The Pseudo P Pulmonale

By Te-Chuan Chou, M.D., and Robert A. Helm, M.D.

It has been generally accepted that the electrocardiographic pattern of a tall and peaked P wave with a normal duration in leads II, III, or aVF is characteristic of right atrial enlargement.1-3 Such a P wave is called the P pulmonale in contrast to a wide and notched P wave, usually best seen in leads I and II, due to left atrial enlargement, the P mitrale. During the course of interpretation of the routine electrocardiograms in a general hospital, the authors were impressed by the frequent appearance of such a P-pulmonale pattern in patients whose clinical diagnoses did not suggest the presence of right atrial enlargement.

The purpose of the present study was to determine the specificity of such a P-pulmonale pattern by clinical correlation. Vectorcardiographic analysis of the P waves was done in many of the patients in an attempt to obtain additional useful information.

Materials and Method

One hundred consecutive adult patients whose routine electrocardiograms met the following criteria of "P pulmonale" were used as the material for this study: the P wave was tall and peaked with a height of 2.5 mm. or more in leads II, III, or aVF; the duration of the P wave was 0.11 second or less. The age of the patients ranged from 20 to 59 years. The electrocardiograms were taken with a direct-writing recorder at a paper speed of 25 mm. per second and a sensitivity of 1 cm. per mv. The following measurements were made: the duration of the P wave in lead II; the amplitude of the P wave in leads II, III, and aVF; the amplitude and duration of the terminal force of the P wave in lead V1 according to the method outlined by Morris et al.4 The electrocardiographic diagnosis based on the QRS and T-wave changes was also made.

Vectorcardiograms were recorded from 143 normal adults and 20 of the 100 patients with P pulmonale. The normal individuals included medical students, house officers, and members of the local police and fire departments. In all of the 20 patients with the electrocardiographic pattern of P pulmonale, vectorcardiograms were taken either immediately before or after the recording of the corresponding electrocardiograms. The Frank system of electrode placement5 was used. Transverse, right sagittal, and frontal planes were taken in succession. (The right sagittal plane was selected in preference to the left sagittal plane for the reason given previously by one of the authors.6) A Sanborn amplifier Model 185 with an attached Frank lead selector and a Sanborn Viso-Scope Model 569A were used. The vectorcardiograms were photographed through a Dumont oscillograph-recording camera with Polaroid film. In all instances, additional spot films were taken in which the P loops were greatly magnified so that their details could be seen and analyzed. The loops were interrupted every 2.5 milliseconds producing four "tear drops" in each 0.01-second interval. The larger end of the "tear drop" represented the front end.

The criteria for the vectorcardiographic diagnosis of left and right atrial enlargement were based on those described by Massie and Walsh7 except for the magnitude of the P loop, which was modified according to our own findings in the normal individuals as recorded with the Frank system of electrode placement. Left atrial enlargement was considered to be present when the maximum P vector in any plane exceeded that of the normal and the major portion of the P loop was located posteriorly. Right atrial enlargement was considered to be present when the maximum P vector exceeded that of the normal and the major portion of the P loop was located anteriorly. These simple criteria were found to be very satisfactory when applied to patients with demonstrable atrial enlargement.8

The clinical records of the 100 patients were reviewed. The x-ray findings, the hemodynamic data, and the anatomic findings were noted. Special effort was made to determine whether there was evidence of left or right heart failure at the time when the electrocardiograms and vectorcardiograms were recorded. The electrocardiographic findings were considered to be only sup-

From the Cardiac Laboratory, Cincinnati General Hospital, and the Department of Medicine, College of Medicine, University of Cincinnati, Cincinnati, Ohio.

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The clinical diagnoses of the 100 patients were divided into the following groups:

1. Right-sided heart disease, e.g., chronic cor pulmonale, pulmonic stenosis.
2. Pulmonary disease with or without right heart involvement, e.g., pulmonary emphysema.
3. Left-sided heart disease without secondary right-sided involvement, e.g., hypertensive heart disease or aortic valvular disease with no evidence of heart failure.
4. Left-sided heart disease in which secondary right-sided involvement is possible but unlikely or insignificant, e.g., hypertensive heart disease with left-sided heart failure compensated at the time of the study.
5. Left-sided heart disease with evidence of secondary right-sided involvement, e.g., hypertensive heart disease with evidence of secondary right-sided failure.
6. Left and right-sided heart disease due to the same or an unrelated etiology.
7. Coronary artery disease in which the presence of cardiac enlargement was not demonstrated.
8. Questionable heart disease.
9. No demonstrable heart disease.

