A Method for the Electrocardiographic Recognition of Atrial Enlargement

By Radi Macruz, M.D., Joseph K. Perloff, M.D., and Robert B. Case, M.D.

New criteria for the electrocardiographic recognition of right atrial enlargement, left atrial enlargement, and combined atrial enlargement were studied in 110 patients with congenital and acquired heart disease. Precise physiologic data were available in all cases. The method of analysis was designed for application to conventional scalar electrocardiography and hence can be used without modification for routine clinical purposes.

THE current criteria for the electrocardiographic diagnosis of atrial enlargement are seldom adequate for the recognition of even moderate increases in chamber size that may be detectable radiologically. Analysis of tracings has principally considered the configuration, amplitude, and duration of P waves. Although prolongation of P-R interval has also been observed in severe pulmonic stenosis, atrial septal defect, Ebstein's disease, and atroventricularis communis, attention thus far has not specifically been directed toward the P-R segment (time between end of electric atrial systole and onset of electric ventricular systole). This communication proposes a new basis for the diagnosis of atrial enlargement based upon the relation between the durations of P wave, P-R segment, and P-R interval.

MATERIALS AND METHODS

The electrocardiograms of 62 normal adults and 110 patients with congenital or acquired heart disease were analyzed.

This case material is summarized in table 1. The patients were divided into 2 groups—those with diseases in which an increase in left atrial size might be expected and those with diseases in which an increase in right atrial size might be expected. These will subsequently be designated "left atrial group" and "right atrial group."

The width of the P wave and the durations of P-R interval and P-R segment were each measured in lead II according to the following criteria. When the P-R segment was flat, it was measured from the end of the P wave to the onset of the QRS complex. Occasionally the P-R segment was found to slope downwards to the QRS complex, and in this case the onset of the P-R segment was considered to be the point where a line extended from the T-P segment intersected the descending limb of the P wave. The P-R interval was measured from the onset of the P wave to the onset of the QRS complex. Width of the P wave was measured from the onset of the P wave to the onset of the P-R segment. In conventional single channel scalar electrocardiography, the most accurate P-R interval is the sum of the maximum P wave duration (in any lead) plus the minimum P-R segment duration (in any lead). This largely obviates the need for simultaneous leads. When this method was applied to our tracings, it was determined that lead II reflected the correct P-R interval with sufficient frequency to warrant its selection for the type of analyses described in this study.

The evaluation of all patients included fluoroscopy, x-ray, electrocardiogram, and right heart catheterization. In 7 patients with mitral valve disease right heart catheterization was omitted. The left heart was catheterized by the bronchoscopic technic in these 7 patients and in other cases when indicated. Angiocardiography and retrograde aortography were performed in selected cases.

None of the normal and only 5 of the congenital group were receiving digitalis when the tracings were taken. Two of the 6 patients with primary pulmonary hypertension and 65 per cent of those with mitral valve disease were on maintenance doses of digitalis at the time that their tracings were recorded. No tracings were taken during administration of quinidine. Heart rates varied from 60 to 150 beats per minute, a range that was considered to exert negligible rate effect on the P-R intervals. The same patients were occasionally analyzed at materially different rates.

*The left heart catheterizations were done by Dr. Andrew Glenn Morrow, Chief, Surgery, National Heart Institute, National Institutes of Health.

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with no significant variation in P-R interval or P/PR segment ratio.

RESULTS

In the series of normal adults, the P-R interval varied from 0.12 to 0.20 second. It should be noted that there was only 1 case below 0.14 second. The P wave varied from 0.06 to 0.11, with only 1 case above 0.10. The mean values were 0.09 second for the P wave, 0.16 second for the P-R interval, and 0.07 second for the P-R segment. The ratio of the duration of the P wave to the duration of the P-R segment varied from 1.0 to 1.6, with a mean value of 1.2.

