Relationship of Fibrillatory Waves and P Waves in the Electrocardiogram

By Robert H. Peter, M.D., James J. Morris, Jr., M.D., and Henry D. McIntosh, M.D.

ANALYSIS of the P wave in the electrocardiogram has been found to be a useful indicator of certain alterations of cardiac function. The patterns of P mitrale and P pulmonale are generally well recognized. Morris and co-workers reported that differences in the contour of the terminal portion of the P wave in precordial lead $V_1$ separated subjects with essentially normal hemodynamic findings from those with significant left-sided valvular lesions. This index, called the "P terminal force," has also been shown to be an accurate indicator of left ventricular hypertrophy in patients with nonvalvular heart disease.2

Little diagnostic importance has been attached to the atrial fibrillatory wave. Coarse fibrillatory waves in lead $V_1$ are said to be more common in patients with rheumatic heart disease, while fine fibrillatory waves are generally found in patients with arteriosclerotic heart disease.3-5

The present study was designed to determine whether there was a relationship between the P terminal force and the size of the fibrillatory wave.

Method

One hundred consecutive patients with chronic atrial fibrillation successfully converted to sinus rhythm by direct-current shock were studied. Electrocardiograms obtained during atrial fibrillation were compared to subsequent tracings in which normal sinus rhythm was present. The interval between these two records did not exceed 72 hours and frequently was as short as 1 hour. Twelve-lead electrocardiograms were obtained by direct or photographic recorders at standard speed and sensitivity. Patients with tracings showing premature atrial contractions, nodal rhythm, or atrial flutter were excluded from this study.

Each patient was on a maintenance dose of a digitalis preparation for control of the ventricular rate in atrial fibrillation. The drug was omitted for 24 hours prior to restoration of sinus rhythm and resumed the following day. No patients had

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recognizable symptoms or signs of digitalis intoxication. Administration of quinidine gluconate,* 0.33 or 0.50 g every 8 hours was commenced at least 24 hours before the cardioversion and continued indefinitely.

The fibrillatory wave with the greatest size (amplitude) was measured in lead V1 with the aid of calipers. This measurement was made from the upper edge of the trough to the upper edge of the peak, and expressed in millimeters (fig. 1). Fibrillatory waves in lead V1 with an amplitude of 1.0 mm or greater were designated as coarse waves; those less than 1.0 mm were defined as fine fibrillatory waves. The presence of a single coarse fibrillatory wave was sufficient to classify the patient as having coarse fibrillatory waves. Care was taken to avoid measuring a wave that was superimposed on a T or U wave (fig. 2). Initially, long strips of lead V1 were recorded and reviewed for the largest fibrillatory wave present. However, fibrillatory waves occurring during three or four cardiac cycles were found to be equally satisfactory for evaluation.

The P terminal force was measured as previously described when normal sinus rhythm was present. A division of the P wave in lead V1 into initial and terminal portions was made on the basis of a change in contour. The terminal portion may be a positive or negative wave. The duration of the terminal portion of the P wave was measured in seconds. The largest amplitude with its proper sign, positive or negative, was measured in millimeters. The product of the amplitude (mm) and the duration (seconds) gives a vector measure termed the "P terminal force" (fig. 3). On the basis of a previous study of 100 normal subjects and 150 autopsies, values of −0.03 or more positive were considered normal, and values of the P terminal force of −0.04 or more negative were considered abnormal.

The size of the left atrium was estimated from the roentgenograms independently by two of the authors and graded as normal or slightly, moderately, or markedly enlarged. A four-view cardiac X-ray series with barium swallow was available for this phase of the study in 75% of the cases; in 25% on posteroanterior and lateral standard roentgenograms were available.

A fourfold table was employed for statistical comparison of the fibrillatory wave amplitude and P terminal force, as well as for the left atrial size. Statistical significance was tested by applying the chi square test.8

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*Kindly supplied as Quinaglute(R) by Wynn Pharmaceuticals, Philadelphia, Pennsylvania.
coarse fibrillatory wave and an abnormal P terminal force, and 17 had a fine fibrillatory wave and a normal P terminal force. Only 17 patients did not have this association: eight had an abnormal P terminal force and a fine fibrillatory wave and nine had a normal P terminal force and a coarse wave. Thus, with an 83% degree of accuracy, one could predict from the amplitude of the fibrillatory wave whether a normal or abnormal P terminal force would be present when sinus rhythm was restored. This association between fibrillatory wave size and P terminal force was statistically significant ($P < 0.001$).

