**Electrocardiographic Features of P Waves from Patients with Transient Atrial Fibrillation**

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**Summary**

The electrocardiographic characteristics of P waves from patients having more than two episodes of transient atrial fibrillation (T-af) were examined during an episode-free period. The P waves of these patients recorded from the X Y Z leads of Frank's lead system on high-speed and high-gain recordings revealed longer durations than those of the normal control subjects (p<0.001), while the standard 12 lead ECG did not show any difference in width between the 2 groups. The configuration of the P loops on VCGs showed abnormal irregularities, such as bites or notches, in 16 of 28 cases of the T-af group. The maximum magnitudes of the P loops in VCGs and those in SVECGs were greater in the T-af group than those in the normal group (p<0.05). Body surface maps during atrial excitation showed that the duration of the anterior maximum was longer than that of the left maximum, and that the values of both maxima of the T-af group were longer than those of the normal group (p<0.01). These findings suggest the presence of intra-atrial conduction disturbances in patients with this disease, which might contribute to the genesis of this arrhythmia. Furthermore, these electrocardiographic characteristics of the P waves in the T-af group can be used as predictive and diagnostic signs of this arrhythmia even during an episode-free period.

**Additional Indexing Words:**

Spatial velocity electrocardiogram, Abnormal P vector loop, Body surface map of P wave, Conduction disturbance

**A Trial fibrillation** may develop either as a paroxysmal transient form or as an established chronic form. The former is usually characterized by attacks of arrhythmias lasting no more than several days and reverting to sinus rhythm spontaneously or after therapeutic means. Therefore, the basic rhythm in these cases is usually of sinus origin. Transient atrial fibrillation is known to occur not only in the presence of organic heart disease, but also in

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In the former condition, left atrial enlargement or hypertrophy appeared to be a contributing factor in the genesis of this arrhythmia. In the latter condition, without gross anatomical changes in the heart, the major factors causing this arrhythmia have yet to be clarified. Evaluation of the amplitude, duration and morphology of the P waves in the latter group of patients with transient atrial fibrillation (T-af) during the episode-free period has been utilized in attempts to identify functional abnormalities of the right or left atrium. Electrocardiographic features of the P waves shortly before the onset or immediately after the termination of atrial fibrillation have been described by Masini et al and Bellet. They described increased amplitude and abnormal morphologies of the P waves during these periods. As to the P wave morphology during the episode-free period in patients with transient atrial fibrillation, however, there have been conflicting reports in the literature. Bellet indicated that there were no P wave abnormalities in benign atrial fibrillation. On the other hand, Davies and Ross pointed out the prolongation of P wave duration in lead II in significant numbers of these patients. Robitaille and Phillips reported that electrocardiographic analysis of the P wave in 36 patients with this arrhythmia showed no prolongation of the P wave in lead II, but demonstrated a significant increase in the P terminal force and the P total force in V1 compared to those of the normal group. Therefore, the present study was done to examine the electrocardiologic characteristics of the sinus P wave using noninvasive techniques during the episode-free period in those patients with frequent attacks of T-af and to identify some predictive factors for the diagnosis of this condition.

MATERIALS AND METHODS

In this study, we defined “transient atrial fibrillation” as the paroxysmal form of this arrhythmia lasting between 2 and 36 hours based on subjective symptoms with attacks having been documented by ECG recordings on at least two occasions. The arrhythmia reverted to sinus rhythm spontaneously or following the use of antiarrhythmic agents. Therefore, the basic rhythm was sinus rhythm. Twenty-eight patients having episodes of T-af were studied. There were 20 men and 8 women, aged from 21 to 75 years (mean 50). All patients had at least two documented episodes of transient atrial fibrillation within 3 months before the study. Most of them visited our hospital because of this arrhythmia, but 5 cases presented with mild hypertension and 2 ischemic heart disease. Patients with hypertension showed normal blood pressure with treatment with either thiazide or β-blocker. They showed no abnormal ECG changes, except high voltage QRS in V5 exceeding 2.7 mV, or cardiac en-
largement on the chest X-ray. Echocardiography revealed normal cavity size and wall thickness in both ventricles, and normal left atrial dimension (less than 40 mm). Patients with ischemic heart disease received Ca$^{2+}$-antagonists because of anginal pain. They had normal ECGs at rest but demonstrated slight (0.05 mV) ST depression after the submaximal exercise test. Normal cardiac silhouette on chest X-ray and normal echocardiographic findings were obtained. Since these 7 cases showed no apparent signs of left or right atrial enlargement, they were included in the present study as the T-af group. We excluded those cases with valvular or congenital heart disease, and cases showing an enlarged atrium on chest X-ray or on UCG. Cases of hyperthyroidism were also excluded.

