Orthogonal electrocardiogram, apex cardiogram, and atrial sound in normotensive and hypertensive 50-year-old men

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The prevalence of signs of heart involvement was studied non-invasively in a group of untreated hypertensives (n=35) and a reference group (n=73), all derived from a random population sample of 50-year-old men. Signs of left ventricular hypertrophy were studied by means of orthogonal electrocardiography and conventional electrocardiography. Signs of decreased distensibility of the left ventricle were studied by apex cardiography and registration of atrial sounds.

Left ventricular hypertrophy among hypertensives was significantly more common according to orthogonal electrocardiography (33%) than according to conventional electrocardiography (9%), indicating that the former may be a better method for detection of left ventricular hypertrophy than the latter. In the hypertension group the amplitude of the R wave in lead X on orthogonal electrocardiography was positively correlated to casual diastolic blood pressure (r=0.40) and to diastolic blood pressure after one hour's rest (r=0.65). The degree of pressure load leading to left ventricular hypertrophy seems to be better reflected by resting than by casual blood pressure.

There was no hypertensive subject with both signs of left ventricular hypertrophy on orthogonal electrocardiography and either an a/H ratio over 15 per cent or an abnormal atrial sound, indicating two different forms of cardiac involvement as the result of hypertension. Casual blood pressures became normal during rest in hypertensives with a/H ratio over 15 per cent on apex cardiography or abnormal atrial sound, but not in hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiography.

The natural history of hypertension is extremely varied. One-third of a hypertensive population survived to old age without signs of organic damage while in others the prognosis was poor (Fry, 1974). Hypertensives with signs of left ventricular hypertrophy on electrocardiography have a poorer prognosis than hypertensives with a normal electrocardiogram (Bechgaard, 1967; Sokolow and Perloff, 1961; Kannel et al., 1970). In mild to moderate hypertension, antihypertensive treatment had a beneficial effect in hypertensives with signs of cardiac abnormality on electrocardiography or x-ray, while no benefit could be shown in hypertensives without such abnormality (Veterans Administration Cooperative Study Group on Antihypertensive Agents, 1972). Thus, it seems important for prognostic and therapeutic purposes to diagnose cardiac abnormality in hypertensives. Conventional electrocardiography and chest x-ray are, however, normal in many hypertensives, partly because of low sensitivity, and are less suitable for quantification of left ventricular hypertrophy (Sokolow and Lyon, 1949; Gamboa, Hugenholtz, and Nadas, 1965; The National Center for Health Statistics, 1966; Tibblin, 1967, Romhilt and Estes, 1968; Sannerstedt, Bjure, and Varnauskas, 1970; McCaughan, Littman, and Pipberger, 1973).

Vectorcardiography and orthogonal electrocardiography have been shown to be superior to conventional electrocardiography in the diagnosis of left ventricular hypertrophy (Gamboa et al., 1965; McCaughan et al., 1973). Apex cardiography, especially the a wave in the apex cardiogram, and the analogous acoustical event, the atrial sound, provide non-invasive ways of assessing changes in
left ventricular distensibility (Bethell and Nixon, 1973; Gibson et al., 1974; Tavel, 1974). In the present investigation signs of left ventricular hypertrophy were studied by conventional and orthogonal electrocardiography (Frank), and signs of decreased left ventricular distensibility by apex cardiography and registration of atrial sounds phonocardiographically.

The aim of the present study was to determine the prevalence of heart involvement, assessed by these non-invasive methods, in groups of untreated hypertensive and normotensive men, homogeneous with respect to age, and to relate the findings to casual blood pressure and resting blood pressure.

**Subjects**

From a screening examination, which was part of a multifactorial, primary preventive trial (Wilhelmsen, Tibblin, and Werko, 1972) in a randomly selected third (n=1122) of the 50-year-old male population in Göteborg, Sweden, all subjects with untreated, essential hypertension were allocated to a hypertension group (n=35) (see Fig. 1). The diagnosis of essential hypertension was based on a casual blood pressure above 175 mmHg (23.3 kPa) systolic and/or 115 mmHg (15.3 kPa) diastolic on two separate occasions two weeks apart (the casual blood pressure values given in the tables refer to the first measurement), and a negative standard diagnostic examination for secondary hypertension (Wilhelmsen, Berglund, and Werko, 1973). Thus, one subject with chronic glomerulonephritis was excluded (Fig. 1). For the hypertension group, 18 subjects were classified as belonging to WHO stage 1, 13 to stage 2, and 4 to stage 3.

