Electrocardiographic Changes Reflecting Left Atrial Abnormality in Hypertension

By Robert C. Tarazi, M.B., M.D., Anthony Miller, M.D., Edward D. Frohlich, M.D., and Harriet P. Dushman, M.D.

Increasing attention has been directed to the effect of the left atrium on ventricular function\(^1,2\) and arterial pressure.\(^3-9\) Atrial abnormalities, as a reflection of left ventricular involvement in hypertension, have not been defined, although P-wave changes have been described previously.\(^7-9\) Since these earlier descriptions, better understanding of the development of P-wave abnormalities has allowed establishment of more precise criteria for their recognition.\(^10-12\) Using these criteria, correlation of changes with the clinical course of hypertensive patients affords an opportunity to evaluate the diagnostic importance of left atrial abnormalities and their hemodynamic implications.

The frequent association of hypertension with atherosclerotic heart disease makes it difficult to distinguish their separate effects on either P-wave configuration or cardiac function. The present study was designed to evaluate P-wave abnormalities in hypertension and to separate the effects of hypertension from those of coronary atherosclerosis as judged by coronary cinearteriography. By using this radiographic technique, it was possible to demonstrate that in some patients the electrocardiographic changes of the left atrium can most likely be ascribed to hypertension alone.

Methods

The electrocardiograms of 76 normal subjects and 76 hypertensive patients, matched for age and sex, were examined. The normal subjects were selected from hospital and laboratory personnel who had complete examinations including chest x-rays and electrocardiogram. All hypertensive patients were followed from 2 to 20 years as part of a long-term study of hypertension and had been hospitalized at least once; each had had repeated clinical, laboratory, and radiographic examinations. The first 39 patients were selected at random, and the remaining 37 included all in whom selective coronary cinearteriography had been performed.

The first interpretable electrocardiograms of the first 39 patients were examined, whereas the one immediately preceding arteriography was used for the 37 patients. When available, serial tracings were examined for evidence of evolution of P-wave changes.

The criteria used for diagnosis of left atrial abnormality were: (1) terminal forces in V\(_1\) equal to or more negative than \(-0.04\) mm sec, as obtained from the product of the depth of the terminal negative deflection and its duration\(^12\); (2) biphasic peak in deeply notched P waves wider than \(0.04\) sec in any lead\(^10\); (3) P-wave/PR-segment ratio (Macruz index) greater than 1.6 in lead II\(^11\); and (4) P\(_{II}\) higher than \(0.3\) mv or wider than \(0.12\) sec.

In addition, the following indices were noted: the duration of P\(_{II}\) and peak delay in lead II when clearly defined.\(^8\) Changes in the ventricular complex were carefully analyzed by recognized criteria and followed in available serial tracings. Electrocardiograms were interpreted separately by two observers; their conclusions and measurements agreed closely, and only tracings were accepted which were considered by both to be positive for left atrial abnormality.

Correlated with electrocardiograms were: the average blood pressure, calculated from four daily readings for the week during which the tracing was obtained, and cardiac size, determined by chest roentgenograms made during the same admission, often on the day during which the electrocardiogram was obtained.

Coronary cinearteriographic studies were performed in the laboratory of Dr. F. M. Sones.

Results

Left atrial abnormality was present by one or more criteria in 45 of the 76 patients with hypertension; 28 of these had two or more positive criteria (table 1). In contrast, only four of the 76 normal subjects had abnormal...
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Table 1
Analysis of P Waves in Seventy-six Hypertensive Patients and Seventy-six Matched Normal Control Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean duration of P wave (sec)</th>
<th>No. of patients with P &gt; 0.10 sec</th>
<th>No. of patients with one or more abnormal criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>P/PR-segment &gt; 1.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Single</td>
</tr>
<tr>
<td>Normals</td>
<td>0.09</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Hypertensives</td>
<td>0.11</td>
<td>30</td>
<td>11</td>
</tr>
</tbody>
</table>

P waves by one criterion (two abnormal bipeak interval and two high P-wave to PR-segment ratio); none had more than one abnormal criterion. The mean P-wave duration (lead II) of normals was 0.09 sec and that of hypertensives was 0.11 sec (P < 0.05). The values for normal subjects are identical to those reported by Graybiel and associates,13 and Stewart and Manning.14

The most common single positive index of left atrial abnormality was an increased P-wave /PR-segment ratio; it was present alone in 11 hypertensive patients. An abnormal bipeak interval was the sole abnormality in three, and abnormal terminal forces in V1 were found alone in three. In patients showing progressive development of left atrial abnormality, the order of appearance of abnormal indices did not follow a set pattern.