As the control group, 40 normal subjects, who were volunteers, students and members of the medical staff of our university, were examined. There were 28 men and 12 women, with ages between 22 and 45 years (mean 38). They were defined as normal based on history, physical findings and the chest roentgenogram. UCGs of these normal groups were not examined. None of them demonstrated abnormalities on the standard 12 lead ECG or in the Frank lead VCG. We examined all cases in the T-af and normal groups using ECG, VCG spatial velocity electrocardiogram (SVECG) (VA 3H, Fukuda Denshi Co., Tokyo) and spatial magnitude electrocardiogram (SM-ECG). Body surface maps of P waves were recorded from 30 normal subjects and 13 T-af cases using the mapper (HPM 5100 or HPM 6500, Chunishi Denshi Co., Nagoya) originally described by Yamada. All of these examinations were done during regular sinus rhythm within a month after the last episode of T-af and during a time when patients had been free of T-af episodes for at least 3 days. Five of 28 cases were given digitalis, but other antiarrhythmic agents were withdrawn at least 24 hours before the study. The readings of the VCGs were done by 3 cardiologists independently and defined as abnormal when all 3 of them were in agreement.

Data were analyzed statistically using Student's t-test.

**Results**

Conventional 12 lead ECG recordings in the T-af group showed the following findings: The amplitudes of P waves in lead II were within normal range (<0.25 mV) in all 28 cases. The P wave durations in lead II were less than 110 msec in 26 of 28 cases, but in 2 cases the P waves were longer than 110 msec. The mean P wave duration in 28 cases was 92 ± 12 msec. The P terminal forces in V1 were within normal range except in 4 of the 28 cases in the T-af group in which they exceeded −0.003 mm·sec.
In the normal group, all 40 subjects demonstrated normal configuration, amplitude and duration of P waves, and normal P terminal force in V1. The mean values of all these parameters were not significantly different between the normal and the T-af group.

The high-speed (100 cm/sec) and the high-gain (0.1 mV/cm) recordings of the orthogonal XYZ leads of Frank's lead system, however, showed a longer duration of the P waves in the T-af group as compared to the control.

![Duration of P Wave](image)

**Table I. The Values of the Direction and the Magnitude of the P Vector in the Normal and T-af Groups**

<table>
<thead>
<tr>
<th>Plane</th>
<th>Direction (degree)</th>
<th>Amplitude (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>T-af</td>
</tr>
<tr>
<td>F</td>
<td>70±13</td>
<td>68±16</td>
</tr>
<tr>
<td>S</td>
<td>105±13</td>
<td>108±22</td>
</tr>
<tr>
<td>H</td>
<td>21±29</td>
<td>2.5±42</td>
</tr>
</tbody>
</table>

Values are mean±SD.

F, S, H denote the frontal, the left sagittal and the horizontal planes, respectively. The comparison was done between the normal and the T-af groups of the values for each plane but there were no significant differences for any of the parameters.
The former was $126 \pm 12$ msec (mean ± SD) and the latter was $103 \pm 20$ msec (Fig. 1). The two values were statistically different ($p < 0.001$). The magnitudes of the maximal P vector in each plane are summarized in Table I. There were no significant differences in the frontal, the left sagittal and the horizontal planes between the 2 groups. However, the spatial magnitude of the P wave was significantly greater in the T-af group ($0.26 \pm 0.1$ mV) than that in the normal group ($0.15 \pm 0.06$ mV, $p < 0.001$) (Table II).

With respect to the configurations of the P loop in VCG, many patients in the T-af group had abnormal shapes, as shown in Fig. 2. In case 1, the P loop in the frontal and left sagittal planes had irregular bites. The P loop of case 2 showed abnormal bites and notches in the left sagittal plane and that of case 3 demonstrated similar findings in all three planes. The P loop of case 4 also showed abnormal notches in the frontal and left sagittal planes. Thus,

### Table II. The Spatial Magnitude Electrocardiogram (SMECG)

<table>
<thead>
<tr>
<th></th>
<th>SMECG = $\sqrt{X^2 + Y^2 + Z^2}$</th>
<th>mean ± SD (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal group</td>
<td></td>
<td>$0.15 \pm 0.06$</td>
</tr>
<tr>
<td>T-af group</td>
<td></td>
<td>$0.26 \pm 0.10^*$</td>
</tr>
</tbody>
</table>

* $p < 0.001$.