A reference group (n=73) was obtained from the same trial by drawing a 10 per cent subsample at random. The non-participation rate in the subsample was 18 per cent (20/110). Five subjects in the reference group fulfilled the criteria for hypertension and joined the hypertension group. Twelve subjects in the reference group were excluded, 1 with mitral stenosis, 1 with aortic regurgitation, 5 who were on antihypertensive treatment, and 5 with casual blood pressure above 175 mmHg (23.3 kPa) systolic and/or 115 mmHg (15.3 kPa) diastolic at screening but not at the subsequent blood pressure control two weeks later. No subject in the hypertension group had a history of valvular heart disease.

In addition to the above-mentioned groups, 3 untreated, male hypertensives (aged 45, 50, and 50 years) with casual blood pressures above 240 mmHg (31.9 kPa) systolic and 160 mmHg (21.3 kPa) diastolic, recruited from the same screening examination, were examined in order to obtain data from subjects with extremely high blood pressures. These 3 subjects all belonged to WHO stage 3.

Orthogonal electrocardiograms and conventional electrocardiograms were recorded in all subjects (n=111). Registration of apex cardiogram, atrial sounds, and resting blood pressure were performed in a randomly selected half of both the reference (n=36) and the hypertension group (n=19) and in the 3 hypertensives with extremely high blood pressure. Analysis of each variable was performed without knowledge of the results of the other examinations or to which group the subject belonged. Resting blood pressure values were, for technical reasons, lacking for 4 subjects selected for apex cardiography; all 4 belonged to the reference group. At analysis of signs of left ventricular hypertrophy on electrocardiography, 2 subjects in the hypertension group and 1 in the reference group with left or right bundle-branch block or left anterior hemiblock, were excluded, since changes in left ventricular activation have an influence on the amplitudes (Fernandez, Scabat, and Lenegre, 1970). In 4 subjects in the reference group (11%) and 1 hypertensive subject (5%), an acceptable apex cardiogram could not be recorded.

**Methods**

Conventional electrocardiograms, apex cardiograms, phonocardiograms, and resting blood pressures were all registered on a direct writing ink-jet 7-channel
The three scalar electrocardiographic leads X, Y, and Z were simultaneously recorded on magnetic tape (Hellige) using the Frank's corrected orthogonal lead system (Frank, 1956). Computer analysis was performed according to Arvedson (1968, 1973) with a sampling frequency of 200 per second. The following criteria for left ventricular hypertrophy were used: the amplitude of the R wave in lead X (R_x) above 1.8 mV, the R wave in lead X and the S wave in lead Y (R_x+S_y) above 1.9 mV or the R wave in lead Z (R_z) above 1.3 mV. Since most electrocardiographic measurements are not normally distributed the amplitude limits were drawn at the upper 97.5th centile in the reference group in analogy with a previous study (McCaughan et al., 1973). The upper 97.5th centile in the reference group was also studied for R_x+R_z (2.8 mV), the spatial maximal amplitude (2.2 mV), ventricular activation time (0.06 s for both R_x and the spatial maximal amplitude), the duration of the P wave (0.13 s) and the following amplitudes of the P wave in the orthogonal electrocardiogram: sum of positive P amplitudes in lead X and lead Z (0.17 mV), positive P amplitude in lead Z (0.07 mV) (Ishikawa, Kini, and Pipberger, 1973). Conventional electrocardiograms were recorded as standard 12 lead electrocardiograms using the right arm as the indifferent electrode for the praecordial leads. Electrocardiograms were coded in accordance with the Minnesota code (Blackburn et al., 1960). Amplitude measurements in praecordial leads were done in accordance with the revision for CR leads (Åstrand et al., 1967). As signs of left ventricular hypertrophy, a combination of amplitude criteria, 3:1 or 3:3, and ST or T criteria, 4:1-3 or 5:1-3, were used (Sokolow and Lyon, 1949).