**Result**

**Correlation of the Electrocardiographic and Clinical Findings**

The number of patients with the electrocardiographic pattern of P pulmonale in each of the above-mentioned groups is listed in table 1. Thirty-nine patients had either pulmonary or right-sided heart disease (groups 1 and 2). They represented the patients in whom right atrial enlargement might be expected. Thirty-three patients had left-sided heart disease and represented the patients in whom left atrial enlargement might be present (groups 3 and 4). Twenty of them had no evidence of heart failure (group 3). Eight had a history of left ventricular failure but were compensated at the time of the study; two had questionable early left ventricular failure; and three had definite left heart failure (group 4). None of them had a history or any sign of right ventricular failure. Thirteen patients had biventricular involvement and represented patients in whom both atria might be enlarged (groups 5 and 6). In 10 patients there was no reason for the enlargement of either the right or left atrium (groups 7 and 9).

Since groups 3 and 4 patients (patients with left-sided heart disease) presented special interest, the incidence of the various etiologies encountered are summarized in table 2. Twenty-five of the 33 patients in the combined group had hypertension or hypertensive heart disease. Five patients had aortic valvular disease. A rheumatic mitral lesion was responsible in only one patient.

**Table 1**

**Analysis of the Clinical Diagnosis in 100 Consecutive Patients with the Electrocardiographic Pattern of P Pulmonale**

<table>
<thead>
<tr>
<th>Group</th>
<th>Clinical diagnosis</th>
<th>No. cases and percentage of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right-sided heart disease</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>Pulmonary disease</td>
<td>24</td>
</tr>
<tr>
<td>(1 &amp; 2)</td>
<td></td>
<td>(39)</td>
</tr>
<tr>
<td>3</td>
<td>Left-sided heart disease</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>Left-sided heart disease with possible minor secondary right-sided involvement</td>
<td>13</td>
</tr>
<tr>
<td>(3 &amp; 4)</td>
<td></td>
<td>(33)</td>
</tr>
<tr>
<td>5</td>
<td>Left-sided heart disease with secondary right-sided involvement</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>Bilateral involvement due to the same or an unrelated etiology</td>
<td>7</td>
</tr>
<tr>
<td>7</td>
<td>Coronary artery disease without cardiomegaly</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>Questionable heart disease</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>No demonstrable heart disease</td>
<td>7</td>
</tr>
</tbody>
</table>

100
Table 2

Incidence of the Various Diseases Affecting the Left Heart That Were Associated with the P-Pulmonale Pattern

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Aortic insufficiency, syphilitic</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Rheumatic heart disease with mitral lesion</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Arteriosclerotic heart disease with cardiomegaly</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>20</strong></td>
<td><strong>13</strong></td>
</tr>
</tbody>
</table>

Figure 1 demonstrates the presence of a P-pulmonale pattern in the electrocardiograms of patients who had evidence of left-sided heart disease only. All four patients had hypertensive cardiovascular disease. Two had no symptoms or signs of congestive heart failure. The other two had early or questionable left ventricular failure.

Table 3 lists the number and percentage of patients in groups 1, 2, 3, 4; groups 1 and 2 combined; and groups 3 and 4 combined who had abnormal terminal P force in lead V1; and QRS and T-wave changes in support of right or left atrial enlargement by inference. The terminal P force in V1 was abnormal and suggestive of left atrial involvement in 18 per cent of patients of groups 1 and 2 combined, and in 42 per cent of groups 3 and 4 combined. The QRS and T-wave changes that met the criteria for the diagnosis of right ventricular hypertrophy9 or were consistent with pulmonary emphysema10 were considered to support the presence of right atrial enlargement. They were found in 64 per cent of the combined groups 1 and 2 patients but in only 3 per cent of the combined groups 3 and 4 patients. The electrocardiographic diagnosis of left ventricular hypertrophy9 was considered to be supportive of left atrial enlargement and was found in only 8 per cent of the combined groups 1 and 2 patients but in 67 per cent of the combined groups 3 and 4 patients.
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Figure 1

Four examples demonstrating the presence of a P-pulmonale pattern in patients with left-sided heart disease. A and B, patients with hypertensive heart disease without heart failure. C and D, patients with hypertensive heart disease with early or questionable left ventricular failure.

