P-Wave Analysis in Valvular Heart Disease

By James J. Morris, Jr., M.D., E. Harvey Estes, Jr., M.D., Robert E. Whalen, M.D., Howard K. Thompson, Jr., M.D., and Henry D. McIntosh, M.D.

Deviations from the normal pattern of the P wave, in the presence of normal sinus rhythm, are caused by alterations in depolarization of the atrial chambers. Based on this assumption, a number of methods for analyzing the P wave have been devised to detect left atrial involvement in mitral valve disease. An investigation of these technics in our laboratory, applied to a series of patients with proved mitral valve lesions, demonstrated a marked lack of specificity in P waves for detecting valvular disease.

This lack of specificity prompted a study of normal P-wave morphology and more critical analysis of the P-wave changes that occurred in a series of patients with precisely defined cardiovascular abnormalities. As a result of these observations, we have proposed a new electrocardiographic measure to detect or confirm the presence of any form of left-sided valvular lesion. The significance of this measure was evaluated by correlation with the clinical, hemodynamic, and radiographic findings of the individual patients. The pathophysiologic changes that could account for these P-wave alterations are discussed.

Method

The control group consisted of 100 normal subjects (ages 20 to 69) with no historical evidence of cardiopulmonary disease and with normal physical examinations, chest x-rays, and 12-lead electrocardiograms and vectorcardiograms.

The study group included 111 consecutive patients with regular sinus rhythm who were selected because they had had complete cardiac catheterization with no evidence of intracardiac shunt. The hemodynamic evaluation in all patients except those with isolated pulmonary stenosis included catheterization of all four chambers of the heart by previously described technics.1-3 Most of the patients with pulmonary stenosis were evaluated with retrograde left ventricular catheterization as well as conventional right heart catheterization. Incompetency of the mitral or aortic valves was estimated by selective cine-angiographic or indicator-dilution technics.2 Thirteen of these patients had no hemodynamic abnormalities; the remaining patients were subdivided according to the type of valvular involvement (table 1).

Standard 12-lead direct or photographic electrocardiograms were taken at a paper speed of 25 mm. per second and a sensitivity of 1 mv. per cm. P-wave measurements were made with calipers and a hand magnifying lens. The duration and amplitude of the P wave and the duration of the P-R interval and P-R segment were measured in leads I, II, aVf, V1, V2, and V6. Spatial orientation of the mean QRS and P vectors in the frontal plane was estimated by the method of Grant.4

In addition to the standard P-wave measures noted above, the P wave in lead V1 was divided into two portions on the basis of morphology (fig.

---

From the Cardiovascular Laboratory, Department of Medicine, Duke University Medical Center, Durham, North Carolina.

Supported in part by Grants H-6960 and HE-07563-01 from the National Heart Institute, National Institutes of Health, U. S. Public Health Service, and a Grant-in-Aid from the Life Insurance Medical Research Fund, and the Martin County and Palm Beach, Florida Heart Associations.

Work completed during Dr. Morris' tenure of a U.S.P.H.S. Postdoctoral Research Fellowship.

---

Table 1

<table>
<thead>
<tr>
<th>Diagnosis Following Cardiac Catheterization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of valvular lesion</td>
</tr>
<tr>
<td>-------------------------------------------</td>
</tr>
<tr>
<td>Normal subjects</td>
</tr>
<tr>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>Mitral stenosis and aortic insufficiency</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
</tr>
<tr>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
</tr>
<tr>
<td>Mitral stenosis and insufficiency</td>
</tr>
<tr>
<td>Mitral and aortic valve lesions</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

---

Circulation, Volume XXIX, February 1964
P-WAVE MEASURES -V₁

The P wave at lead V₁ is divided into initial and terminal portions by noting the point of change in morphology. In biphasic P waves the point of reversal of polarity is selected as the point of separation into initial and terminal portions. The initial duration and amplitude and terminal duration and amplitude are measured separately.