The top shows the formula of the SMECG and the bottom indicates the mean values from the normal and the T-af groups.

![Fig. 2. The P vector loops from 4 representative cases of the T-af group. F, S, H denote the frontal, the left sagittal and the horizontal planes, respectively. See further descriptions in the text.](image-url)
the P loops in 16 of 28 cases (57%) in the T-af group showed abnormal configurations such as 'bites' and 'notches'. Among these 28 patients, 10 experienced attacks of transient atrial fibrillation more frequently than once a week. Eight of these 10 cases showed abnormal configurations of the P loops.

In the spatial velocity electrocardiogram (SVECG), the numbers of peaks during the atrial excitation were similar to those in the normal group; 58% of cases in the T-af group showed three peaks and 42% had four peaks. In normal subjects, P waves have been shown to be composed of three or four peaks. The normal group in the present study also showed patterns similar to those in the previous study as well as to those in the T-af group in the present study. However, the maximum value of SVECG was greater in the T-af group than in the normal group. The values were 5.4±2.0 mV/sec in the former and 3.9±0.8 mV/sec in the latter (p<0.05, Fig. 3).

Body surface maps were recorded during sinus rhythm in 13 of 28 cases in the T-af group and the findings compared to those in the normal group. The map from the T-af patient in Fig. 4 shows the maximum initially appearing near the sternum and the minimum on the right shoulder. They stayed there for about 30 msec and their voltages increased over time. Then, the maximum moved to the left side of the chest and the anterior chest was covered with the negative potential area. At the later half of the P wave, the maximum moved further to the left and the minimum went down from the upper to the lower chest. These moving patterns of the maximum and the minimum are similar to those in normal subjects, as reported previously.

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**Fig. 3.** Spatial velocity electrocardiogram (SVECG). The top shows the formula to obtain the value of the spatial velocity electrocardiogram (SVECG). The middle shows typical records from the normal group (left) and from the T-af group (right). Note that the configurations of the P waves of both groups resemble each other, except for the larger maximal value in the latter than in the former. The maximal values of the SVECG from both groups are shown at the bottom.
The quantitative analysis of the maps using parameters described previously,\textsuperscript{12) however, gave different values in the 2 groups. The mean values in the normal group were as follows: The duration of the anterior maximum was \(32 \pm 11\) msec, that of the left maximum was \(38 \pm 6\) msec, and the shifting time from the

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure4}
\caption{The body surface maps of atrial excitation. The striped area indicates the area of positive potentials and the white one that of negative potentials. The symbols (+) and (−) indicate the maximum and the minimum, respectively. Isopotential lines are drawn at 50 \(\mu\)V intervals. Times after the onset of the P wave in lead II are shown at the left side of each picture. The maps are recorded from a 62 year old male patient having episodes of the T-af.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure5}
\caption{The analysis of the parameters from the P maps in the normal and T-af groups. On the abscissa, the time after the onset of the P wave in lead II is plotted. The mean durations of the anterior maximum (dotted box) and of the left maximum (black box), from the normal (top) and from the T-af groups (bottom) are shown.}
\end{figure}
anterior maximum to the left maximum was $51\pm12$ msec. In the T-af group, the duration of the anterior maximum and that of the left maximum in the T-af group were significantly prolonged compared to the values in the normal group ($p<0.05$ and $p<0.01$, respectively). The shifting time of the anterior maximum to the left maximum was $56\pm9$ msec. This value was not different from that in the normal group (Fig. 5).

The results of these various electrocardiographic examinations demonstrated that patients with T-af had abnormal findings in the high-speed, high-gain recordings of the XYZ leads, P loops of the VCG, SVECG, SMECG and in body surface maps.

**DISCUSSION**

The present study disclosed that P waves from patients having episodes of transient atrial fibrillation without apparent anatomical changes in the atria showed abnormal findings in VCG, spatial velocity and spatial magnitude ECG (SVECG and SMECG, respectively), and in the body surface maps during the episode-free period, although their standard 12 lead ECGs did not reveal any abnormalities. The abnormal findings are as follows: (1) Prolonged duration and increased amplitude of the P wave in the high-speed and the high-gain recordings of the orthogonal XYZ leads of Frank’s lead system (>120 msec). (2) Abnormal bites or notches in the P loops in the VCG. (3) Increased values of the P waves in the SVECG (>4.7 mV/sec) and SMECG (>0.156 mV) and (4) Prolongation of both the anterior and the left maximum in the body surface P maps.