**Rheumatic Heart Disease**

**Analysis of P Terminal Force**

Forty-nine of 60 patients (82%) with rheumatic heart disease had abnormal P terminal forces with values ranging from $-0.04$ to $-0.35$.
to $-0.40$ mm. In table 1 the 60 patients are classified according to their valvular lesions. The incidence of abnormal P terminal forces varied from 70 to 88% in each diagnostic category (table 1). As only two patients had isolated aortic valve disease, the incidence of an abnormal P terminal force in this subgroup could not be ascertained.

**Analysis of Fibrillatory Waves**

In the 60 patients with rheumatic heart disease, coarse fibrillatory waves with amplitudes of 1.0 to 4.5 mm were present in 51 patients (85%). In the various types of valvular lesions coarse fibrillatory waves were found in 63 to 93% (table 1). Again in isolated aortic valve disease, the numbers were insufficient for evaluation.

**Comparison of Fibrillatory Wave Amplitude and P Terminal Force**

From the preceding observations in patients with rheumatic heart disease, the similarity in frequency of coarse fibrillatory waves (85%) and an abnormal P terminal force (82%) is apparent. Comparison of individual patients before and after conversion from atrial fibrillation demonstrates the close association of fibrillatory and P waves (fig. 5). Fifty of the 60 patients (83%) had the association of fine fibrillatory waves with normal P terminal force or coarse fibrillatory wave with an abnormal P terminal force. This relationship was statistically significant by chi square test ($P < 0.01$).

**Nonrheumatic Heart Disease**

**Analysis of P Terminal Force**

Forty patients with nonrheumatic heart disease were studied: 25 (63%) had abnormal P terminal force with a range in values of $-0.04$ to $-0.30$. Thirty of the 40 patients (75%) had either coronary artery disease, manifested by angina or myocardial infarction, or both, or significant diastolic hypertension. Seventeen of these 30 patients (57%) had abnormal P terminal force. Ten of the 40 patients with nonrheumatic heart disease had miscellaneous forms of heart disease; eight of them (80%) had abnormal P terminal force (table 2).

**Analysis of Fibrillatory Waves**

Of the 40 patients with nonrheumatic heart disease, 24 (60%) had coarse fibrillatory waves ranging in amplitude from 1.0 to 3.0 mm. Of the 30 patients with coronary artery or hypertensive vascular disease, 16 (53%) had coarse fibrillatory waves (table 2). Of the 10 patients with miscellaneous forms of heart disease, eight (80%) had coarse fibrillatory waves.

**Comparison of Fibrillatory Wave Amplitude and P Terminal Force**

Again the similarity in the frequency of coarse fibrillatory waves (60%) and abnormal P terminal force (63%) in these patients with nonrheumatic heart disease is apparent. Twenty-one patients had coarse fibrillatory waves and subsequently an abnormal P terminal force. Twelve patients had fine fibrillatory waves associated with normal P terminal force. This type of agreement in the two measures was, therefore, present in 33 of the 40 patients (82%). This relationship between coarse and fine fibrillatory waves and ab-

**Table 1**

*Measures of Atrial Activity in Sixty Patients with Rheumatic Heart Disease*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Sinus rhythm: P terminal force</th>
<th>Atrial fibrillation: fibrillatory waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td>Mitral insufficiency</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Mitral stenosis and insufficiency</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Aortic stenosis and insufficiency</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Mitral and aortic lesions</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>49</td>
</tr>
</tbody>
</table>

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FIBRILLATORY WAVES AND P WAVES IN ELECTROCARDIOGRAM

FIBRILLATORY AND P WAVES
RHEUMATIC HEART DISEASE

E.S., 48 yrs. W.M.
Combined Valvular Lesions

Electrical Conversion

A - Coarse fibrillatory wave

B - Abnormal P Terminal force

H.B., 53 yrs. W.M.
Mitral Insufficiency

Electrical Conversion

C - Fine fibrillatory wave

D - Normal P Terminal force

Figure 5
Sample electrocardiograms of lead V1 obtained from two white men (WM) with rheumatic heart disease in atrial fibrillation and then in sinus rhythm. A and B show coarse fibrillatory waves (1.8 mm) followed by an abnormal P terminal force (−0.12). C and D show fine fibrillatory waves (less than 0.5 mm) followed by a normal P terminal force (0.0).

Table 2
Measures of Atrial Activity in Forty Patients with Nonrheumatic Heart Disease

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Sinus rhythm: P terminal force</th>
<th>Atrial fibrillation: fibrillatory waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Coronary artery and hypertensive vascular disease</td>
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<td>17</td>
</tr>
<tr>
<td>Miscellaneous:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital heart disease</td>
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<td>1</td>
</tr>
<tr>
<td>Idiopathic myocardial hypertrophy</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonary heart disease</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Marfan's syndrome</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>&quot;Lone&quot; fibrillator</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>25</td>
</tr>
</tbody>
</table>

normal P terminal force was statistically significant (P < 0.001).

