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Electrocardiographic Changes in 50-Year-Old Men with Different Levels of Diastolic Arterial Blood Pressure

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ABSTRACT

An ECG study of P wave abnormalities and signs of left ventricular hypertrophy (LVH) was performed in a series of 76 50-year-old men with hypertension detected in a population study. Two control groups were obtained from the same study. P wave abnormalities were common (36/76) among the hypertensives but were frequently seen also among the controls (41/152). Thus it seems that P wave abnormality, with the current criteria, is not a reliable early sign to discriminate hypertensives from persons with a normal blood pressure. High QRS voltage or left axis deviation was not uncommon in the control groups, while any combination of at least two criteria of LVH (high voltage, prolonged ventricular activation time, ST–T changes or left axis deviation) was far more common in the hypertensive group. In cases with high voltage as the single criterion of LVH examination of the P wave may be helpful to discriminate hypertensives from persons with a normal blood pressure.

P wave changes indicating left atrial abnormality have previously been described in connection with hypertension (6). In 1966 Tarazi et al. reported on ECG evidence of left atrial abnormality as an early sign of cardiac involvement in systemic arterial hypertension (9). Various ECG criteria for the diagnosis of left ventricular hypertrophy (LVH) have been suggested. Subsequently, the accuracy of these criteria has been studied by a number of investigators (1) but it is generally agreed that there is no satisfactory single such criterion for LVH.

The purpose of the present investigation was to evaluate electrocardiographic P wave abnormalities in hypertension by comparing a group of hypertensives from a population study with two control groups from the same population study. The signs of atrial abnormality were also related to changes indicating LVH.

MATERIAL

In 1970–73 a broad screening of risk factors for cardiovascular disease has been performed in 50-year-old men in the city of Uppsala, including all men born in 1920–24. The participation rate was 84%. All persons having a diastolic blood pressure in the recumbent resting position of 105 mmHg or more, measured on two separate occasions, were judged as hypertensives requiring treatment. With this criterion the total number of hypertensive men born 1920–22 was 80 (5.7%) of an investigated population of 1 415 persons. Of the 80 in the hypertensive group (H) two were excluded from the ECG study because of right bundle branch block (RBBB) and two more persons because of atrial fibrillation. The H group therefore includes 76 men.

One control group (M) is a random sample of 76 men, matched for age, from the same population study with a diastolic blood pressure of 85–100 mmHg.

A further control group (L) is a random sample of 76 men, matched for age, from the same study with a diastolic pressure of 80 mmHg or less.

Persons previously treated for hypertension but still having a diastolic blood pressure of 105 mmHg or more were included in the H group while those treated with a diastolic blood pressure of 100 mmHg or less were excluded from the study.

None of the 228 persons in the three groups had digitalis or quinidine medication or was aware that he suffered from heart disease. Nobody had a kyphoscoliotic spine or other thoracic configuration anomaly of significance for the study. In the H group all persons were examined by heart auscultation—without detection of valvular disease—and most of them also by chest radiogram. In the control groups most of the persons had mass chest X-ray and about half of them had heart auscultation. None of these investigations revealed any sign of cardiovascular disease.

METHODS

On the same day as the general health survey was performed, an ECG was taken. The persons were resting in the recumbent position. A 12-lead ECG (I, II, III,

Table I

No. of cases in each group=76. H=high (≥105 mmHg); M=medium (85–100 mmHg); L=low (<85 mmHg) diastolic arterial blood pressure

	P wave				QRS and ST-T						
						High voltage				Left-axis deviation	
Group	Normal	Duration >11 cs	Peak interval >4 cs	Terminal force <−3 μV·s	Normal	Combined	Single	Prolonged VAT	ST-T changes	Combined	Single
H	40	29	11	5	30	18	17	5	6	13	8
M	60	9	7	1	44	4	10	0	2	2	18
L	51	19	7	3	59	0	10	0	0	0	7

aVR, aVL, aVF, V_{1-6}) was recorded with the conventional amplification (1 mV–10 mm) and with a paper speed of 50 mm per second, using a direct-writing ECG machine (Mingograf 61, Siemens-Elema Ltd., Solna, Sweden). The obtained paper speed was intermittently controlled and the deviation from the correct paper speed was always less than 2%.