Apex cardiograms and phonocardiograms were recorded during the resting period preceding the measurement of resting blood pressure. The apex cardiogram was recorded simultaneously with electrocardiogram lead II and a phonocardiogram from the third left intercostal space parasternally with the subject in the left lateral position during relaxed expiratory apnoea. The pick-up was held by hand at the point of maximal impulse of the apex beat. The a wave percentage amplitude of the total deflection of the apex cardiogram (H), was calculated as the mean of five consecutive beats (Fig. 2). An a/H ratio above 15 per cent was considered abnormal (Tavel et al., 1965; Epstein et al., 1968; Voigt and Friesinger, 1970). Registration of the apex cardiogram by two different examiners with one hour's interval, the second examiner not being allowed to see the recording made by the first examiner, gave a correlation coefficient of 0.88 (n = 81) between the two calculated a/H.
FIG. 2 Apex cardiogram and phonocardiogram in a hypertensive subject in the left lateral position. Upper panel: apex cardiogram with an abnormal a wave (a). The a wave percentage amplitude of the total deflection of the apex cardiogram (H) was 22 per cent calculated as the mean a/H ratio of 5 consecutive beats. The apex cardiogram was recorded simultaneously with electrocardiogram lead II, phonocardiogram from the third intercostal space parasternally, and the first derivative of the apex cardiogram (dA/dt). Screening blood pressure (BP) was 182/120 mmHg (24.2/16.0 kPa), resting blood pressure was 122/80 mmHg (16.2/10.6 kPa). Lower panel: phonocardiogram from the apex region showing an atrial sound of intensity 4. The phonocardiogram was recorded with six frequency ranges giving five nominal frequencies from 25 to 400 Hz and one aural frequency range.
ratios, with a standard deviation of a single determination of 2-5 per cent.

Phonocardiograms were recorded during relaxed expiratory apnoea simultaneously with electrocardiographic lead II from the routine clinical auscultation points including registration over the apex in the left lateral position (Fig. 2). One of the authors (I.W.) with long experience of phonocardiography, classified the atrial sound in the phonocardiogram with regard to its intensity, on a five-point subjective scale, where I was judged as 'very weak' and 5 as 'very strong'. Atrial sounds of intensity 4 and 5 were arbitrarily classified as abnormal. A blind re-evaluation of the phonocardiograms with respect to the intensity of the atrial sound was performed after one year. One atrial sound in the hypertension group that had previously been evaluated as 4 was now judged to be 3. This subject had an abnormal a wave in the apex cardiogram. At the latter evaluation the proportion of subjects with abnormal atrial sounds was not significantly higher in the hypertension group than in the reference group.

An abnormal atrial sound is said to have the same haemodynamic significance as an abnormal a wave in the apex cardiogram (Tavel, 1974; Bethell and Nixon, 1973). Good correlation has been shown between the relative magnitude of the a wave and an index of the left ventricular distensibility (Gibson et al., 1974). Therefore, an abnormal a wave in the apex cardiogram and an abnormal atrial sound were both regarded as signs of decreased left ventricular distensibility.

Standard methods were used for calculation of the mean ($\bar{x}$), the standard deviation ($s_x$), the standard error of the mean ($s_x$), and the linear correlation coefficient ($r$). The hypothesis of no differences in means was tested with Student's t-test or the Wilcoxon Rank Sum Test for two samples. The hypothesis of no differences between paired observations in the same subjects was tested with Student's t-test for paired observations. Where the standard deviation and range are given the t-test was used; where the standard error of the mean and range are given the Wilcoxon test was used. The hypothesis of no differences in proportions between two groups was tested with the Fisher exact test for $n < 60$ and with the $\chi^2$ test for $n \geq 60$. The hypothesis of no differences in proportions when examining one group of subjects with two separate methods was tested with McNemar's test for correlated proportions (Remington and Schork, 1970).

The formula $\sqrt{\frac{y_n^2}{2n}}$ was used to calculate the standard deviation of a single determination in a series of determinations of which the mean difference was not significant (Dahlberg, 1940). Only two-tailed tests were used and differences were considered significant for $P$ values 0.05 or less.