Correlation of the Vectorcardiographic and Clinical Findings

The means and the 97.5 and 2.5 percentile values of the magnitudes of the maximum P vector in the transverse, right sagittal, and frontal planes obtained from the vectorcardiograms (Frank system) of the 143 normal adults are listed in table 4. Other measures of the normal P loop will be reported elsewhere. The 97.5 percentile values were considered as the upper limits of normal. According to the criteria mentioned previously, among the 20 patients with the P-pulmonale pattern in their scalar electrocardiograms, there were nine patients with vectorcardiographic signs of right atrial enlargement, nine of left atrial enlargement, one with bialtrial enlargement, and one in whom the diagnosis was uncertain. When the vectorcardiographic results were correlated with the clinical diagnosis, a high degree of positive correlation existed (table 5). Eight of the nine
patients with the vectorcardiographic signs of right atrial enlargement had pulmonary or right-sided heart disease (groups 1, 2, and 6) and all nine patients with left atrial enlargement had left-sided heart disease (groups 3, 4, and 6).

The electrocardiogram and vectorcardiogram of a patient with valvular pulmonic stenosis are shown in figure 2. Both graphic findings agreed with each other and were consistent with the clinical diagnosis. Figure 3 demonstrates an example in which the electrocardiogram revealed a P-pulmonale pattern. The vectorcardiogram showed that the maximum P vector exceeded the upper limits of normal in all three planes and the majority of the P loop was located posteriorly (as viewed in the transverse and right sagittal planes). The vectorcardiographic diagnosis was left atrial enlargement. The patient was a 53-year-old man with aortic insufficiency. Physical examination and x-ray revealed left ventricular hypertrophy. There was no history or sign of congestive heart failure. No evidence of pulmonary or right heart disease could be elicited.

**Discussion**

Winternitz first described a characteristic pattern of the P wave in the limb leads consisting of a small upright P wave in lead I and tall, upright peaked P waves in leads II and III, all of normal duration; this combination was designated as P pulmonale and the changes were attributed to strain on the right atrium. Such a pattern of P pulmonale has been observed at various frequency in patients with pulmonary emphysema, chronic cor pulmonale (up to 85 per cent), and congenital or acquired diseases of the pulmonic and tricuspid valves. Transient appearance of the P-pulmonale pattern has been observed in pulmonary embolism, bronchial asthma, especially status asthmaticus, increased cardiac output in exercise, arterial venous aneurysm, diabetic acidosis, acute heart failure, and tachycardia. The changes in the P-wave morphology have been attributed to the enlargement (dilatation or hypertrophy, or both) of the right atrium and the pattern has commonly been considered to be diagnostic of such. However, increased atrial pressure, vertical position of the heart, and hypoxia have all been suggested as the contributing factors.

In 1942, Shleser and Langendorff studied 50 consecutive patients with the P-pulmonale pattern and found 40 cases with evidence of chronic pulmonary disease and one case each of congenital heart disease and acute

**Table 4**

<table>
<thead>
<tr>
<th>Planes</th>
<th>Maximal P vector in millivolt Mean</th>
<th>97.5 percentile</th>
<th>2.5 percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transverse</td>
<td>0.07</td>
<td>0.10</td>
<td>0.03</td>
</tr>
<tr>
<td>Right sagittal</td>
<td>0.11</td>
<td>0.18</td>
<td>0.04</td>
</tr>
<tr>
<td>Frontal</td>
<td>0.12</td>
<td>0.22</td>
<td>0.05</td>
</tr>
</tbody>
</table>

