Clinical Studies on the Nature of the Auricular Flutter

By

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It is widely known, that there are two different theories concerning the nature of the auricular flutter; namely the ectopic theory and circus movement theory. In the previous papers1,2 we attempted to clarify this problem experimentally and reported that the electrically induced auricular flutter is differed from the aconitine-induced flutter in their nature. The analysis of the intrinsic deflections in the direct auricular unipolar leads exhibited to us that in the former the excitation wave propagates in a circus way, while in the latter it starts at the aconitine-injected portion and propagates radially.

Accordingly it was considered that there are two different mechanisms in the experimental flutter which shows the identical figure.

Now it must be examined which of these states corresponds to the clinical flutter. In the literature, the circus movement theory of the auricular flutter was claimed at first by Lewis3 in 1921 on the basis of the fact that the electrical axis of the F wave rotates in 360 degrees. Later, Decherd and others4 and Grishman and others5 also observed the same findings. In recent years, Wenger and others6 and Sodi Pallares and others,7 analyzing the esophageal, intracavity or precordial leads, obtained the results explanable by the circus theory and claimed the existence of the flutter due to this mechanism.

On the other hand, Prinzmetal and others8,9 advocated the ectopic theory on the basis of the nearly synchronous contraction of both auricles in the high speed motion picture recorded during commissurotomy. They pointed out, furthermore, the excitation wave propagates in same direction
both in the esophageal leads and in the direct leads from the auricular surface.

Thus, each of these theories seems to have the firm base. Our purpose in this paper is to solve the problem in the mechanism of the clinical flutter.

**Experimental**

**Material and Methods**

Five cases of the auricular flutter administered in the Medical Clinic of Prof. Mikamo, University of Tokyo and in the Medical Clinic of Prof. Nakamura, Tohoku University were analyzed. The clinical findings, the effects of the treatments, etc. are summarized in Table I. The criteria for the diagnosis of the clinical flutter used by us are regularity and uniformity of the F wave with the restless undulation of the baseline. In addition to the usual three limb leads and the six precordial leads, the electrocardiograms were taken from the cavity of the right auricle, the coronary vein and the esophagus at the auricular levels. The former two kinds of leads were carried out by means of the venous catheterization. The modes of the conduction of the auricular excitation were analyzed by comparing the instants of the intrinsic deflections of the F wave in these leads with that of the II Lead recorded simultaneously by multichannel electrocardiograph. The leads from the esophagus and the coronary vein are considered to indicate the excitation of the

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Patient Age</th>
<th>Sex</th>
<th>Duration of symptoms</th>
<th>Frequency of attacks</th>
<th>Duration of the attack in observation</th>
<th>Aur. rate</th>
<th>Degree of A-V block</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>62</td>
<td>M</td>
<td>8 weeks</td>
<td>One attack</td>
<td>8 weeks</td>
<td>272</td>
<td>2:1</td>
</tr>
<tr>
<td>2</td>
<td>41</td>
<td>M</td>
<td>2 years</td>
<td>One attack</td>
<td>2 years</td>
<td>316</td>
<td>2:1 to 3:1</td>
</tr>
<tr>
<td>3</td>
<td>62</td>
<td>M</td>
<td>6 months</td>
<td>One attack</td>
<td>6 months</td>
<td>353</td>
<td>2:1</td>
</tr>
<tr>
<td>4</td>
<td>22</td>
<td>M</td>
<td>4 or 5 years</td>
<td>Several attacks</td>
<td>3 days</td>
<td>240</td>
<td>1:1 to 2:1</td>
</tr>
<tr>
<td>5</td>
<td>49</td>
<td>M</td>
<td>3 months</td>
<td>One attack</td>
<td>3 months</td>
<td>272</td>
<td>3:1</td>
</tr>
</tbody>
</table>

**Table**

Details of the
Results of Analysis

Case 1. The electrocardiogram shows the regular F wave of 0.22 sec. in length with the 2:1 auriculo-ventricular conduction, as shown in Fig. 1 and 2.

Analyzing the records, the time references of the intrinsic deflections were arranged in the order from cranial to caudal in the right auricular cavity leads, while from caudal to cranial in leads from the coronary vein and the esophagus, as shown in Fig. 3. The time interval between the intrinsic deflections of the lower and upper portions were estimated as 0.12 sec. in the right auricular cavity leads, 0.10 sec. in the coronary venous leads, and 0.13 sec. in the esophageal leads, respectively. These values correspond to nearly half of the cycle length of the F wave, and the instants of the intrinsic deflections cover the whole length of an auricular cycle. Therefore, it was concluded that the excitation wave in this case propagates in a circus way; from cranial to caudal in the anterior portion and from caudal to cranial in the posterior portion.

Case 2. The electrocardiogram shows the regular F wave, whose length is 0.19 sec. (Fig. 4). The time references of the intrinsic deflections are distributed in the order from cranial to caudal in the right auricular cavity leads and from caudal to cranial in the esophageal leads (Fig. 5).