1), and the duration and amplitude of each portion were determined. The algebraic products of the duration (sec.) and amplitude (mm.) were designated as the P initial force V₁ and the P terminal force V₉. Figure 2 gives sample calculations of the P terminal force. The spatial orientation of the initial and terminal portions of the P wave in the precordial leads was estimated separately.

Reproducibility of the measures was tested by analyzing the same record on several different occasions, as well as by analyzing several different electrocardiograms made within a period of several days. The P initial and P terminal forces could be reproduced within ±0.01 arbitrary units.

Statistical analyses with variance ratios by the Fisher F test and correlation coefficients by the Pearson product-moment method were obtained with an IBM 7072 digital computer. The data are expressed as the mean plus or minus one standard deviation from the mean.

Results

Normal Subjects

The normal group consisted of 113 persons. One hundred were chosen specifically because they lacked evidence of cardiopulmonary disease, and 13 because they were found to be normal following catheterization. Table 2 shows the results from other methods of P-wave⁵⁻¹² analysis applied to 100 normal subjects. Note that, with the three following exceptions, the P waves in this group conformed to these previous criteria for absence of left atrial disease. (1) Eleven per cent had abnormal P-wave configurations in lead II. This increased incidence of abnormal configurations can probably be explained by the strict adherence to the suggestion that any P-wave notching, except that occurring just before the peak, is abnormal. Distinct peaking or flat-topped waves were also considered abnormal. (2) Sixty-four per cent showed plus-minus type diphasic waves at V₁, compared to Leatham's finding of 13 per cent.¹² In many instances the depth of the negative waves was small and unapparent unless specifically sought. (3) Sixty-one per cent of the patients had abnormal P-wave duration to P-R segment ratios.¹⁰ Of these, 56 per cent had

Calculation of the P terminal force V₁; two representative P waves from lead V₁. The wave is divided into initial and terminal portions. The amplitude and duration are measured separately. Terminal duration multiplied by terminal amplitude = P terminal force. Note that the P terminal force may be positive (as in upper tracing) or negative (as in lower tracing). The same measures are made for the initial portion of the P wave. The value is expressed in arbitrary units (mm. sec.).
Table 2

<table>
<thead>
<tr>
<th>P-wave Analysis Applied to 100 Normal Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criterion</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>P-wave duration, lead II</td>
</tr>
<tr>
<td>P-wave amplitude, lead II</td>
</tr>
<tr>
<td>P-wave configuration</td>
</tr>
<tr>
<td>P-R interval</td>
</tr>
<tr>
<td>Frontal P vector</td>
</tr>
<tr>
<td>P/PR-segment ratio</td>
</tr>
<tr>
<td>P wave, biphasic, V₁</td>
</tr>
<tr>
<td>P wave negative deflections, V₁</td>
</tr>
</tbody>
</table>

ratios suggestive of left atrial hypertrophy (above 1.6) and 5 per cent had indices indicative of right atrial hypertrophy (below 1.0). This same lack of specificity has been noted by Human and Snyman.  

Because no normal values for the initial and terminal portions of the P wave in lead V₁ could be found, the parameters were determined in the 100 normal subjects (Table 3).  

Thirteen asymptomatic patients with normal hemodynamic findings at catheterization showed P waves comparable to those listed above. When hemodynamic data were correlated to the P-wave measurements, both left atrial pressure and pulmonary artery pressure had a negative correlation with the frontal angle of the P wave (n = 13, r = −0.68, p < 0.01). There was no significant relationship between age, sex, or QRS frontal angle and any of the P-wave data.