Serial Observations of the Relationship of Fibrillatory Wave Amplitude and P Terminal Force

In 10 patients multiple comparisons were made between the size of the fibrillatory wave and the P terminal force. In these patients atrial fibrillation recurred after sinus rhythm had been present for at least several days. These patients underwent electrical conversion two to four times and thus provided the opportunity to compare the P and fibrillatory wave measurements on several occasions. Measurement of the fibrillatory wave correctly predicted whether a normal or an abnormal P terminal force would be present after 22 of 25 conversions to sinus rhythm. Furthermore, only two patients demonstrated the contradictory finding of a coarse fibrillatory
wave with a normal P terminal force or a fine fibrillatory wave with an abnormal terminal force. These observations demonstrated the stability and reproducibility of both of these measures.

Comparison of Left Atrial Size and Electrocardiographic Measures

In 60 patients with rheumatic heart disease, 43 had roentgenograms suitable for evaluation of atrial size. Enlargement of the left atrium was present in 72%. The majority of the patients with rheumatic heart disease did have an abnormal P terminal force, coarse fibrillatory waves, and an enlarged left atrium. Superficially this would suggest a relationship between these measures. However, the distribution of cases did not show a statistical association by chi square testing between atrial size and either of the electrocardiographic measures.

Twenty-eight of the patients with non-rheumatic heart disease had suitable roentgenograms available for review. An increase in the size of the left atrium was detected in five (18%). Again, no association by chi square testing was evident between the electrocardiographic data of atrial activity and the X-ray assessment of left atrial size. Thus, roentgenographic enlargement of the left atrium was neither necessary for, nor associated with, abnormal P terminal force or coarse fibrillatory waves.

Discussion

This study demonstrates that the amplitude of fibrillatory waves is not randomly fine or coarse. The association of coarse fibrillatory waves with abnormal P terminal force and fine fibrillatory waves with normal P terminal force was of a high order of statistical significance. On the basis of this relationship, it is possible to define accurately the limits of coarse and fine fibrillatory waves, and also to consider their implications.

Previous reports have suggested that a fibrillatory wave size (amplitude) of 0.5 mm represents the dividing line between a coarse and fine fibrillatory wave.3,4 Inspection of the data in figure 4 shows that if a fibrillatory wave of greater than 0.5 mm were considered as coarse rather than a value of 1.0 mm or greater, little difference in the total number of patients with coarse fibrillatory waves would have resulted. Furthermore, statistical evaluation with 0.5 mm as the upper limits of a fine wave did not significantly alter the results from those presented above using less than 1.0 mm as the level. Since the results were comparable, and a fibrillatory wave amplitude of 1.0 mm is more easily measured, this value was chosen as the lowest limit of a coarse fibrillatory wave.

It has been implied in the past that the P wave can be used to indicate the etiological type of heart disease present, that is P pulmonale, P mitrale. More recently, studies from this laboratory and others have shown that an abnormal P terminal force occurs in many situations. With this observation, one is no longer justified in assuming that an abnormal P wave is indicative of a particular etiological type of heart disease. Thus, the abnormal P terminal force reflects a variety of pathological conditions that influence left atrial electrical activity. In mitral stenosis the abnormal P terminal force correlates with the estimated valve size, in aortic stenosis with the aortic systolic gradient, and in mitral insufficiency with the degree of incompetency of the valve.1 In nonvalvular forms of heart disease where this measure is abnormal, that is, in hypertensive vascular disease,2 idiopathic myocardial hypertrophy,7 and acute congestive heart failure,8 it probably reflects atrial contraction into a noncompliant left ventricle.

In the instances just cited, there is no common feature which would explain the abnormal P terminal force. An elevated mean left atrial pressure, an enlarged left atrium, or a particular etiological form of heart disease can be associated with an abnormal P terminal force at times, but in fact, any or all of these features may be absent. Thus, an abnormal P terminal force does not imply a specific pattern of atrial size, hemodynamic abnormality, or etiological diagnosis.

In the case of the electrocardiographic interpretation of left ventricular hypertrophy,
it has long been recognized that this electrical diagnosis does not carry implications about the etiology of the heart disease present. The electrical pattern of hypertrophy can occur in congenital, rheumatic, and other acquired heart disease, in dilatation or concentric hypertrophy of the ventricle, in normal sized and enlarged hearts, as well as in hearts with increased or normal intraventricular pressures. Thus, the electrical diagnosis of left ventricular hypertrophy does not imply a specific hemodynamic measure, an etiological type or pattern of heart disease.

It would appear, then, that the former practice of reading abnormal P waves as indicating an etiological form of heart disease is incorrect. An abnormal P terminal force should be interpreted with the same implications as the ventricular complexes are in left ventricular hypertrophy. We, therefore, suggest that an abnormal P terminal force represents left atrial hypertrophy or strain.