I
Examined Cases

<table>
<thead>
<tr>
<th>Etiologic heart disease</th>
<th>Congest. heart failure</th>
<th>Ineffective treatment</th>
<th>Effective treatment</th>
<th>Result of treatment</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknown</td>
<td>+</td>
<td>Carotid sinus or occular pressure, oral digitalis</td>
<td>Quinidine</td>
<td>Sinus rhythm</td>
<td></td>
</tr>
<tr>
<td>Hypertensive sclerotic heart disease</td>
<td>+</td>
<td>Carotid sinus or occular pressure, Oxetyl, quinidine, digitalis</td>
<td>None</td>
<td>Flutter persists</td>
<td>Flutter persists over 5 years.</td>
</tr>
<tr>
<td>Hypertensive sclerotic heart disease</td>
<td></td>
<td>Quinidine, digitalis</td>
<td>None</td>
<td>Flutter persists</td>
<td>Flutter persists over 3 years.</td>
</tr>
<tr>
<td>Unknown</td>
<td>+</td>
<td>Occular pressure</td>
<td>Digitalis, quinidine</td>
<td>Occasional recurrences of flutter attack</td>
<td></td>
</tr>
<tr>
<td>Hypertensive sclerotic heart disease</td>
<td></td>
<td>Occular pressure</td>
<td>Digitalis</td>
<td>Nodal rhythm to sinus rhythm</td>
<td>Occasional recurrences of flutter</td>
</tr>
</tbody>
</table>
Fig. 1. Case 1. Auricular flutter with 2:1 A-V conduction. Auricular rate, 272 per minute; ventricular rate, 136 per minute.

Fig. 2. Case 1. Curves taken from the coronary vein, right auricular cavity and the esophagus, showing the time references of the intrinsicoid deflections in each leads on the left side. The time signal is 0.02 sec.

Fig. 3. Case 1. A diagram showing the readings for arrival of the excitation wave. In the right auricular cavity leads the excitation wave propagates from upper to lower and in the esophageal leads and the coronary venous leads from lower to upper. This indicates the circus motion. Auricular cycle length, 0.214 sec.
On the Nature of Auricular Flutter

Fig. 4. Case 2. Auricular flutter with 2:1 A-V conduction. Auricular rate, 316 per minute; ventricular rate, 158 per minute.

The time interval between the upper and lower parts were estimated as 0.08 sec. in the esophageal leads and 0.10 sec. in the auricular cavity leads. These values correspond to nearly half of an auricular cycle length. Furthermore, the instants of the intrinsic deflections cover the whole cycle length of an auricular excitation, as shown in the former case. It was concluded, therefore, that the excitation wave propagates in a circus way.

Case 3. The electrocardiogram showed the regular F wave of 0.17 sec. in length (Fig. 6). In the right auricular cavity leads, it was observed that the excitation wave propagates from lower to upper as shown in Fig. 6 and 7. The instants of their intrinsic deflections are distributed over the interval of 0.10 sec. It corresponds to more than half of the F wave. The esophageal leads failed to explore the auricular excitation in this case. Refering to the experimental findings in our previous paper and the observations in the above mentioned cases, the excitation wave was presumed to propagate also in a circus way.

Case 4. A patient suffering from attacks of arrhythmias and brady-cardia of unknown etiology since childhood. The previous electrocardiograms showed sometimes the sinoauricular block and sometimes the nodal rhythm. Recently, an attack of the tachycardia as shown in Fig. 8 occurred. This electrocardiogram was at first considered to be the ventricular tachycardia, but after the oppression of the eye-balls, the ventricular rate decreased suddenly and the characteristic F waves became visible in the electrocardiogram, as shown in Fig. 9.

The analysis was carried out by the esophageal and auricular cavity leads recorded simultaneously, and it was observed that the excitation wave propagates from caudal to cranial in the esophageal leads spending 0.12 sec. and from cranial to caudal in the right cavity leads spending 0.13 sec. Therefore the nature of the tachycardia was concluded to be the auricular flutter due to the circus movement also in this case.

Case 5. A patient suffering from mild attacks of angina pectoris.
Fig. 5. Case 2. A diagram showing the readings for arrival of the excitation wave, which propagates from cranial to caudal in the right auricular cavity leads, while in the esophageal leads from caudal to cranial. Auricular cycle length, 0.20 sec.

Fig. 6. Case 3. The II, right auricular cavity and esophageal leads. Auricular flutter with 2:1 A-V conduction. Auricular rate, 353 per minute.

Fig. 7. Case 3. A diagram of the cephalic surface of the auricles, showing the time references of the intrinsic deflections. The excitation wave propagates from lower to upper in the right auricle. The interval between the earliest and the latest intrinsic deflections is 0.10 sec.

In the electrocardiograms the auricular flutter with the regular F wave of 0.22 sec. in length was observed (Fig. 10). Analysis of the esophageal, right auricular cavity and coronary venous leads showed that the excitation wave propagated upwards from lower in every leads (Fig. 11 and 12).
Fig. 8. Case 4, before the ocular pressure. Auricular flutter with 1:1 ventricular response. Auricular complexes are indiscernible, and ventricular complexes seem to be the right bundle branch block. Auricular as well as ventricular rate, 240 per minute.

