being generally accepted.

(1) Depression of S–T segments or low, flat, diphasic, or inverted T waves in the standard limb leads and V5 or V6.
(2) R waves in leads V5 or V6 exceeding 26 mm. and the sum of the R wave in V5 and the S wave in V1 exceeding 35 mm.

(3) Delayed onset of the intrinsicoid deflection in the leads V5 or V6.

(4) The incidence of left axis deviation, i.e. when the algebraic sum of Q, R, and S was positive in lead I and negative in lead III.

(5) Left bundle-branch block, when the QRS interval in V6 was 0.12 sec. or more.

(6) The amplitude of the S wave in V1, over 15 mm. being regarded as abnormal.

### TABLE I

**Electrocardiographic Findings in Clinical Hypertension**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
<th>Women</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All men</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>All women</td>
<td>A</td>
<td>B</td>
<td>C</td>
</tr>
<tr>
<td>S–T depression in V5 and V6</td>
<td>21</td>
<td>11</td>
<td>23</td>
<td>44</td>
<td>21</td>
<td>23</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>T wave changes</td>
<td>53</td>
<td>36</td>
<td>56</td>
<td>79</td>
<td>29</td>
<td>28</td>
<td>26</td>
<td>44</td>
</tr>
<tr>
<td>R in V5 or V6 &gt; 26 mm.</td>
<td>26</td>
<td>16</td>
<td>27</td>
<td>57</td>
<td>15</td>
<td>8</td>
<td>17</td>
<td>30</td>
</tr>
<tr>
<td>S in V1 + R in V5 or V6 &gt; 35 mm.</td>
<td>13</td>
<td>5</td>
<td>15</td>
<td>29</td>
<td>10</td>
<td>6</td>
<td>9</td>
<td>26</td>
</tr>
<tr>
<td>Delayed intrinsicoid deflection</td>
<td>14</td>
<td>10</td>
<td>20</td>
<td>12</td>
<td>7</td>
<td>0</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>L.A. deviation</td>
<td>47</td>
<td>29</td>
<td>53</td>
<td>71</td>
<td>66</td>
<td>59</td>
<td>67</td>
<td>81</td>
</tr>
<tr>
<td>L.B.B.Block</td>
<td>7</td>
<td>7</td>
<td>3</td>
<td>20</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>S in V1 &gt; 15 mm.</td>
<td>32</td>
<td>23</td>
<td>35</td>
<td>50</td>
<td>33</td>
<td>27</td>
<td>30</td>
<td>59</td>
</tr>
<tr>
<td>Normal electrocardiogram</td>
<td>22</td>
<td>25</td>
<td>25</td>
<td>0</td>
<td>26</td>
<td>28</td>
<td>26</td>
<td>22</td>
</tr>
</tbody>
</table>

The incidence of electrocardiographic changes is expressed as the percentage with the abnormality, arranged according to sex and blood pressure grouping in 333 patients (118 men and 215 women).

Group A (44 men and 86 women) had blood pressures over 160/95 to 190/105, group B (60 men and 102 women) had pressures over 190/105 to 220/130, and Group C (14 men and 27 women) pressures over 220/130 mm. Hg.

### FINDINGS IN OUR PATIENTS

Table I shows that in both sexes electrocardiographic changes directly attributable to left ventricular hypertrophy were most frequent in the group with the highest blood pressure.

S–T depression in V5 or V6 was present in about a fifth of the total number of patients. T wave abnormalities occurred in almost half, and were more common in the severe grades of hypertension, especially in men.

A high voltage R in V5 and 6 was present in 26 per cent of men and 15 per cent of women, but in patients with severe hypertension (viz. Group C), it was seen in 57 per cent of the men and 30 per cent of the women. In 13 per cent of men and 10 per cent of women combined voltages of more than 35 mm. were obtained by adding the amplitude of S in V1 to R in V5 or V6.

Delay of more than 0.05 sec. in the onset of the intrinsicoid deflection in V5 and V6 occurred in about 10 per cent of patients.

Left axis deviation was usually present in patients with the higher ranges of blood pressure. 115 of 140 women and 43 of 54 men with left axis deviation had a horizontal heart position cardiographically.