**Table 5**

<table>
<thead>
<tr>
<th>Clinical diagnoses</th>
<th>Normal</th>
<th>RAE</th>
<th>LAE</th>
<th>BAE</th>
<th>Undertermined</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Group 2</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>(1 &amp; 2)</td>
<td>(0)</td>
<td>(7)</td>
<td>(0)</td>
<td>(1)</td>
<td>(1)</td>
<td>(9)</td>
</tr>
<tr>
<td>Group 3</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Group 4</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>(3 &amp; 4)</td>
<td>(0)</td>
<td>(1)</td>
<td>(7)</td>
<td>(0)</td>
<td>(0)</td>
<td>(8)</td>
</tr>
<tr>
<td>Group 6</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>0</td>
<td>9</td>
<td>9</td>
<td>1</td>
<td>1</td>
<td>20</td>
</tr>
</tbody>
</table>

RAE, Right atrial enlargement; LAE, Left atrial enlargement; BAE, Bialtrial enlargement.
The pseudopulmonale. Sano et al.\textsuperscript{16} however, described a type of left atrial hypertrophy characterized by a tall P wave, especially in leads II and III, and which resembled pulmonale P wave although it tended to be less peaked. Abildskov\textsuperscript{15} also observed tall P waves in a number of patients with mitral valvular lesions in whom left atrial enlargement might be expected. Our observation of the frequent appearance of the P-pulmonale pattern in patients in whom right atrial abnormality was not expected prompted us to reexamine systematically the specificity of the pattern.

Our method of correlating the P-wave changes with the disease entities instead of the actual size of the atrium has its obvious defect. However, by including all patients whose diseases may predispose them to abnormality of the particular atrium, the possibility of false-negative correlation can be reduced to a minimum. The method would also take into consideration the cases in which the P-wave changes were due to mechanisms other than enlargement as previously mentioned.

In contrast to the results reported by Shleser and Langendorf,\textsuperscript{14} in the 100 consecutive patients whose electrocardiograms showed a P-pulmonale pattern, there were only 49 patients in whom right atrial enlargement (groups 1, 2, 5, and 6) may be expected. They included all patients with pulmonary and right-sided heart diseases (the right heart involvement may be either primary or secondary to the left-sided disease). In fact, it can be reasonably assumed that the actual number of the cases in whom the right atrium was involved would be smaller for the obvious reason.
The electrocardiogram and vectorcardiogram of a 53-year-old man with aortic insufficiency. The electrocardiogram revealed a P-pulmonale pattern. The P loop in the vectorcardiogram was abnormally large and displaced posteriorly suggesting left atrial enlargement.

Among the 43 patients whose clinical findings failed to support the presence of a right atrial enlargement (or abnormality), 33 of them did have diseases that might cause enlargement of the left instead of the right atrium (groups 3 and 4 patients). Coexisting right atrial involvement might be present in three of the group-4 patients because of left ventricular failure. However, this was unlikely in the absence of right heart failure, and the P-wave changes seen in acute left ventricular failure have been attributed to acute left atrial dilatation.\(^{18}\)

Recent literature\(^4,19\) has emphasized the inadequacy of the diagnostic criteria for atrial enlargement based on the findings in the limb leads alone. Soloff and Zatuchni\(^19\) observed an increase in the negative component of the P wave in the right precordial leads associated with increasing size of the left atrium demonstrated angiographically. Arevalo and associates\(^20\) studied 88 young patients with rheumatic heart disease and found that 29 of the 35 patients with roentgenographic signs of left atrial enlargement had a terminal negative portion of the P wave in lead V\(_1\) of greater than 1 mm. in depth or 0.05 second in duration. Morris et al.,\(^4\) using the criteria they described (see footnote to table 3), were able to separate correctly normal subjects from those patients with mitral or aortic valvular lesions in 92 per cent of the cases. The application of their criteria improved the clinical correlation in our series, as 42 per cent of the patients in whom left atrial enlargement could be expected clinically (groups 3 and 4) had abnormal terminal force in V\(_1\) in contrast to the 18 per cent in patients who had pulmonary or right-sided heart disease.

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The possibility that the P-pulmonale pattern of patients in groups 3 and 4 may be due to left atrial enlargement is further suggested by the fact that in 67 per cent of the patients the pattern was associated with QRS and T-wave changes suggestive of left ventricular hypertrophy and only 3 per cent of the patients revealed QRS and T changes consistent with pulmonary or right ventricular diseases. The reverse was true in patients of groups 1 and 2 (8 and 64 per cent). Since the electrocardiographic findings were not used as the decisive data in arriving at the clinical diagnoses, the findings can be considered as significant.