The duration of the P-R interval in patients over 16 years of age was compared in the normal, right atrial, left atrial, and uncomplicated left atrial groups (table 2). This last group consisted of left atrial cases in which pulmonary arteriolar resistance was normal and hence in which right atrial enlargement might not be expected. The 22 uncomplicated left atrial cases contained 17 with mitral valve disease, 2 with aortic stenosis, and 1 with ventricular septal defect.

The mean ages of all groups were approximately the same. The mean P-R interval in the right atrial group was 0.20 second, a statistically significant increase from the normal of 0.16 second. Duration of the P-R interval in the left atrial group was identical to that in the normal. However, in the uncomplicated left atrial group it was 0.15 second, a statistically significant decrease from the normal.

The average duration of the P wave in adults was found to be 0.09 second in the right atrial group and 0.11 second in the left atrial group. The ratio of P wave to P-R segment was determined in all cases.

As stated before, the mean value of the P/PR segment ratio in our group of normal adults was found to be 1.2, with a range of 1.0 to 1.6. From Ziegler’s data for children the P/PR segment ratio was calculated by us, and found to be a mean of 1.2 from birth to 16 years. It is interesting to note the constancy of the P/PR segment ratio in spite of

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of cases</th>
<th>Mean age (years)</th>
<th>Age range (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>16</td>
<td>20 ± 10</td>
<td>3-49</td>
</tr>
<tr>
<td>Atrial septal defect with anomalous venous drainage</td>
<td>3</td>
<td>27 ± 11</td>
<td>6-34</td>
</tr>
<tr>
<td>Atrial septal defect with pulmonic stenosis</td>
<td>4</td>
<td>7 ± 3</td>
<td>1-11</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
<td>9</td>
<td>11 ± 5</td>
<td>6-30</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
<td>6</td>
<td>31 ± 9</td>
<td>14-</td>
</tr>
<tr>
<td>Ebstein’s disease</td>
<td>1</td>
<td>—</td>
<td>18</td>
</tr>
<tr>
<td>Ruptured aneurysm of sinus of vasa salva into right atrium</td>
<td>1</td>
<td>—</td>
<td>26</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>7</td>
<td>6 ± 3</td>
<td>2-15</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrial cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>10</td>
<td>12 ± 6</td>
<td>2-23</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>9</td>
<td>19 ± 14</td>
<td>1-40</td>
</tr>
<tr>
<td>Coarctation of aorta</td>
<td>3</td>
<td>20</td>
<td>2-29</td>
</tr>
<tr>
<td>Acquired aortic stenosis</td>
<td>2</td>
<td>26</td>
<td>21-31</td>
</tr>
<tr>
<td>Congenital aortic stenosis</td>
<td>3</td>
<td>9 ± 2</td>
<td>7-31</td>
</tr>
<tr>
<td>Eisenmenger’s complex*</td>
<td>1</td>
<td>—</td>
<td>31</td>
</tr>
<tr>
<td>Congenital mitral incompetence</td>
<td>1</td>
<td>—</td>
<td>5</td>
</tr>
<tr>
<td>Rheumatic mitral incompetence</td>
<td>13</td>
<td>39 ± 8</td>
<td>21-59</td>
</tr>
<tr>
<td>Rheumatic mitral stenosis</td>
<td>18</td>
<td>37 ± 6</td>
<td>17-49</td>
</tr>
<tr>
<td>Aortic incompetence with mitral valve disease</td>
<td>3</td>
<td>44 ± 7</td>
<td>36-55</td>
</tr>
<tr>
<td>Total</td>
<td>63</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>62</td>
<td>33 ± 8</td>
<td>18-59</td>
</tr>
</tbody>
</table>

*This case can be considered in either left atrial or right atrial groups.

the wide changes in P-wave duration and P-R interval that occur with age.