Abnormal Subjects

Patients with Left-sided Valvular Involvement

Eleven of the 98 abnormal subjects had pulmonary stenosis and will be analyzed separately. The remaining 87 patients had involvement of the aortic or mitral valves, or both. Application of the previously published criteria for abnormal P waves to those 87 patients is shown in Table 4. (Patients missed by these criteria are listed as false negatives). This table illustrates that the former criteria for P-wave abnormalities failed in a high percentage of cases. Failures were of two types: (1) certain criteria showed a high percentage of accuracy in valvular disease but also a high percentage of false positives in the normal groups, i.e., P/PR-segment ratios and biphasic P waves in V₁, and (2) other criteria, although suggesting few false positives in the normal subjects, failed to detect valvular disease when present, i.e., P-wave duration, amplitude, frontal angle, and P-R interval.

Study of the entire group of abnormal subjects revealed several consistent deviations. A prolongation in the P-R interval over the normal value, regardless of the lead, was common. This prolongation resulted from an increase in the P-R segment and P-wave duration, mainly the latter. The spread of these

![Figure 3](http://circ.ahajournals.org/)

**Figure 3**

*Mean and range of the P terminal force V₁. The mean value from 100 normal subjects was −0.01 and he 95-per cent confidence interval was +0.01 to −0.03. In the lower sample an abnormal P terminal force is shown. The mean value from 87 cases of left-sided valvular lesions was −0.08, the range −0.03 to −0.30.*

Circulation, Volume XXIX, February 1969
values in normal patients was so great, however, that considerable overlap occurred with those patients with valvular disease. P-wave amplitude in lead II and aV1 was increased in the abnormal group, but again showed considerable overlap with the normal group. Spatial orientation of the frontal plane vector remained unchanged by valvular disease, as did the horizontal vector of the initial portion of the P wave in the precordial leads. Vector orientation in the second portion of the P wave in the precordial leads, however, exhibited a posterior rotation with aortic and mitral disease. Precordial leads reflected this rotation by a deep negative terminal portion of the P wave at V1. The total duration of the P wave was prolonged in lead V1. This prolongation occurred entirely in the terminal portion of the P wave (table 3).

In summary, the P-wave parameters that most effectively distinguished between normal subjects and those with left-sided valvular disease were those measures centering about the terminal portion of the P wave at V1.

**Table 3**

<table>
<thead>
<tr>
<th>Measure</th>
<th>100 Normal subjects *</th>
<th>87 Patients * with aortic and mitral disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>P initial duration (sec.)</td>
<td>0.05 ± 0.02</td>
<td>0.05 ± 0.02</td>
</tr>
<tr>
<td>P initial amplitude (mm.)</td>
<td>0.6 ± 0.3</td>
<td>0.9 ± 0.7</td>
</tr>
<tr>
<td>P initial vector (degrees)</td>
<td>55° ± 14°</td>
<td>50° ± 21°</td>
</tr>
<tr>
<td>P initial force</td>
<td>0.03 ± 0.02</td>
<td>0.05 ± 0.04</td>
</tr>
<tr>
<td>P terminal duration (sec.)</td>
<td>0.03 ± 0.02</td>
<td>0.06 ± 0.03</td>
</tr>
<tr>
<td>P terminal amplitude (mm.)</td>
<td>-0.2 ± 0.3</td>
<td>-1.2 ± 0.8</td>
</tr>
<tr>
<td>P terminal vector (degrees)</td>
<td>+7° ± 12°</td>
<td>-7° ± 15°</td>
</tr>
<tr>
<td>P terminal force</td>
<td>-0.01 ± 0.01</td>
<td>-0.08 ± 0.06</td>
</tr>
</tbody>
</table>

* Mean ± 1 standard deviation.

Figure 3 shows the normal value and range for the P terminal force at V1. The value is abnormal if more negative than −0.03.

**Specific Valvular Disease**

Figure 4 shows the P terminal force at V1 for each of the 87 patients with proved left-sided valvular lesions. The type of valvular disease did not influence the percentage of abnormal values.

*Mitral Stenosis.* Twenty patients had pure mitral stenosis and seven had mitral stenosis with insignificant aortic insufficiency. These two groups did not differ in the hemodynamic, clinical, or electrocardiographic measures and were thus considered as one group. The mean left atrial pressure varied between 8 and 32 mm. Hg and the mitral diastolic gradient between 4 and 28 mm. Hg.