The duration of the P wave in lead II, the maximum bipeak interval in bifid P waves in any lead (10) and the P terminal force (4) in V_1 was measured. The terminal force of the P wave was defined as the product of the maximum amplitude and the duration of the terminal portion of the P wave. Upward deflections are denoted positive and downward negative.

The terminal force will consequently be given in microvolts times seconds (μV·s). A P wave duration of more than 11 cs (2), a bipeak interval of more than 4 cs and a P terminal force more negative then −3 μV·s were registered as signs of left atrial abnormality.

The R wave amplitude in leads V_{5-6}, I and aVL and the S wave amplitude in leads V_1 and III were measured. In the precordial leads, an R of more than 2.7 mV in V_5 or V_6 or a total amplitude of (SV_1+RV_5) or (SV_1+RV_6) greater than 3.5 mV was classified as high voltage (2). In the extremity leads an R wave of more than 1.3 mV in aVL or a total amplitude of (R_I+S_{III}) more than 2.6 mV was considered as high voltage (2).

The left ventricular activation time (VAT) was measured in leads V_5 and V_6 if there was no notch on the R wave before the peak, and the R wave amplitude was more than 1.5 mV. Five cs was considered to be the upper limit of normal (2). A direction of the maximum QRS vector in the frontal plane of 0° to −90° was registered as left-axis deviation. The ST segment in left precordial leads was classified according to a three-grade scale of 0–2 points. A normal ST segment scored 0 points, an ST segment with a depression of 0.05–0.10 mV scored 1 point and an ST segment with a horisontal or decremental depression of more than 0.10 mV scored 2 points.

The T wave in left precordial leads was classified in a similar way. A T wave with an amplitude of 10% or more of the preceding R wave scored 0 point, a flat or diphasic T wave with less than 0.10 mV negative phase scored 1 point, and a T wave with a negative amplitude of 0.10 mV or more scored 2 points (if R in the same load was more than 0.5 mV).

If the scored sum for the changes in ST segment and T wave was 2 or more, then the case was registered as having ST–T changes.

The investigator who made the measurements of the ECG records had no knowledge of the other findings in the health survey.

The ECG diagnosis of LVH was defined in the following way:

A. *No LVH:* no criterion fulfilled or only left axis deviation.

B. *Possible LVH:* only high voltage, or only prolonged VAT, or only ST–T changes.

C. *Probable LVH:* any combination of 2 criteria.

D. *Typical LVH:* any combination of 3 criteria or all 4 criteria.

RESULTS

The occurrence of P wave abnormalities in the three groups is presented in Table I. P wave abnormalities were more frequent in group H than in the groups M and L but they were common also in the control groups. In the H group an increased P duration was the single sign of left atrial abnormality in 22 cases. In 4 cases, this sign was combined with a prolonged peak interval, in 2 cases with an increased P terminal force and in one case all three criteria were found. A prolonged peak interval was the only abnormality in 5 cases, while in one case an increased P terminal force was the only abnormality.

In group M, an increased P duration was the single abnormality in 8 cases, while in one case it was combined with a prolonged peak interval. In 6 cases a prolonged peak interval and in one case an increased P terminal force were the single

Table II. *ECG diagnosis of LVH*
A=no sign of LVH, B–D increasing probability of LVH

Group	A	B	C	D
H	38	18	19	1
M	62	11	3	0
L	66	10	0	0

abnormalities. In group L an increased P duration was the only abnormality in 17 cases, while in one case it was combined with prolonged peak interval. One case fulfilled all three criteria. In 4 cases a prolonged peak interval and in one case an increased P terminal force were the only abnormalities.

Table I also shows the occurrence of a high QRS amplitude, prolongation of VAT, ST–T changes and left-axis deviation in the three groups. As for the P waves, positive findings were more frequent in the H group but a high QRS amplitude and left-axis deviation were also common in the control groups.

ECGs with a high QRS amplitude are divided into two groups: one with an increased amplitude as the single criterion of LVH, and the other with one or more additional criteria of LVH. In the control groups, an increased QRS amplitude was often the only abnormal finding, while in the H group more than half of the ECGs with high amplitude also showed other signs of LVH.

The ECGs with high QRS amplitude as a single sign of LVH were further divided according to abnormality or normality of the P waves. In the control groups, 6 out of 20 ECGs showed an abnormal P wave, while in the H group 8 out of 17 of the ECGs showed at least one P wave abnormality. The difference is, however, not statistically significant (χ^2).