Figure 1 illustrates the separation of left atrial and right atrial cases in relation to the normal P/PR segment ratio of 1.0 to 1.6. There is striking separation of the right atrial cases below the normal mean and of the left atrial cases above the normal mean. However, 22 of the cases in the right atrial group and 17 of the cases in the left atrial group fell within the normal range, 1 of the latter having a ratio of less than 1.0. The right atrial section in figure 1 is separated into the individual disease states, from 1 through 6 as
Table 2.—Comparisons of P-R Intervals in All Four Groups

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of cases</th>
<th>Age (mean)</th>
<th>P-R interval (mean)</th>
<th>Value of ( p ) in relation to normal</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>62</td>
<td>33±8</td>
<td>0.161±0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total right atrial cases</td>
<td>19</td>
<td>29±7</td>
<td>0.195±0.03</td>
<td></td>
<td>Significantly longer than normal</td>
</tr>
<tr>
<td>Total left atrial cases</td>
<td>47</td>
<td>36±9</td>
<td>0.162±0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncomplicated left atrial cases</td>
<td>22</td>
<td>33±10</td>
<td>0.149±0.02</td>
<td></td>
<td>Significantly shorter than normal</td>
</tr>
</tbody>
</table>

Discussion

Explanation of the changes in P duration, P-R interval, P-R segment, and P/PR segment ratio that occur with atrial enlargement might be approached in the following way. Atrial activation is considered to follow the pattern schematically illustrated in figure 2. The depolarization originates in the sinoatrial node (SA) and proceeds through the atrial muscle in concentric waves. After an interval it arrives at the atrial septum and initiates left atrial depolarization (point 1), followed in temporal sequence by arrival at the AV node (point 2), by completion of right atrial depolarization (point 3), and finally by completion of left atrial depolarization (point 4). The linear velocity of this impulse is about 1,000 mm. per second.\(^8\)-\(^9\) If this velocity remained relatively constant,\(^*\) right atrial enlargement would then increase the transit time from SA node to AV node and prolong both P-R interval and P-R segment (fig. 3) (fig. 4, nos. 5-10). However, the duration of the P wave would be unaffected, since the terminal part of the P wave is normally written by left atrial depolarization. Thus, right atrial enlargement would prolong the P-R interval and alter the configuration of the P wave but would not affect P-wave duration except for those instances in which transit time in the right atrium was exceptionally prolonged by the magnitude of chamber size. The terminal portion of the P wave would then be written by the right atrium, resulting in P-wave prolongation. That this possibility may occur is supported by the observation of a

\(^*\)If velocity were impeded by an intra-atrial conduction defect, then P-R interval or P duration or both might be prolonged, depending upon the respective involvement of right or left atrial conduction.
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Fig. 3. Diagram of transmission of impulse from S-A node in normal, right atrial enlargement, and left atrial enlargement.

decrease in P-wave duration following valvotomy for pulmonic stenosis.10

Left atrial enlargement, on the other hand, should exert no influence on the transmission time from the SA to the AV node, and hence should be associated with a normal P-R interval (fig. 3). However, the P-wave duration might be prolonged by the increased transit time through the enlarged left atrium because, as noted above, the left atrium normally writes the terminal inscription of the P wave. The net result should be prolongation of the P wave and shortening of the P-R segment but no change in P-R interval. Indeed, the P-R segment may virtually disappear (fig. 4, nos. 1-4). Gross evidence of the validity of these observations is seen in the P-wave pattern of "P pulmonale"—with its high amplitude, normal duration, and prolonged P-R interval and P-R segment; and in the P-wave pattern of "P mitrale"—with its delayed secondary peak (bifid), prolonged duration, normal P-R interval, and shortened P-R segment.