The measure designated as the P terminal force V1 was the most common abnormal P-wave measure in this group, with 25 of the 27 patients having values outside the normal range (fig. 4).

P-wave duration, amplitude, P-R interval, P/PR-segment ratios in lead II, and frontal P vector all failed to correlate with any of the hemodynamic measures of mitral stenosis. The P terminal amplitude and direction at V1 correlated with the left atrial pressure; however, the most significant relationship was found between the P terminal force at V1 and left atrial pressure (n = 27, r = −0.50, p < 0.01). Mitral valve area was also correlated with the P terminal force (n = 27, r = + 0.38, p < 0.01) (fig. 5).
Mitral Insufficiency. Fifteen patients had pure mitral insufficiency, with mean left atrial pressure varying from 6 to 44 mm. Hg. Five of the patients were asymptomatic, and at catheterization all were graded as having mild mitral insufficiency. The remaining 10 patients were judged both by clinical evaluation and by catheterization to have severe mitral insufficiency.

In these 15 patients, abnormal P terminal forces at V₁ were found in nine. All the patients with mild mitral insufficiency had normal values, and nine of 10 patients with severe mitral insufficiency had abnormal P terminal forces at V₁ (fig. 4).

In these patients, no relationship existed between any of the conventional P-wave data and the catheter data. However, a highly significant relationship between the severity of the mitral insufficiency, as determined by indicator dilution and cinefluorographic techniques, and the P terminal force at V₁ was demonstrated (p < 0.01, f = 9.0).

Aortic Stenosis. Valvular aortic stenosis was present in 15 patients, 13 of whom had accompanying aortic insufficiency. Three patients, ages 13, 14, and 21, had congenital aortic stenosis. Their mean systolic gradients between the left ventricle and aortic root were 25, 32, and 60 mm. Hg, respectively. The remaining 12 patients (ages 33 to 60) had sys-
tolic gradients of 55 to 126 mm. Hg, with a mean of 89 mm. Hg. In a high percentage of cases, the only abnormal findings occurred in the second portion of the P wave in V₁. Twelve of the patients had abnormal P terminal forces and three were within the normal range; these were the three patients with congenital aortic stenosis (fig. 4).

Standard methods of P-wave analysis failed to reveal more than 20 per cent abnormal values in this group, with the exception of the P/PR-segment ratios, which showed 53 per cent abnormal values. The only correlation between the hemodynamic data and the P-wave measurements was a significant correlation coefficient between the aortic systolic gradient and the P terminal force at V₁ (n = 15, r = −0.50, p < 0.05) (fig. 6). This statistical association between the P-wave data and the systolic gradient was surprising, since eight of the 15 patients had normal mean left atrial pressures.

Aortic Insufficiency. Three patients in this series had pure aortic insufficiency. All three were male patients (ages 40, 42, and 43) with symptoms of left ventricular failure for at least 1 year. Two had normal mean left atrial pressures and one had an elevated left atrial pressure of 17 mm. Hg. The P terminal force at V₁ was abnormal in two of the three (the normal case being one of the patients with normal left atrial pressure) (fig. 4).

Mitral Stenosis and Insufficiency. Sixteen patients had combined lesions of the mitral valve. The left atrial pressure varied between 8 and 32 mm. Hg, with a mean of 18 mm. Hg. Application of the previous criteria for P-wave abnormalities failed to detect abnormal P waves in more than 50 per cent of this group, with the two following exceptions: P-wave duration exceeded 0.12 second in 11 cases and the P/PR-segment ratios were abnormal in 12. Again the major abnormality centered about the terminal portion of the P wave in V₁, and all 16 subjects had abnormal P terminal forces at V₁ (fig. 4).