Six cases in the H group showed ST–T changes in left precordial leads, and in three of them there were also negative T waves in lead aVL.

Table I also presents the occurrence of left-axis deviation alone or in combination with one or more criteria of LVH. In combination with other criteria of LVH left-axis deviation was far more common in the hypertensive group whereas the single abnormality of left-axis deviation was not more common in the H group than in the control groups.

In Table II the probability of LVH has been judged according to the criteria previously mentioned. In 9 out of 18 hypertensive persons in group B (possible LVH) the ECG showed at least one P wave abnormality, while the same was found in groups M and L in 6 out of 21 cases. If in the hypertensive group the number of ECGs with the diagnosis B with at least one P wave abnormality were added to the number of ECGs with diagnoses C and D, a sum of 29 out of 76 ECGs results. The corresponding figure for the M group is 5 out of 76 and for the L group 4 out of 76. Of the 20 patients in the H group with at least two signs of LVH 12 had at least one P wave abnormality. In the hypertensive group 4 of the 5 patients with increased P terminal force had at least two signs of LVH.

DISCUSSION

Electrocardiographic atrial abnormalities have been reported to occur in arterial hypertension (6, 8, 9). Thus Tarazi et al. found P wave changes in 45 of 76 patients with hypertension while only four of 76 normal subjects had such abnormalities. In our series 36 of 76 hypertensives and 41 of 152 controls had ECG atrial abnormalities. The criteria for P wave abnormality were essentially the same in the two studies. The occurrence of abnormality among hypertensives is not significantly different in the two studies, while there is a marked difference in the occurrence among control subjects. The difference mainly consists of a more frequent occurrence of prolonged P in the present study. Differing ages of the controls in the two studies might be one explanation for the difference. Variations in recording technique, such as differences in paper speed and in base line width, should also be considered.

It seems from the present study that P wave abnormality, with the current criteria, is not a reliable early sign to discriminate hypertensives from persons with a normal blood pressure.

The occurrence of a high QRS voltage was significantly ($p<0.005$, χ^2) higher in the present hypertensive group. A high voltage alone was not significantly more common in the H group than in the control groups, and therefore—with the current criteria—is not a specific sign of LVH. Variations in body build (3) may contribute to variations of QRS voltage in normal persons. High voltage in combination with other criteria of LVH can better

discriminate between hypertensives and normals. In the cases with high voltage as the single criterion of LVH examination of the P wave may be helpful.

Prolonged VAT was found in only 5 of 76 patients in the H group and always in combination with at least one other sign of LVH. It seems that prolonged VAT is a fairly insensitive but specific sign of LVH in hypertension. In a comparative ECG and autopsy study Rosenfeld et al. (5) found a prolongation of VAT beyond 5 cs in 7.4% of cases with an anatomical LVH.

In the present study, ST–T-changes were found in 6 cases in the H group and in 2 cases in group M. There are many conditions (electrolyte disturbances, increased sympathetic tone, myocarditis etc.) causing ST–T-changes. Because of this it is not possible to evaluate LVH in hypertension from this criterion alone. Sannerstedt et al. (7) have reported that a negative T wave in aVL is often the first sign of LVH in hypertension. In the present study only 3 of the 76 patients with hypertension had a negative T wave in aVL. All of these as well as 3 other patients in the H group had ST–T-changes in the precordial leads.

Left-axis deviation is more common in the H group than in the control groups. However, the occurrence of left-axis deviation as the only sign of LVH is not higher in the H group. Factors such as body build and intraventricular conduction defects can cause left-axis deviation in the absence of LVH. Left-axis deviation in combination with at least one more criterion of LVH is far more common in the H group. Thus, it seems that left axis deviation alone does not discriminate between hypertensives and controls, while left-axis deviation in combination with at least one other sign appears to do so.

LVH may be suspected from the ECG if at least two of the previously mentioned criteria of LVH are fulfilled. The ECG changes can be explained by hypertension but if the patient is normotensive at rest, other conditions (e.g. valvular disease or paroxysmal hypertension) must be considered.

The possibility that a patient may have normal blood pressure at rest and abnormal elevation of the blood pressure during mental or physical stress should be recorded. In the ECGs with a high voltage as the single sign of LVH a concomitant P wave abnormality seems to slightly increase the probability of left ventricular overload.

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