It has been stated that coarse fibrillatory waves occur in rheumatic heart disease and fine fibrillatory waves in arteriosclerotic heart disease. Eighty-five per cent of the patients in this series with rheumatic heart disease and 60% with nonrheumatic heart disease had coarse fibrillatory waves. Although these data indicate that the correlation is generally true, the difference is so slight that it is useless as an electrocardiographic sign for separating these two forms of heart disease. Since a coarse fibrillatory wave corresponds to an abnormal P terminal force, it appears more accurate to regard a coarse fibrillatory wave as indicating left atrial hypertrophy or strain.

The selection of the term "left atrial hypertrophy" seems to be appropriate for many of the conditions in which these measures remain constant over months or years. The use of the term "left atrial strain" requires justification. Sutnick and Soloff reported the frequent occurrence of posterior rotation of the atrial vector in acute left ventricular failure. Posterior rotation of the atrial vector will produce an abnormal P terminal force. We have noticed the appearance and disappearance of an abnormal P terminal force with the onset and regression of acute congestive failure. Skoulas and Horlick noted, with a few important exceptions, the constancy of the amplitude of fibrillatory waves over a period of months. These observers reported that coarse fibrillatory waves could become fine when congestive failure was relieved, and that coarse waves might appear when congestive failure developed. Therefore, both the P terminal force and the fibrillatory wave amplitude have been shown to change in acute congestive heart failure. Changes in these measures occurring in a period of days are not consistent with atrial hypertrophy, but are better described as atrial "strain."

The use of an abnormal P terminal force and a coarse fibrillatory wave in routine electrocardiography can be demonstrated by several examples. Although tracings with a vertical QRS axis and a normal P terminal force cannot be read as abnormal, one with a vertical QRS complex and an abnormal P terminal force should be read as showing a vertical axis and left atrial hypertrophy or "strain." This combination is strongly suggestive of mitral stenosis and may prove a helpful electrocardiographic clue. Analysis of fibrillatory waves in the electrocardiogram of a patient with a vertical QRS axis could be equally important. From an electrocardiographic point of view, the combination of a vertical axis and a coarse fibrillatory wave indicating left atrial hypertrophy or "strain" strongly suggests that mitral stenosis is present. As a second example, the electrocardiographic diagnosis of left ventricular hypertrophy can be cited. This electrocardiographic diagnosis is generally based on the presence of several abnormalities in the QRS complex, the ST segment, and the T wave. An abnormal P terminal force has recently been shown to be just as accurate and sensitive a criterion of left ventricular hypertrophy as those mentioned above. Since the coarse fibrillatory wave is so closely related to the presence of an abnormal P terminal force, we suggest that it would be a valuable additional criterion in diagnosing left ventricular hypertrophy when atrial fibrillation is present.
The data in this study do not permit an assessment of the fibrillatory wave amplitude as related to right atrial hypertrophy. It is entirely possible that coarse fibrillatory waves might be found in patients with right atrial hypertrophy. Unfortunately, the rarity of atrial fibrillation in pure forms of right-sided heart disease (tricuspid stenosis, pulmonic stenosis, and primary pulmonary hypertension) was such that the necessary observations could not be made.

It is possible that a relationship exists between the atrial flutter waves in the electrocardiogram and left atrial hypertrophy. We examined the records of 15 patients with atrial flutter who underwent electrical conversion to sinus rhythm. Measurement of the flutter waves and the P terminal force did not reveal any consistent relationship. The number of tracings for comparison was small, and this point, therefore, deserves further consideration.

Finally, a word should be said about the actual clinical use of these criteria for left atrial hypertrophy or strain when either sinus rhythm or atrial fibrillation is present. Both measures can be read with the aid of calipers, without special measuring devices, and from a short strip of lead V1 in routine clinical tracings. Left atrial hypertrophy or strain is present when a P terminal force at lead V1 is −0.04 or more negative, or a fibrillatory wave at lead V1 is 1.0 mm or greater.

Summary

The amplitude of the fibrillatory waves and the P terminal force were compared in 100 consecutive patients. These electrocardiographic measures of atrial activity were made first in atrial fibrillation and then after electrical conversion to sinus rhythm. When the same patient was studied, the occurrence of a coarse fibrillatory wave and an abnormal P terminal force, or a fine fibrillatory wave and a normal P terminal force, was found to be a highly significant relationship.

This relationship suggests that the factors responsible for the characteristic of the terminal portion of the P wave recorded in sinus rhythm probably determine the magnitude of the fibrillatory wave that occurs when atrial fibrillation is present. The finding of a coarse fibrillatory wave or an abnormal P terminal force should suggest the electrocardiographic diagnosis of left atrial hypertrophy or "strain."

References

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