Wide P waves occurred commonly; they measured 0.12 sec or more in 21 patients (28%) and 0.10 sec or more in 30 patients (39%). Only four of the 76 had P waves less than 0.08 sec in duration. In 54 patients, the delayed peak time in lead II could be measured8; it occurred later than 0.05 sec in 20; however, this added little to the evaluation of the tracing and was not considered further.

Abnormal P waves usually occurred in association with signs of left ventricular hypertrophy, nonspecific ST-segment and T-wave changes, or myocardial infarction. In 15 patients, however, the abnormal P wave was the sole electrocardiographic abnormality, occurring in some before other signs developed or, in others, remaining after other electrical abnormalities had disappeared. In seven of the 15, abnormal P waves persistently represented the sole electrical evidence of cardiac involvement.

Cardiac size was measured on the chest roentgenograms and interpreted on the basis of the patient's height and weight (Ungerleider scale). Using these criteria, 26 patients had cardiac enlargement (greater than 10% of predicted normal) and 50 had hearts of normal size. Nineteen of 45 patients (42%) with electrocardiographic evidence of left atrial abnormality had cardiac enlargement as indicated by the x-rays, but only seven of the 31 (19%) with normal P waves had enlarged hearts (table 2). First oblique views for specific left atrial enlargement had not been performed routinely.

Eleven of 45 patients with left atrial abnormality had repeated episodes of atrial arrhythmias. All of these had more than one criterion of P-wave abnormality; six had repeated paroxysmal auricular fibrillation; one, paroxysmal atrial flutter; two, premature atrial beats;

Table 2
Relationship of Cardiac Size in Roentgenograms to Left Atrial Abnormality Among Hypertensive Patients

<table>
<thead>
<tr>
<th></th>
<th>Patients with normal P waves</th>
<th>Patients with left atrial abnormality</th>
<th>Total patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac enlargement</td>
<td>7</td>
<td>19</td>
<td>26</td>
</tr>
<tr>
<td>Normal-sized hearts</td>
<td>24</td>
<td>26</td>
<td>50</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
<td>45</td>
<td>76</td>
</tr>
</tbody>
</table>

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and two, recurrent paroxysmal atrial tachycardia. By contrast, of the 31 patients with normal P waves, only one had episodes of auricular fibrillation ($P < 0.05$) (table 3).

Patients with left atrial abnormality had significantly higher systolic arterial pressures than patients with normal P waves ($P < 0.005$), but there was no significant difference in diastolic blood pressures (table 4). The duration of hypertension did not differ in the two groups.

A left atrial abnormality was present in 24 of the 37 hypertensive patients who had been studied by cinearteriography. Nineteen of these 24 had arteriographic evidence of coronary atherosclerosis, but five had no evidence of arterial disease. None of these five had any other clinical cause for the electrocardiographic P-wave changes such as mitral stenosis, congestive heart failure, and fever. Of the 13 patients with normal P waves, 11 had evidence of coronary atherosclerosis and two did not.

Discussion

Left atrial abnormality is a common electrocardiographic finding in hypertension. Since these hypertensive patients were studied over long periods of time, it was possible to recognize the fleeting character of some signs, the persistence of others, and the prognostic importance of a few.

The electrocardiographic findings compatible with left atrial abnormality are not specific for hypertension but are common to all diseases involving the left side of the heart. The term "left atrial abnormality"10 used throughout this report was chosen in preference to "left atrial hypertrophy" since the precise anatomic changes underlying these electrical signs have not been established as yet. The combination of signs derived from different spatial planes allows a more complete evaluation of atrial phenomena. The importance of this is seen in our normal controls in whom the combination of two positive criteria for left atrial abnormality has not yet been found.

P waves were wider in hypertensive patients than in normals, but voltage was not greater than normal. Many factors contribute to the discrepancy between width and voltage. Abildskov15,16 reported that widening of the P wave is a more sensitive measurement of small degrees of atrial hypertrophy than increase in voltage when routine electrocardiographic techniques are used. This emphasizes the value of criteria based on the duration of the atrial complex, such as the biphasic interval and the P-wave to PR-segment ratio.

It is not clear whether these changes reflect an interference in atrial conduction; their persistence in most cases, despite effective treatment of hypertension,8 suggests this possibility.