### Results

#### Blood pressure and heart rate

In both the hypertension and the reference group the resting systolic and diastolic blood pressures and heart rates were significantly lower ($P < 0.001$) than the casual (Table 1). The hypertensives showed significantly higher ($P < 0.05$) casual heart rate than the reference group, but after one hour's rest in a sound-protected room there was no significant difference. The differences in resting systolic and diastolic blood pressure between the two groups were significant ($P < 0.001$).

### Table 1  Casual and resting blood pressure and heart rate in subjects randomly selected for apex cardiography

<table>
<thead>
<tr>
<th></th>
<th>Hypertension group</th>
<th>Reference group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. = 19</td>
<td>No. = 32</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$</td>
<td>Range</td>
</tr>
<tr>
<td>Systolic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casual</td>
<td>197</td>
<td>(220–164)</td>
</tr>
<tr>
<td>Resting</td>
<td>154</td>
<td>(192–111)</td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casual</td>
<td>119</td>
<td>(138–98)</td>
</tr>
<tr>
<td>Resting</td>
<td>96</td>
<td>(120–64)</td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casual</td>
<td>84</td>
<td>(114–61)</td>
</tr>
<tr>
<td>Resting</td>
<td>61</td>
<td>(74–43)</td>
</tr>
</tbody>
</table>

Conversion factor from Traditional to SI Units: 1 mmHg = 0.133 kPa.
TABLE 2  Selected scalar measurements on orthogonal and conventional electrocardiography that discriminate between hypertension and reference groups

<table>
<thead>
<tr>
<th></th>
<th>Hypertension group</th>
<th>Reference group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Orthogonal electrocardiogram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rx &gt; 1:8 or Rz &gt; 1:3 or</td>
<td>11/33</td>
<td>33</td>
</tr>
<tr>
<td>Rx + S_y &gt; 1:9 mV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conventional electrocardiogram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3:1 or 3:3 plus</td>
<td>3/33</td>
<td>9</td>
</tr>
<tr>
<td>3:1 or 3:3 alone</td>
<td>6/33</td>
<td>18</td>
</tr>
</tbody>
</table>

**Orthogonal electrocardiogram and standard 12-lead electrocardiogram**

The proportion of hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiogram (11/33, 33%) was significantly higher (P < 0.001) than that in the reference group (2/72, 3%), see Table 2. The proportion of hypertensives with signs of left ventricular hypertrophy with conventional electrocardiogram was 9 per cent (3/33), compared with 3 per cent (2/72) in the reference group. Minnesota code 3:1 or 3:3 alone was seen in 18 per cent (6/33) of the hypertensives and in 7 per cent (5/72) of the reference group. The proportion of hypertensives with signs of left ventricular hypertrophy by orthogonal electrocardiogram was significantly higher (McNemar, P < 0.025) than by conventional electrocardiogram. In the reference group there was no overlapping between the two methods, i.e. two subjects showed signs of left ventricular hypertrophy by orthogonal electrocardiogram, and another two by conventional electrocardiogram. Of the hypertensives, one fulfilled the criteria for left ventricular hypertrophy with conventional electrocardiogram only, two with both methods, and nine with orthogonal electrocardiogram only. Of these nine subjects, none had a Minnesota code 4:1-3 or 5:1-3, i.e. ST or T changes on conventional electrocardiogram, and thus the only sign of left ventricular hypertrophy in these nine hypertensives was high QRS amplitude on orthogonal electrocardiogram. Other measurements, such as Rx + Rz, the spatial maximal amplitude, ventricular activation time, or the duration or amplitude of the P wave did not improve the discrimination between the hypertension and reference group.

**Atrial cardiogram and atrial sound**

The proportion of hypertensives with a/H ratio above 15 per cent (7/18, 39%) was significantly higher (P < 0.05) than found in the reference group (2/32, 6%), see Fig 3. The mean a/H ratio in the hypertension group (X=13.9, s_x=1.3, range 6 to 28) was significantly higher (P < 0.01) than the mean in the reference group (X=9.9%, s_x=1.0, range 0 to 33).