A positive correlation between catheter data and P-wave measurements was demonstrated between the left atrial pressure and the P initial force in V₁ (n = 16, r = +0.67, p < 0.01). A significant correlation also existed between both the pulmonary artery pressure and right atrial pressure as compared with the P initial amplitude (n = 16, r = +0.80 and +0.63, p < 0.01). There was a negative correlation between left atrial pressure and P terminal amplitude.

Combined Aortic and Mitral Disease. Significant aortic and mitral disease was present in 11 patients. Most frequently, those P-wave

![Figure 5](image-url)

**Figure 5**
The P terminal force V₁ is plotted against the mitral value area estimated from cardiac catheterization. Note the tendency for the P terminal force at V₁ to become more abnormal as the mitral valve area becomes smaller (r = +0.38, p < 0.01).

![Figure 6](image-url)

**Figure 6**
Fifteen patients with aortic stenosis. Note that the higher the aortic systolic gradient, the more abnormal is the P terminal force at V₁ (r = −0.50, p < 0.05). The three points in the lower left corner have low aortic systolic gradients and normal P terminal forces.
measures which included the second portion of the P wave in V₁ were abnormal. The P terminal force value was abnormal in all 11 cases (fig. 4).

When catheter data were compared with P-wave measures, a negative correlation was found between the mitral diastolic gradient and the P terminal amplitude and force (n = 11, r = -0.70, p < 0.05).

Patients with Pulmonic Stenosis

In 11 patients with congenital valvular pulmonic stenosis (ages 15 to 47), the systolic gradient varied from 10 to 152 mm Hg (mean 61 mm Hg). Six of the 11 patients were asymptomatic, and the remainder showed varying degrees of congestive failure.

In none of these patients did standard technics reveal abnormal P waves. Two patients had P/PR-segment ratios indicative of right atrial hypertrophy and four patients had ratios indicative of left atrial hypertrophy. The measures of the terminal portion of the P wave in V₁ were normal in all cases; the initial portion of the P wave in V₁ was abnormal in five patients, i.e., they exceeded the normal range of this measure (-0.01 to +0.07), with a mean of 0.03. These were the five patients with the highest pulmonary valvular gradients. There was a significant correlation between the pulmonary gradient and the P initial force (n = 11, r = +0.62, p < 0.05), and negative correlation between the P-wave duration in V₆ and the pulmonary gradient (n = 11, r = -0.72, p < 0.01).

Relationship of Clinical and Catheter Data to P Terminal Force

Division of the patients according to various observations on their catheter data, x-rays, electrocardiograms, and clinical status permits analysis of the results independent of the type of valvular disease. In this way the relationship of pressure, heart size, etc., to P-wave data can be evaluated.

Left Atrial Pressure. In the 50 patients with mean left atrial pressures elevated above 12 mm Hg, a significant correlation was found between left atrial pressure and the P terminal amplitude, direction, and force (n = 50, r = -0.43 to -0.59, p < 0.01). In all leads the P-wave duration correlated positively with the left atrial pressure. It should be emphasized that many of the patients with normal left atrial pressures had an abnormal P terminal force.

Congestive Heart Failure. The presence of chronic congestive heart failure within each subgroup had no effect on the measures of the terminal portion of the P wave in V₁.

Electrocardiographic Data. Thirty-two of the 87 patients with valvular disease had normal QRS, S-T, and T-wave readings by standard electrocardiographic technics. Of these 32 patients, 25 had abnormal P terminal force values. Therefore, with use of QRS, S-T, or T-wave changes alone, 55 patients of this group of 87 with valvular disease were abnormal, with the P terminal force, 74 were abnormal; and with both, 80 of the 87 cases would have had abnormal electrocardiographic readings.