If both atria are hypertrophied, it might be expected that the coexistence of the prolonged P-R segment of right atrial enlargement and the prolonged P duration of left atrial enlargement would counterbalance each other resulting in a normal P/PR segment ratio. Hence when the P/PR segment ratio is normal, the finding of distinct prolongation of P-R interval and P duration identifies the presence of combined atrial enlargement. This is consistent with the observation that as both right and left atrial masses increase from infancy to adult life there is an increase in P-R interval, P duration, and P-R segment. As noted, if depolarization in an enlarged right atrium is unusually prolonged because of a markedly increased chamber size, the terminal portion of the P wave may be written by the right rather than the left atrium. In this study right atrial enlargement was found to prolong the P duration to a maximum of 0.12 second in adults and 0.10 second in children (Ziegler's tables were referred to for normal P duration below the age of 16 years). Fur-
ther prolongation of P duration was seen only when the left atrium was enlarged. It should be emphasized, therefore, that the 22 cases in the right atrial group and the 17 cases in the left atrial group that had normal P/PR segment ratios must be analyzed further according to the foregoing criteria for combined enlargement or right atrial enlargement with prolonged P duration.

Figure 1 illustrates the specific distributions of P/PR segment ratio in all categories. The cases with ratios of less than 1.0 are in the group with right atrial enlargement and those with ratios above 1.6 are in the group with left atrial enlargement. The cases in the range of 1.0 to 1.6 include normal adult controls, cases of combined atrial enlargement, and cases of right atrial enlargement with prolonged P duration.

Among the normal adult controls (fig. 1) it was found that although P duration and P-R interval varied with age, the P/PR segment ratio remained constant irrespective of age.

None of the cases of tetralogy of Fallot (category 1) fell into the normal range. Indeed, this category had the smallest P/PR segment ratio in the entire study. This appeared to be a consequence of unusually short P durations that averaged 0.06 second compared to a mean P duration of 0.08 second for all other right atrial cases in the same age group.

In category 2 (primary pulmonary hypertension), 2 cases had normal ratios. One of these had a pulmonary artery pressure of 70/30, the lowest in this category. Right atrial enlargement was not evident by any electrocardiographic criteria. The other patient was a 14-year-old child with a pulmonary artery pressure of 180/118, P-wave amplitude upper limits of normal, a P-R interval of 0.18, and a P duration of 0.10. For this age group the mean normal P-R interval is 0.15 and the mean normal P duration is 0.08. This patient, therefore, would be suspected by the above criteria to have right atrial enlargement not otherwise evident.

In category 3 (pulmonic stenosis), 6 cases were in the normal range. One represented right atrial enlargement with prolonged P duration. Four were normal by all electrocardiographic criteria; 2 of these were mild, 1 was severe. The sixth case, one of severe stenosis, was normal by our criteria but had a P amplitude increased to 3.6 mm.

In category 4 (pulmonic stenosis with atrial septal defect) all 3 cases in the normal range represented right atrial enlargement with prolonged P duration.

In category 5 (atrial septal defect), 9 cases had normal P/PR segment ratios. Two represented right atrial enlargement with prolonged P duration, 3 had P durations that were the upper limits of normal, and 4 were normal by all criteria.

In category 6 (atrial septal defect with anomalous venous drainage, Ebstein's disease, congenital aneurysm of the sinus of Valsalva with rupture into the right atrium), the 2 cases in the normal range were atrial septal defects with anomalous venous drainage, one representing right atrial enlargement with prolonged P duration, the other (with a small shunt) normal by all criteria.

In category 7 (congenital cases with left atrial preponderance), 10 were within the normal range. One with Eisenmenger's syndrome (pulmonary artery pressure of 160/32) and another with a reversed shunt patent ductus arteriosus (pulmonary artery pressure 132/64) had no evidence of atrial enlargement by any electrocardiographic criteria. These remain unexplained. The single patient with coarctation of the aorta that fell into the normal range had a very slight brachial-femoral gradient, normal data on right heart catheterization, and therefore no evidence physiologic basis for left atrial enlargement. The remaining 7 with normal ratios had ventricular septal defects. One of these had no electrocardiographic evidence of atrial enlargement in spite of a bidirectional shunt (predominant left-to-right), 2 were very mild, a fourth had a P duration the upper limits of normal, and the remaining 3 fulfilled the criteria for combined enlargement. The single case that fell into the P/PR seg-
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A subsequent range of right atrial enlargement had a patent ductus arteriosus with a pulmonary arterial pressure of 106/64 and reversal of the shunt.