Atrial sounds of intensity 1 to 3 were common in both groups (Fig. 4). The proportion of hypertensives with atrial sounds of intensity 4 and 5 (5/19, 26%) was significantly higher (P < 0.05) than in the reference group (1/36, 3%). All abnormal atrial sounds had their maximum intensity over the apex in the left lateral position. No measurements of the atrial sound percentage amplitude of the first heart sound could discriminate between the groups. Three of the seven hypertensives who had a/H ratio above 15 per cent also had abnormal atrial sounds. One of the two subjects in the reference group, who had an abnormal a wave on the apex cardiogram, had diabetes mellitus and a high casual blood pressure (172/98 mmHg (22.9/ 13.0 kPa)). The other subject with a/H ratio 33 per cent had angina pectoris. He also had an abnormal atrial sound (intensity 5) and ST and T changes on conventional electrocardiography.
Heart involvement in hypertension

Signs of left ventricular hypertrophy and a/H ratio

The mean a/H ratio in the group of hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiogram (X = 9.0%, sX = 1.0, range 6 to 12, n = 5) was significantly lower (P < 0.01) than in the group of hypertensives without signs of left ventricular hypertrophy (X = 16.1%, sX = 1.8, range 7 to 28, n = 11). In this analysis two subjects with ventricular conduction defects were excluded.

Blood pressure and type of heart involvement

There was no hypertensive showing both signs of left ventricular hypertrophy on orthogonal electrocardiogram and an abnormal a wave on the apex cardiogram or an abnormal atrial sound, i.e. signs of decreased distensibility of the left ventricle. Table 3 shows that hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiography had significantly higher (P ≤ 0.02) casual diastolic blood pressure and significantly higher systolic (P < 0.05) as well as diastolic (P ≤ 0.01) resting blood pressures than the group of hypertensives with signs of decreased left ventricular distensibility. There was no significant difference in heart rate between the two groups. If the two hypertensives with only abnormal atrial sound are removed from the group with a/H ratio greater than 15 per cent, there still was a significant difference (P ≤ 0.02) in resting diastolic blood pressure between the two groups.

Findings in three hypertensives with extremely high blood pressure

The casual blood pressures for these three subjects were 240, 268, and 250 mmHg (31.9, 35.6, and 33.3 kPa) systolic and 172, 164, and 160 mmHg (22.9, 21.8, and 21.3 kPa) diastolic, and the casual heart rates were 99, 72, and 89 beats/min. The third subject was treated immediately; the resting blood pressures for the other two were 196/130 mmHg (26.1/17.3 kPa) and 198/107 mmHg (26.3/14.2 kPa), respectively. All three had signs of left ventricular hypertrophy on orthogonal electrocardiogram (Rx = 2.3, Rx + Sx = 1.9, and Rx = 2.5 mV, respectively) and showed abnormal a/H ratios (50, 32, and 27%).

### Table 3 Blood pressure and heart rate in two groups of hypertensives with signs of left ventricular hypertrophy (Rx > 1.8 or Rx > 1.3 or Rx + Sx > 1.9 mV) and signs of lowered left ventricular distensibility (a/H ratio > 15% or atrial sound of intensity 4 or 5)

<table>
<thead>
<tr>
<th>Type of left ventricular involvement</th>
<th>Hypertrophy</th>
<th>Lowered distensibility*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. = 5/19</td>
<td>s(\bar{X})</td>
<td>s(\bar{X})</td>
</tr>
<tr>
<td>Casual</td>
<td>Rx</td>
<td>Range</td>
</tr>
<tr>
<td>Systolic</td>
<td>206</td>
<td>(216-198)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>125</td>
<td>(132-118)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>79</td>
<td>(114-61)</td>
</tr>
<tr>
<td>Resting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>171</td>
<td>(192-146)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>107</td>
<td>(120-98)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>59</td>
<td>(74-50)</td>
</tr>
</tbody>
</table>

*One subject with left bundle-branch block was excluded since left ventricular hypertrophy on orthogonal electrocardiography could not be analysed.

Conversion from Traditional to SI Units: 1 mmHg ≈ 0.133 kPa.
Two had abnormal atrial sounds as well. These three hypertensives with extremely high blood pressures thus showed signs of both left ventricular hypertrophy and decreased left ventricular distensibility.