Left bundle-branch block was present in 8 men and 5 women, and was more common in the higher grades of hypertension in men.
A deep S in V1 was present in 32 per cent of men and 33 per cent of women; of patients in the group with the highest blood pressure a deep S in V1 was present in the electrocardiograms of 50 per cent of men and 59 per cent of women.

**Fig. 1.**—Electrocardiogram of patient with clinical hypertension (B.P. 220/130 mm.). A deep S in V1 is the most obvious electrocardiographic sign suggesting left ventricular enlargement, as in many patients.

**Discussion**

Approximately 25 per cent of all patients in this series had normal electrocardiograms. It was only in the group of men with blood pressures of 220/130 mm. Hg, or more, that normal records
were absent, and this is in keeping with the more eventful clinical course of severe hypertension in men compared with women. Selzer et al. (1958) and Scott (1960) found that the electrocardiogram gave a reasonably accurate picture of left ventricular hypertrophy, as later verified at autopsy. It would seem, therefore, that the group with normal records may represent a relatively benign form of hypertension without cardiac enlargement, which Evans would prefer to call hypertonia. Moreover, the difficulty in defining the difference between normal blood pressure and hypertensive levels has been emphasized.

The classical cardiographic changes of left ventricular hypertrophy may be difficult to recognize in the greatly enlarged or transverse type of heart. In these patients the record obtained at V5 and V6 will not record the maximum electrical events due to the left ventricle as the transition zone is displaced to the left. For the same reason the voltage of R in V6 and the sum total of S in V1 and R in V6 will also tend to be too low, and the intrinsicoid deflection will not be delayed. Wilson et al. (1944) and Sokolow and Lyon (1949) found delayed intrinsicoid deflection in left ventricular surface leads in a high proportion of their patients with left ventricular hypertrophy but they did not relate these findings to the heart size and position. A deep S in V1 will more accurately measure left ventricular depolarization, since it is known that hypertrophy of the left ventricle will open the spatial QRS loop of the vectorcardiogram and create a prominent posterior component during the last half of the QRS cycle. The electrocardiogram will record depolarization in this region as a deep S wave in V1 and V2 (Lamb, 1957).

Left axis deviation occurred more frequently in the patients with higher ranges of blood pressure, being usually related to horizontal cardiographic heart position and accompanied by other criteria of left ventricular hypertrophy. This is not necessarily inconsistent with Grant's (1956) view that marked left axis deviation is caused by an alteration in the direction of QRS forces due to a left bundle-branch conduction defect with normal QRS duration.

Left bundle-branch block is probably not simply an expression of delay in depolarization over the left ventricle (Rasmussen and Moe, 1948) although in this series in men it was more frequent in the higher ranges of blood pressure than in minor degrees of hypertension.

S–T segment and T wave changes in the presence of left ventricular hypertrophy are largely due to alteration in the process of ventricular repolarization (Wilson et al., 1944). However, the relatively large number of patients with T wave changes over the left ventricular surface leads, particularly in the more severely hypertensive men, would suggest myocardial ischaemia as a possible contributory factor. Bridges et al. (1946) and Leishman (1951) reported reversal of T wave changes to normal following sympathectomy for hypertension. Leishman (1951) suggested a relative ischaemia of the enlarged cardiac muscle fibres as the cause of the changes, in addition to coronary atherosclerosis, and this was confirmed by the concurrent decrease in heart size reported in some of their cases.

**SUMMARY AND CONCLUSIONS**

The 12-lead electrocardiograms of 333 hypertensive subjects over the age of 40 years were studied. Six parameters of the electrocardiographic diagnosis of left ventricular hypertrophy were examined and the changes were related to the height of the blood pressure in these patients. The abnormalities indicating left ventricular hypertrophy were more common in the higher grades of hypertension.

Approximately 25 per cent of subjects with raised blood pressure were found to have none of these criteria for the diagnosis of left ventricular hypertrophy and may represent in this respect a benign form of hypertension or hypertonia.

The most useful findings in the diagnosis of left ventricular hypertrophy in the twelve-lead electrocardiogram appeared to be left axis deviation, T wave changes in the standard limb leads and V5 or V6, and a deep S of more than 15 mm. amplitude in V1.

We wish to thank Dr. Frances Gardner for her advice and for her criticism of this work.
REFERENCES