### Table 5

**Relationship of Left Atrial Enlargement to Type of Valve Lesion**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Normal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral stenosis</td>
<td>3 (1)*</td>
<td>6 (1)*</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>5 (3)*</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>11 (1)*</td>
<td>3 (1)*</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>1 (1)*</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mitral stenosis and insufficiency</td>
<td>0</td>
<td>3</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Aortic and mitral valve disease</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total left-sided valve disease</td>
<td>21 (6)*</td>
<td>15 (2)*</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonic stenosis</td>
<td>6 (6)*</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Numbers in parentheses are the number of patients in a particular category with normal P terminal force V₁. The remainder in each category had abnormal P terminal force.


X-ray Data. X-rays in four views and barium swallow were suitable for grading left atrial size in 55 of the 111 patients who underwent complete cardiac catheterization. Table 5 shows the grading of the left atrial size, grouped according to the type of valve lesion present. It was interesting to note that of the 21 patients with normal-sized left atria and proved left-sided valvular lesions, 15 had abnormal P terminal forces. Thus, the presence of a large left atrium (radiographically) was unnecessary to produce an abnormal P terminal force. Conversely, two patients with mild left atrial enlargement had normal P terminal forces.

Relationship of Catheter Data to P Initial Force

Since there existed a correlation between the P terminal force at V1 and the presence of left-sided valvular lesions, an attempt was made to determine if there was a relationship between the P initial force at V1 and the pulmonary artery pressure in patients with left-sided valvular lesions. Such a relationship has already been shown to exist in the cases of pulmonic stenosis. Statistical analysis in the 26 patients with a mean pulmonary artery pressure greater than 30 mm. Hg indicated a correlation between the P initial force and the pulmonary artery pressure \((n=26, r=+0.42, p<0.05)\). The magnitude of this increase in the P initial force at V1 was so slight as to be inapparent except by statistical analysis. In addition, no significant correlation was found with the frontal angle of the P wave and the P-wave amplitude in leads II, aVR, or V1.

Discussion

Application of standard methods of P-wave analysis to the 200 patients in this study (113 normal subjects and 87 with aortic or mitral valvular disease) has shown that each measure is indicative of left atrial involvement with varying degrees of sensitivity and selectivity (table 6). The measure we have designated as the P terminal force, which appears to be the most sensitive and selective method, was successful in 92 per cent of the entire 200 cases in this series for predicting aortic or mitral valve involvement.

In actual practice, measurement of the P terminal force is simple. Visual inspection alone can easily identify an abnormal P wave at V1. A terminal portion of the P wave at V1, one box in depth \((-1.0 \text{ mm.})\) and one box in duration \((0.04 \text{ sec.})\), yields a P terminal force of \(-0.04\). Any P-wave negativity of this size or larger is abnormal (fig. 3).

Previous methods of P-wave analysis probably failed for two major reasons: (1) selection of frontal plane leads for study of P-wave abnormalities is ineffective because the spatial orientation of the atrial vector fails to change direction in this plane with known atrial involvement,14, 15 and (2) many criteria depend upon changes in the duration of the P wave. We have demonstrated in this study that although changes in P-wave duration do consistently occur with aortic and mitral disease, these changes are small; furthermore, the variations occurring in normal subjects are rather wide.

The second portion of the P wave in the surface electrocardiogram has been shown to represent electrical depolarization of the left atrium alone.16-18 Therefore, to detect left atrial involvement electrocardiographically, a method that utilizes this evidence would seem most logical. In normal subjects the vector from the second portion of the P wave is most nearly perpendicular to the axis of V1 and, therefore, little deflection is inscribed. With left-sided valvular lesions, data from this laboratory and others9 have shown that the vector from the second half of the P wave rotates posteriorly in the horizontal plane. With this rota-