**Relations between signs of left ventricular hypertrophy on orthogonal electrocardiogram, a/H ratio, and blood pressure**

Linear correlation analysis for the relation between \( R_x \) on orthogonal electrocardiogram and resting diastolic blood pressure in the hypertension group (see Fig. 5) gave \( r = 0.65 \) (\( n = 17, P < 0.01 \)). When the subjects with extremely high blood pressure were included in the linear regression analysis, the \( r \) value rose to 0.74. A significant positive correlation was also found in the hypertension group for the relation between \( R_x \) and resting systolic blood pressure \( (r = 0.50, n = 17, P < 0.05) \), as well as casual diastolic blood pressure \( (r = 0.40, n = 33, P < 0.05) \). There was also a significant positive correlation in the hypertension group between resting diastolic blood pressure and \( R_x + S_y \) \( (r = 0.67, n = 17, P < 0.01) \) and spatial maximal amplitude \( (r = 0.55, P < 0.05) \). There was no significant linear correlation in the hypertension group either for the relation between casual systolic blood pressure and \( R_x \) \( (r = 0.03, n = 33) \), \( R_x + S_y \) \( (r = 0.01) \), \( R_z \) \( (r = 0.33) \), or spatial maximal amplitude \( (r = 0.05) \). In the hypertension group \( R_x \) above 1.8 mV was only seen when the casual diastolic pressure exceeded 110 mmHg (14.6 kPa) or resting diastolic pressure exceeded 95 mmHg (12.6 kPa). In the reference group there was no significant correlation of \( R_x \) either with casual or with resting blood pressure. No significant linear correlations of a/H ratio to blood pressure were found either in the hypertension, or in the reference group.

**Discussion**

The subjects of the study were selected at random from a total male population. As most of the variables studied are age and sex dependent, we chose to study normotensive and hypertensive men of the same age. To our knowledge, no previous study of cardiac function in relation to blood pressure has used these quantitative, non-invasive methods in subjects derived from a screening examination for blood pressure in a total population.

In the hypertension group, orthogonal electrocardiography showed a higher prevalence of signs of left ventricular hypertrophy than conventional electrocardiography. Since the prevalence of signs of left ventricular hypertrophy in the reference group was the same with both methods, orthogonal electrocardiography seems to be a better method for discrimination of left ventricular hypertrophy in hypertension. Furthermore, orthogonal electrocardiography is more practical, since simple QRS amplitude measurements are sufficient, while on conventional electrocardiography, QRS amplitude measurements have to be combined with other criteria, such as ventricular activation time or ST or T changes for the diagnosis of left ventricular hypertrophy (Sokolow and Lyon, 1949). Measurement of the spatial maximal amplitude was found redundant, as had also been found in a previous study (McCaughan et al., 1973).

Atrial sounds can be recorded in the majority of
normal individuals aged 50 (Recta et al., 1972) and
this is also evident from the present study. This
means that the atrial sound as well as the a wave in
the apex cardiogram has to be in some way quanti-
tied to be of use in differentiating normal from
abnormal. The judgement had to depend on the
empirical judgement of one of the authors. Even if
the hypertensives with only abnormal atrial sound
are removed from the hypertensives with a/H ratio
greater than 15 per cent the figures still allow the
separation of two types of heart involvement in
hypertension. Apex cardiogram and recording of
atrial sounds complement each other, since the
overlapping between the two methods was less than
often claimed (Tavel et al., 1965; Epstein et al.,
1968; Tavel, 1974). Different filter properties of the
thoracic wall for sound and volume displacement
as well as the difference in origin mechanisms for the a wave and the atrial sound may explain the
finding that there was no total overlapping.

The morphological evidence of left ventricular
hypertrophy is increased left ventricular mass
(Rombilt and Estes, 1968). Mass itself has been
shown to be a poor predictor of distensibility, while
wall thickness was an excellent predictor (Grossman
et al., 1974). The mean a/H ratio in patients with
signs of left ventricular hypertrophy on conventional electrocardiography has been shown to be
significantly higher than in patients without left
ventricular hypertrophy (Gibson et al., 1974). In
the present study mean a/H ratio was significantly
lower in hypertensives with signs of left ventricular
hypertrophy on orthogonal electrocardiography
than in hypertensives without these signs, which is
in contrast to the above-mentioned studies.