In category 8, (acquired heart disease with the left atrial preponderance), 7 patients had normal P/PR segment ratios. One with severe aortic stenosis (gradient of 153 mm. Hg) and a left atrial pressure of 22/11 mm. Hg had no evidence of atrial enlargement by any electrocardiographic criteria. Of the 2 with severe mitral stenosis, one had a pulmonary artery pressure of 60/32, a P-R interval of 0.18 second, a P duration of 0.11 second, and no electrocardiographic criteria of atrial enlargement. The other had a P-R interval of 0.22 second and a P duration of 0.12 second, evidence of combined enlargement. The remaining 4 with normal P/PR segment ratios had mitral incompetence, 3 of which were mild. The fourth had a pulmonary artery pressure of 69/16, electrocardiographic evidence of right ventricular hypertrophy, a P-R interval of 0.20 second, and a P duration of 0.12 second. The increased P duration and upper normal P-R interval suggest combined enlargement.

In this same category, the adult cases with mitral valve disease or left-to-right shunts other than atrial septal defects, but with normal or slightly elevated pulmonary arteriolar resistances had P-R intervals significantly shorter than the comparable normal adult cases (table 2).

It appeared from the analysis of the cases with right atrial preponderance that the P-R interval prolongation occasionally was greater than could be accounted for by increased transit time through the enlarged right atrium. The following evidence suggests that right atrial hypertension may sometimes prolong the P-R interval and P-R segment by a selective effect on A-V conduction and hence explain this additional prolongation of P-R interval in these cases. A patient with a congenital sinus of Valsalva aneurysm that had ruptured into the right atrium had both enlargement and hypertension of the right atrial chamber. Before rupture the P-R interval was 0.16 second, the P-R segment 0.07 second (fig. 4, no. 8a). Immediately prior to operation (5 months after the first electrocardiogram), the P-R interval was 0.24 second, the P-R segment 0.15 second (fig. 4, no. 8b). Within 20 days after surgical closure of the rupture with a polyvinyl prosthesis, the P-R interval fell to 0.20 second and the P-R segment to 0.11 second (fig. 4, no. 8c). Since the size of the right atrial wall could hardly have been altered significantly in so brief a period, it might be inferred that the shortening of the P-R interval correlated instead with a fall in atrial pressure. This shortening could be due either to more rapid conduction through the atrial muscle or to more rapid transit through the AV nodal tissue.

The effect that left atrial hypertension has on P-R interval and P-wave duration was further considered in the following way. In mitral stenosis the left atrial pressure is typically elevated. It was noted that the P-wave duration was unaffected by mitral valvulotomy. This suggests that in the left atrium at least, the duration of electric systole is un influenced by the relief of atrial hypertension itself. Nor is there any immediate change after valvulotomy in P-R interval or P-R segment. Hence, relief of left atrial hypertension appears to leave unchanged the velocity of conduction through its wall or through AV nodal tissue, whereas right atrial hypertension apparently may delay conduction through AV nodal tissue and hence prolong the AV to QRS interval.

Conclusions

The accepted range of normal for P-R interval is so broad that many patients with atrial enlargement fall within the normal range. Nor can one materially increase the accuracy of electrocardiographic diagnosis of atrial enlargement by analysis of the P-wave duration alone. However, when one observes the relative amount of the P-R interval occupied by the P wave—which can be expressed as the ratio \[
\frac{\text{P duration}}{\text{P-R segment}}
\] —the identification
of atrial enlargement becomes more precise. Normally the P duration is 50 to 60 per cent of the P-R interval at heart rates up to age 16 years according to Ziegler and above 16 years according to our data. That is, the ratio of \( \frac{P}{P-R \text{ segment}} \) varies within the narrow limits of 1.0 to 1.6.