Although the number of patients in our series in whom the vectorcardiograms were recorded was comparatively small, the high degree of positive vectorcardiographic clinical correlation renders the findings meaningful. Among the 20 patients, the vectorcardiographic diagnosis of right or left atrial enlargement were consistent with the clinical findings in nearly 90 per cent (17 of 20) of the cases. All the patients (9) with clinically left-sided heart disease have vectorcardiographic signs of left atrial enlargement. The results indicate again that in the group of patients with left-sided heart disease, the P-pulmonale pattern was probably a manifestation of left atrial enlargement.

The different configurations of the P wave in P pulmonale and P mitrale have generally been explained in the light of the sequence of atrial excitation. Atrial depolarization spreads radially from the sinoatrial pacemaker and activates first the right and then the left atrium. The first portion of the P wave has been attributed to the excitation of the right atrium; the mid portion, both atria; and the last portion, the left atrium alone. An increase in the right atrial potential affects the early and mid portions of the P wave, resulting in a tall and peaked P wave (leads II, III, or aVF), the P pulmonale. An increase in the left atrial potential, especially when there is associated intraatrial conduction defect, affects the mid and late portions of the P wave and results in a notched P wave with increased duration, the P mitrale. However, it is conceivable that enlargement of the left atrium, in the absence of a marked delay in the intraatrial conduction, could result in an increase in the voltage of the mid and late portions of the P wave without undue prolongation of its duration. The P wave would then appear to be tall but have a normal duration and resemble the P-pulmonale pattern (fig. 4). Most of our cases of probable left atrial enlargement masquerading as "P pulmonale" were patients with hypertensive heart disease (table 2). Only one patient had a rheumatic mitral lesion. As the lesion in the atrial wall in the patients with rheumatic mitral disease has been held responsible for the conduction disturbance with prolongation and notching of the P wave, the absence of such a lesion in the patients with hypertensive heart disease may explain the difference in the P-wave configuration.

Since the vectorcardiographic differentiation of right and left atrial enlargements depends on the anterior or posterior displacement of the P loop, the question may arise as to why such changes are not always accurately reflected in the scalar precordial electrocardiogram. This may be explained on the basis of the anatomic proximity of the right atrium to the recording unipolar electrode. The electric potential associated with right atrial activation would exert relatively greater influence than the comparatively remote, posteriorly located left atrium. Such preferential treatment can be avoided by the use of an ideal vectorcardiographic lead system in which the lead would be influenced equally by the electrical events occurring in all portions of the heart. Although the Frank lead system cannot be considered as the ideal vectorcardiographic lead system, the improvement in the registration of the relative potentials of the two atria offered by the system may explain the very satisfactory results obtained in our limited data.

Summary and Conclusion

One hundred consecutive patients whose electrocardiograms presented a pattern of P pulmonale were studied to determine the specificity of the electrocardiographic pattern.
Forty-nine patients had diseases in which right atrial enlargement might be expected. Forty-six patients failed to reveal any clinical condition that would predispose them to right atrial abnormality.

Vectorcardiograms were recorded in 20 of the patients. The vectorcardiographic diagnoses of atrial enlargement agreed with the clinical findings in nearly 90 per cent of the cases.

In 36 patients, the P-pulmonale pattern probably represented left, instead of right, atrial enlargement. The assumption was supported by the clinical findings, analysis of the P wave in lead V1, associated QRS and T-wave changes, and vectorcardiographic findings.

The possible mechanism for the appearance of the P-pulmonale pattern due to left atrial enlargement and the reason for the improved accuracy of the vectorcardiographic diagnosis were offered.

It is concluded that the electrocardiographic pattern of P pulmonale is not so specific as has been generally believed. Vectorcardiography may be a valuable additional tool in the diagnosis of atrial abnormality.

References

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The Importance of Tradition

Even Pasteur, who enjoyed as much acclaim in his lifetime as any innovator who ever lived, was the object— all his life— of bitter attack and opposition. And he understood that it was inevitable. “One day, when I was a candidate for a vacant seat at the Academy of Sciences . . . one of the oldest and most dignified members said to me . . . ‘My friend, if they stop speaking disparagingly of you in certain journals, tell yourself that you are slipping.’ “

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