Hypertensives in the present study with signs of left
ventricular hypertrophy had no signs of decreased
left ventricular distensibility. One reason for this
might be that the hypertrophy was not of the degree
giving rise to a sufficient increase in wall thickness
to give distensibility changes. The degree of hyper-
trophy in our hypertensives was probably mild
compared with the hypertrophy in the above-
mentioned studies. Another explanation could be
that a relative strengthening of the systolic wave in
relation to the a wave resulting from the hyper-
trophy could have led to a decrease in the a/H ratio.
In two of the three subjects with extreme levels
of casual blood pressure the decreased left ventricular
distensibility might have been caused by left ven-
tricular hypertrophy, namely in Cases 1 and 3 where
the QRS amplitudes on orthogonal electrocardi-
ography were very large.

The signs of decreased left ventricular distensi-
ality in the hypertension group could not be related
to hypertrophy, at least not electrocardiographically
proven. Decreased distensibility of the left ventricle
is not a specific finding of left ventricular hyper-
trophy but is also found in ischaemic heart disease
(Voigt and Friesinger, 1970; Martin, Shaver, and
Leonard, 1972). There was, however, no subject
in the hypertension group with symptoms of angina
pectoris as judged by one of the authors (G.B.).

In any consideration of ventricular distensibility, it
must be borne in mind that the diastolic pressure-
volume curve is not linear. Thus, a structurally
normal ventricle may exhibit the same low distensi-
bility as a scarred or hypertrophied ventricle if its
filling pressure is raised sufficiently (Levine, 1972).
Another explanation for the signs of decreased left
ventricular distensibility could be abnormal
distension of the left ventricle during the atrial con-
traction partly caused by increased central blood
volume. Increased central blood volume has been
described in essential hypertension (Ulrych et al.,
1969; Ellis and Julius, 1973).

A model of the circulation has been described
in which hypertension initially resulting from in-
creased blood volume and/or cardiac output can
subsequently become converted to a hypertension
with high peripheral resistance and normal or
lowered cardiac output (Guyton and Coleman, 1969).
In the present study the resting blood
pressure was returned to normal in the hyper-
tensives with signs of decreased left ventricular
distensibility, but not in the hypertensives with
signs of left ventricular hypertrophy. This might
suggest that the former group is in an earlier state
of hypertensive disease while the other is in a later
state with raised peripheral resistance. Whether the
former group will pass over into a hypertension
of the latter type or not must be studied prospectively.
In advanced left ventricular hypertrophy an
abnormal a wave in the apex cardiogram is a very
common finding (Tavel et al., 1965; Epstein et al.,
1968). It might be that an initial abnormal a wave
caused by volume stiffness of the left ventricle can
return to normal, and then when the left ventricular
hypertrophy becomes pronounced or fibrosis is
added, structural changes in the left ventricular
wall can again give rise to an abnormal a wave.

The correlation coefficient between blood pres-
sure and amplitude measurements on orthogonal
electrocardiography were higher in the present study
than in a previous one (McCaughan et al., 1973)
(r = 0·65 for Rx in the present study, compared
with r = 0·23 for Rx in the previous investigation).
In the latter investigation, however, a group of
patients with severe hypertensive heart disease had
large Rx amplitudes and a concomitant decrease in
Rx. The homogeneity of our hypertension group of
subjects with early hypertension probably implies
that the amplitude of the $R_x$ will reflect the degree of left ventricular hypertrophy better than will $R_y$ in the latter investigation.

In the present investigation there was better correlation between QRS amplitude measurements and resting blood pressure than to casual blood pressure, indicating that resting blood pressure better reflects the degree of pressure load leading to left ventricular hypertrophy. The fact that these hypertensives were untreated and never had been treated probably also implies that the blood pressures were more representative of the pressure load on the left ventricle than blood pressure in treated patients can be.

It is evident from the present study that the orthogonal electrocardiogram, the apex cardiogram, and the recording of atrial sounds complement each other in the investigation of hypertensive heart disease. The combination of the three methods made it possible to detect heart involvement in the majority of hypertensive subjects. The numbers with full studies are, however, small. Prospective follow-up must be undertaken in larger groups to study the haemodynamic pattern and prognosis in hypertensives with the different abnormal findings. These studies are now proceeding.

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