Fig. 9. Case 4, after the ocular pressure. Auricular flutter with 3:1 to 4:1 conduction, showing the characteristic saw-tooth appearance of the auricular complexes. Auricular rate, 300 per minute.

The time interval between the earliest and the latest intrinsic deflections was estimated as 0.06 sec. This value is very short in comparison with the values observed in former four cases as well as with auricular cycle length in this case. These findings indicate that the excitation wave starts at the lower portion and propagates radially over the whole auricular muscle within a relatively short period. It is similar to the experimental aconitine-induced flutter.

From these points the auricular tachycardia in this case was concluded as a flutter due to the ectopic rhythm.

Fig. 10. Case 5. Auricular flutter with 3:1 A-V conduction, showing P' wave of Prinzmetal. Auricular rate, 272 per minute.

DISCUSSION

The above mentioned analysis exhibits that there are two kinds of
Fig. 11. Case 5. The esophageal leads (A), the coronary venous and right auricular cavity leads (B), showing the time references in each leads on the left side.

Fig. 12. Case 5. A diagram showing the readings of the intrinsic deflections. The excitation wave propagates from lower to upper in every leads. This indicates the ectopic rhythm arising in the lower portion of the auricles. Auricular cycle length, 0.22 sec.

mechanism in the clinical auricular flutter; in four cases the circus motion of the excitation wave was established and in one case the auricular ectopic rhythm. The findings in the former coincide with those in the experimental auricular flutter induced by the electric stimulation, while those in case 5 with those in aconitine-induced flutter, which were reported in our previous papers.1) Among these cases the circus motion in case 3 was presumed by the observation in the right auricular cavity leads alone because of the miscarriage of the esophageal leads, but the distribution of the intrinsic deflections in these leads was similar to those of other cases due to the circus motion.
The differences between these two kinds of mechanism observed in our cases lie in the followings: 1) In the case of the ectopic rhythm the order of the intrinsic deflections are same in various leads and in the case of the circus motion they are reversed in the right auricular cavity lead vs. the esophageal or coronary venous leads. 2) The time interval between the earliest and the latest intrinsic deflections in each lead is shorter in the ectopic rhythm and longer in the circus motion. 3) In the former the intrinsic deflections are distributed within less than half of an auricular cycle length, while in the latter they cover a whole length.

According to Prinzmetal's opinion, if the circus motion exists, the esophageal leads at the mid-auricular level should exhibit two intrinsicoid deflections by the reflections of the two-directional excitatory propagation during an auricular period. Because of the lack of such findings, he denied the circus motion. In our opinion, however, since the esophagus lies rather near the left auricle, the left auricle alone may produce the intrinsicoid deflection in this lead and that of the right auricle may not be recognized clearly. Another base of his argument to support the ectopic theory is the fact that the conduction time between the lower and upper portions in the esophageal leads is very short in comparison to the auricular cycle length. Such a finding was observed in our case 5. In other four cases, however, the conduction time is much greater.

The disputation between the circus and ectopic theories, which has been argued for many years, has now arrived to the conclusion that each of the theories are correct and the two different kinds of mechanism present the identical figures in the clinical and electrocardiographic aspects.

The differentiation of the auricular flutter due to the circus motion from that due to the ectopic automatism. It must be necessary to find the signs to differentiate two kinds of the flutter in routine electrocardiograms. Although no differences can be discovered in the clinical findings and the effects of the treatments in our five cases, as shown in Table I, there are some differences in the electrocardiographic findings.

In the first, observing carefully the saw-tooth-like oscillation in the II, III, VF and VI Leads, which are considered as the characteristics of the flutter wave, we might find the downward or upward narrow deflections in them, which correspond to Prinzmetal's P' wave. Since the VF Lead may reflect mainly the excitation of the lower part of the auricles and the VI Lead mainly the excitation of the middle or upper parts of the right auricle, the longer interval between these deflections in both leads recorded simultaneously suggests the circus motion. Case 1. in which this value is estimated as 0.10 sec., may confirm this relations (Fig. 13). On the other hand, if the instants of these deflections are fairly synchronous as in Case 5 (Fig. 14), the excitation wave is supposed to start at one point
and reach both auricles nearly at the same time. But these relations are not always definite in all cases.

Secondly, our case 5, which is attributed to the ectopic rhythm, shows the sharp and narrow deflections within the F wave in the II and III Leads. This is not so definite in other four cases.

![Fig. 13. Case 1. The onset of the intrinscoid deflection in V1 preceds the P' wave in V_F by 0.12 sec.]

![Fig. 14. Case 5. The onset of the intrinscoid deflection in V1 succeeds to the P' wave in V_F by 0.03 sec.]

**SUMMARY**

1. In five cases of the auricular flutter, the circus motion of the excitation wave was established in four and the auricular ectopic automatism in one.

2. It was concluded that there are two kinds of mechanism in the clinical auricular flutter, as in the experimental observations. This conclusion solves the long lasting disputation between two theories.

3. Some considerations on the differences in the routine electrocardiograms of these two kinds of the flutter were described.

**References**


3) Lewis, Drury & Iliescu, Heart, 1921, 8, 341.