**Table 6**

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Per cent correct recognition</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-wave duration, lead II</td>
<td>72</td>
</tr>
<tr>
<td>P-wave amplitude, lead II</td>
<td>63</td>
</tr>
<tr>
<td>P-wave configuration</td>
<td>70</td>
</tr>
<tr>
<td>P-R interval</td>
<td>65</td>
</tr>
<tr>
<td>Frontal P vector</td>
<td>57</td>
</tr>
<tr>
<td>P/PR-segment ratio</td>
<td>52</td>
</tr>
<tr>
<td>P wave, biphasic, lead V1</td>
<td>62</td>
</tr>
<tr>
<td>P wave, negative deflection V1</td>
<td>80</td>
</tr>
<tr>
<td>P terminal force V1</td>
<td>92</td>
</tr>
</tbody>
</table>
tion, the second portion of the P wave in V₁ takes a deep negative deflection. We have also found that the prolongation of the P wave in aortic and mitral valve disease, although rather small, is confined in lead V₁ to the second portion of the P wave at V₁. If this portion does indeed represent left atrial activation, the reason behind the success of this method becomes obvious.

Most studies involving P-wave analysis have noted the abnormalities associated with mitral stenosis or mitral insufficiency. Sodi-Pallares and Calder have noted biphasic P waves in V₁ in the presence of hypertension. Sutnick and Soloff have reported posterior rotation of the atrial vector during acute left ventricular failure. We also have unpublished observations showing abnormal P terminal forces at V₁ in hypertensive vascular disease, idiopathic myocardial hypertrophy, and subaortic stenosis. Thus, the P terminal force not only indicates aortic and mitral valvular disease, but reflects any form of left-sided heart disease. For this reason, we have chosen the nonspecific term "left atrial involvement," instead of stating that this electrocardiographic sign indicates left atrial enlargement, increased pressure, or hypertrophy; in fact, results from this study show that the P terminal force may be abnormal even in the face of normal mean left atrial pressure and normal left atrial size by x-ray examination. Because an abnormal P terminal force is present in all those conditions listed above, this electrocardiographic sign loses its diagnostic significance as a sign of valvular involvement. This measure may be equally abnormal with any type of aortic or mitral valvular disease, or with those other causes listed above. Its clinical value appears to lie chiefly in its use as an electrocardiographic criterion for recognizing the presence of any of these causes of left-sided heart disease.

In addition, we have found the P terminal force V₁ a helpful sign in assessing the hemodynamic significance of a valvular lesion after its clinical detection. Previous attempts to relate P-wave abnormalities to the degree of atrial enlargement, to the left atrial pressure, or to the pulmonary capillary pressure have yielded conflicting results. In the present study, attempts to correlate the magnitude of the change in the P terminal force to any one measure were unsuccessful. Separation of the patients according to type of valvular lesion, however, demonstrated a relationship between the characteristic hemodynamic abnormality of that particular lesion and the P terminal force. In mitral stenosis, the most significant correlation existed between the measures of the P terminal force and the catheter measures of the severity of the stenosis, i.e., mean left atrial pressure, diastolic gradient, and mitral valve area. In mitral insufficiency, the best relationship was found between the P terminal force and the degree of insufficiency. When aortic stenosis was present, the systolic gradient showed a significant correlation with the abnormal P-wave measures. It is these characteristics of the abnormalities of the P terminal force that are clinically useful in assessing the severity of a given valve lesion, once it is detected.

Of the 87 patients with left-sided valvular disease, 75 were found to have an abnormal P terminal force. Of the 12 patients with normal values, the following clinical comments are pertinent: two patients had mitral stenosis, one with minimal symptoms and borderline catheter studies and the other with symptoms and catheter findings serious enough to warrant surgical intervention. Five had mild mitral insufficiency without symptoms and were studied because of the presence of a murmur alone; one patient had severe mitral insufficiency and symptoms of decompensation. Three patients had congenital aortic stenosis; all had low valvular gradients and normal electrocardiograms, and were asymptomatic. One patient had severe aortic insufficiency. Thus, of the 12 cases with a normal P terminal force, nine can be classified as minimal valvular disease and only three as severe.

Attempts to relate other clinical data, such as age, sex, and presence or absence of congestive heart failure, all failed to produce consistent changes in the P-wave measures sufficiently large to be useful.