Contrary to earlier experience,7 left atrial abnormality in hypertension is not necessarily associated with left ventricular failure. Indeed, 17 patients with abnormal P waves had hearts of normal size and no evidence of cardiac failure. Ross8 noted that 22% of hypertensive patients without cardiac enlargement had notched P waves, and this abnormality was more frequent in precordial leads in patients with smaller hearts.8

These atrial electrocardiographic changes may indicate either active atrial participation in ventricular function or may reflect diminished ventricular compliance. Evidence

\[
\begin{array}{|c|c|c|}
\hline
\text{Atrial pressure (mm Hg)} & \text{Systolic} & \text{Diastolic} \\
\hline
\text{Normal P waves} & 168 \pm 4.1 & 111 \pm 2.5 \\
\text{Left atrial abnormality} & 186 \pm 3.7 & 106 \pm 2.6 \\
\text{P} & <0.005 & <0.10 \\
\hline
\end{array}
\]

Table 4

Average Systolic and Diastolic Arterial Pressures in Hypertensive Patients with and without Left Atrial Abnormality

\*Significant difference ($P < 0.05$) between the two groups.
supporting these possibilities is suggested by the results of this study showing a significant correlation between left atrial abnormality and average systolic pressure, by other clinical studies demonstrating that atrial activity influences systolic blood pressure, and by the frequency of occurrence of fourth (atrial) heart sounds in hypertension (50 to 70%).

In this study QRS and ST-segment changes denoting ventricular hypertrophy or nonspecific myocardial abnormalities sometimes disappeared, but abnormal P waves usually persisted, often as the sole evidence of cardiac involvement. Our experience confirms the finding of Sodi-Pallares and Calder that P-wave changes may provide the earliest clue to hypertensive heart disease and adds the observation that this sign is persistent. With correction of hypertensive heart disease, some signs of left atrial abnormality may disappear, but usually others persist. For example, one patient in our series had cardiac failure and two abnormal atrial criteria; after correction of failure, the abnormal Macruz index disappeared, but the wide bipeak interval remained.

The relation of atrial abnormality and repeated atrial arrhythmias is well known in mitral stenosis; it also appears to be true in hypertensive heart disease. The difference in incidence in hypertensive patients with and without abnormal P waves is significant (P < 0.05).

The association of hypertensive heart disease with coronary atherosclerosis makes it difficult to separate the role of either disease in the development of electrocardiographic changes. In this study, most patients with and without left atrial abnormality had abnormal coronary arteriograms. However, in five patients with left atrial abnormality, coronary arteriography revealed normal vessels. A sixth patient with both left atrial abnormality and left ventricular hypertrophy had only minimal irregularity of his dominant left coronary artery. None of these patients had any clinical evidence of myocarditis; accordingly, the electrocardiographic changes were most likely on the basis of hypertension alone. Hemodynamic studies of patients with uncomplicated hypertensive heart disease may help to clarify the mechanism of these atrial changes.

Summary

To separate the effects of hypertension and atherosclerosis on the left atrium, the electrocardiograms of 76 hypertensive patients and 76 normal control subjects, matched for age and sex, were studied. Four established criteria for left atrial abnormality were used. One or more positive criteria were satisfied in 45 of the 76 patients and 28 had two or more positive criteria. In contrast, only four of the 76 normal subjects had one abnormal criterion; none had more than one. In 37 patients selective coronary cinearteriography was performed and five of these with abnormal P waves had no arteriographic evidence of atherosclerosis. Eleven of 45 patients with left atrial abnormality had repeated episodes of atrial arrhythmias, whereas only one of the 31 patients with normal P waves had an atrial arrhythmia, demonstrating that repeated atrial arrhythmias are more frequent in patients with left atrial abnormality.

Left atrial abnormality was, therefore, a frequent finding in hypertension; it may occur even in the absence of cardiac enlargement and coronary arterial disease. It was, at times, the sole sign of cardiac abnormality in hypertension.

Acknowledgment

We wish to thank Drs. F. F. Mason Sones, Jr., Earl K. Shirey, and William C. Sheldon for performing the coronary cinearteriograms and reviewing their findings with us, and Dr. William L. Proudflit for his help and advice. We also wish to thank Misses Enid Davy, Rosemarie Horvath, and Christine Monroe for clerical and secretarial assistance.

References

On Communication with Patients

Now, in all this, while we are observing and trying to understand the patient, we must remember that he is observing and evaluating us. As John Donne remarked in his "Devotions" in 1603, some three hundred and sixty years ago, "I observe the physician with the same diligence as he the disease." Unknowing and unskilled in matters medical, the patient can judge only by the familiar hallmarks of his own experience. He notes whether his physician is like the surgeons of medieval times who washed their hands only once—after the operation was over. The patient observes whether the physician places the stethoscope over the mitral area, seems to lose himself in reflection and then reapplies and reapplies this instrument. After several such reapplications the patient surely ponders the meaning of this stammering performance by the physician with a wandering pacemaker. If the physician then emerges from his reverie and proudly announces, "everything is normal," the patient may well experience the same perplexity as the traveler visiting Hong Kong who beheld this somewhat disconcerting sign displayed by a hotel on the waterfront: "Stay with us—we overlook everything."—Herrman L. Blumgart: Caring for the Patient. New Eng J Med 270: 449, 1964.
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