The cases in our T-af group were selected to have no apparent anatomical changes in the atria. These selections were made by the exclusion of cases with valvular and congenital heart diseases and cases demonstrating enlarged left or right atrium on chest X-ray or on UCG. This may indicate that cases with small morphologic changes in the atria, which we could not detect by conventional diagnostic techniques, were included in the T-af group. Actually, our study included cases with mild hypertension and ischemic heart disease. Therefore, the present study was confined to those cases without abnormal atrial findings on chest X-ray and on UCG. They might have had either mild morphologic changes at the microscopic level or functional abnormalities which were reflected on the electrocardiogram.

Several reports indicated that those subjects whose P waves had durations longer than 120 msec experienced frequent attacks of paroxysmal atrial fibrillation.\(^5\),\(^6\),\(^13\)\(^-\)\(^15\) However, there have been no consistent findings regarding P wave abnormalities in patients with T-af who did not show any
sign of organic heart disease. While Davies and Ross found 20% of such patients demonstrating a prolongation of the P wave in lead II, others indicated either no abnormalities, or a normal P wave duration in lead II but prolongation of the P terminal force and total duration of the P wave in V1. In our study, we were able to demonstrate abnormal P wave findings, as mentioned above, using sophisticated electrocardiographic recording methods, although the conventional 12 lead ECG did not demonstrate any abnormality. Since most of our patients were lacking in gross anatomical changes of the atria, the existence of either functional disturbances or minor morphologic changes, which could not be detected by conventional diagnostic techniques, is suggested. No matter what the real basis of these abnormalities is, the present findings related to P waves in T-af patients can be obtained by noninvasive methods easily and repeatedly. Furthermore, these findings are characteristic of most of our T-af patients.

The wide P waves indicate prolongation of the entire atrial excitatory time. The abnormal bites and notches on the P loops represent the presence of irregular atrial excitatory sequences. Therefore, the above findings suggest the presence of intra-atrial conduction disturbances in this disease. The abnormalities were not confined to either the left or right atrium alone, but were seen in components of both atria equally, as judged by the findings in the P loops and the P maps. The increased maximal value of the SMECG suggests a rapid inscription of the P loops during the course of atrial excitation. This may also indicate the presence of uneven or nonhomogeneous propagation of the excitatory wave throughout the atrial systole. An increased value of the maximal spatial magnitude was also found in the T-af group. This finding may indicate an additional factor involved in this disease, since the higher value of the spatial magnitude in the SMECG represents an increase in the electromotive force of the atria.

Generally, an increase in the spatial magnitude is caused either by myocardial hypertrophy, by the Brody effect due to the enlargement of the intracavitary blood volume, by the approach of the heart to the thoracic wall or by intra- and inter-atrial conduction disturbances. Our cases in this study showed normal cardiac silhouettes on chest X-ray and no findings of enlarged atria on echocardiograms, although mild degrees of abnormality could not be excluded. However, it is indisputable that the increase in the spatial magnitude or SMECG or SMECG indicates hypertrophy or enlargement of the atria to some extent, which is not detectable from the chest X-ray or from the echocardiogram. Nevertheless, after due consideration of all of these findings, we may speculate that these findings are reflections of intra-atrial conduction disturbances rather than due to enlargement or hypertrophy.
of the atria. The results from the body surface P maps also indicate prolonged atrial excitatory times in both atria, and conduction disturbances are supposed to be present in both atria.

The present study did not disclose the precise mechanism of the genesis of atrial fibrillation in our patients, since we examined the P waves only during episode-free periods and did not study the period of T-af onset. We did not measure the conduction velocity, nor did we record the electrical activity directly from various parts of the atria. Nevertheless, our results, which were obtained clinically by noninvasive methods, suggest the presence of intra-atrial conduction disturbances predisposing to slowed or nonhomogeneous propagation and the possible development of one-way block in patients with this arrhythmia. These factors may contribute to the genesis of reentry which is an important mechanism of atrial fibrillation.19),20)

REFERENCES

15. Simpson RJ, Foster JR, Gettes LS: Atrial excitability and conduction in patients with in-