The 11 patients with valvular pulmonic
P-WAVE ANALYSIS

stenosis were included in this study for several reasons. Studies on these patients confirmed a relationship between the P initial force in V1 and right-sided valvular disease, just as the P terminal force in V1 is associated with left-sided valvular disease. This group also exhibited a consistent set of findings that appear in "right-sided strain," findings which were conspicuously absent in the presence of pulmonary hypertension secondary to mitral valve disease. These patients also demonstrated that cardiomegaly, digitalis, edema, etc., play no important role in producing abnormalities of P terminal force found in the patients with aortic and mitral valve disease.

The actual electrophysiologic and anatomic phenomena underlying these changes remain unsettled. Slight posterior rotation of the left atrium could explain an increase in the amplitude without any change being necessary in the amount of voltage generated from the left atrium as recorded at V1. However, Reynolds has demonstrated during thoracotomy that a higher potential is generated in the surface electrograms of patients with mitral stenosis than that generated in normal subjects. He was unable to relate this to the P-wave amplitude in the limb leads. As discussed above, in left atrial involvement little change occurs in the frontal plane of the P vector, but the vector rotates posteriorly in the horizontal plane. Therefore, the changes seen in the precordial leads may well reflect this increased amplitude. The alteration of the vector may be inapparent in the limb leads, however. Thus, actual anatomic positional changes of the left atrium or increased voltage production at the atrium itself could explain some of these changes. Neither explanation can account for the entire change noted, for we have observed a consistent increase in P-wave duration associated with valvular disease. This increased duration could result from decreased conduction velocity through the atria or from a longer path to be traveled.

Summary

Electrocardiographic analysis of the P waves occurring in a series of 113 normal subjects and 100 patients with specifically defined valvular lesions are reviewed. The former methods of analysis showed a marked lack of specificity.

By dividing the P wave in lead V1 into initial and terminal portions, a measure designated as the P terminal force has been derived. This measure is of value in two respects: (1) it correctly separates normal subjects from those patients with left-sided valvular lesions in 92 per cent of this series and (2) once a given valve lesion is suspected clinically, this measure enables one to make an estimation of the severity of that lesion from the degree of abnormality of the P terminal force at V1. The P terminal force does not indicate the type of valvular disease present, nor does it correlate with any one specific hemodynamic measure. The abnormality does appear to be related, within each separate type of valve disease, to the specific hemodynamic abnormality of that type of valvular involvement.

The anatomic and electrophysiologic changes that might relate to these P-wave changes are discussed.

References


The Impervious Interventricular Septum

The idea having cropped up in the first place presumably as a result of Vesalius’ remarks about the septum, it could have occurred to both Servetus and Columbus even theoretically, without experiments. It is quite probable that they discovered the same thing independently of one another. It might have been as with many other discoveries in science: once somebody opens a gate, whoever looks through it has to notice the very same things. And the man who opened that gate for medical science at that time certainly was Vesalius.

Hundreds of thousands of surgeons had been cutting veins for one thousand five hundred years, until the blood flowing from these patients would have filled entire lakes. A slight headache, fever or even sneezing was considered sufficient indication. They knew exactly where to tie the arm and cut the vein, they knew the rules governing the flow of blood by heart. Scientists (Vesalius among them) wrote treatises about the way the blood came down, but no one, with the single exception of the Spanish veterinariansmith Reina, observed the conflict between the theory and their practical experience.—TIBOR DOBY, M.D. Discoverers of Blood Circulation. From Aristotle to the Times of Da Vinci and Harvey. New York, Abelard-Schuman, 1963, p. 168.
P-Wave Analysis in Valvular Heart Disease

JAMES J. MORRIS, JR., E. HARVEY ESTES, JR., ROBERT E. WHALEN,
HOWARD K. THOMPSON, JR. and HENRY D. MCINTOSH

Circulation. 1964;29:242-252
doi: 10.1161/01.CIR.29.2.242

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/29/2/242

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/