In right atrial enlargement the P-R segment increases because of an increased transit time from the SA to the AV node. The P-wave duration remains constant, hence the ratio of P/PR segment falls below the normal range. When the right atrium is sufficiently enlarged so that the transit time of the depolarization impulse through that chamber is longer than the transit time of the depolarization impulse through left atrium, then the right atrium may write the terminal portion of the P wave. In this fashion, right atrial enlargement may also prolong the P duration as well as the P-R interval and hence increase both numerator and denominator of the fraction P/PR segment so that the resulting ratio may be normal. Right atrial enlargement was found to prolong the P duration to a maximum of 0.10 second in children and 0.12 second in adults. Hence, if the P/PR segment ratio is normal and the P-R interval prolonged, the associated finding of a P duration which though prolonged, does not exceed 0.10 second in the age group below 16 years and 0.12 second in the age group above 16 years, suggests the presence of right atrial enlargement.

In left atrial enlargement the terminal portion of the P wave is delayed because of the prolonged transit time of the depolarization impulse through the enlarged left atrial wall. Hence the P-wave duration is prolonged, the P-R segment is shortened, and P-R interval remains unchanged. The result is a P/PR segment ratio above the normal limit of 1.6.

In combined atrial enlargement the right atrium continues to prolong the P-R interval and the left atrium continues to prolong the P wave. Since these respective prolongations would increase both numerator and denominator of the fraction P/PR, it can be seen that in combined atrial enlargement the P/PR segment ratio may be normal. This occurs only in association with distinct absolute prolongation of P-R interval and P wave. Hence, when the \( \frac{P}{P-R \text{ segment}} \) ratio is normal in the presence of distinct prolongation of P-R interval and P wave, combined atrial enlargement can be suspected. Though the P duration may overlap with the cases of right atrial enlargement with P-wave prolongation, if the P duration exceeds 0.10 second below the age of 16 years and 0.12 second above the age of 16 years, then this prolongation can be attributed to left atrial enlargement so that under these circumstances the associated finding of a normal P/PR segment ratio would establish the presence of combined atrial enlargement.

**SUMMARY**

From an electrocardiographic study of 110 patients with congenital and acquired heart disease and 62 normal adults new criteria are suggested for the diagnosis of right atrial enlargement, left atrial enlargement, and combined atrial enlargement. Right atrial enlargement is present when the P/PR segment ratio is less than 1.0. Left atrial enlargement is present when this ratio is greater than 1.6. These values apply regardless of age group. The electrocardiographic criteria for combined atrial enlargement as well as a theoretical basis for these observations are also discussed.

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The authors would like to express their appreciation to Dr. Robert P. Grant for his invaluable criticisms of this paper. May we further thank Dr. Andrew G. Morrow for making the physiologic data from his service so readily available, and Dr. John Smith of the Civil Aeronautics Association for the use of his files of normal electrocardiograms.

**SUMMARIO IN INTERLINGUA**

Super le base de un studio electrocardiographique de 110 patientes con congenite e acquisite morbo cardiac e de 62 adultos normal, nov criterios es sugerite pro le diagnose de

REFERENCES


The excretion of aldosterone and sodium was studied in 30 normal subjects and in 40 patients with various forms of heart failure before, during, and after treatment with digitalis, mercurials, and salt restriction. Healthy people consuming only 60 to 80 mEq. of sodium daily showed increased excretion of aldosterone and decreased excretion of sodium. Nine of 12 untreated patients with severe hydropic heart disease due to combined "left-and right-sided heart failure" excreted moderately increased amounts of aldosterone and reduced amounts of sodium. After cardiae recompensation was re-established sodium and aldosterone excretion was within normal limits. Seven of 8 patients with pulmonary congestion but without systemic symptoms excreted normal amounts of aldosterone, whereas 5 of 7 patients with "right-sided failure" showed moderate to excessive increases in aldosterone excretion. Increased aldosterone activity was also noted in cardiac cirrhosis following abdominal paracentesis and in the first week following myocardial infarction.

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