The Experimental Study of Normal Atrial T Wave (Ta) in Electrocardiograms

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Summary

(1) P wave and Ta wave of 35 open-chest dogs were recorded in high amplitude (100 μV. = 3 cm.) after production of A-V block, and normal patterns of the waves were studied. Direct current amplifier which yielded no untoward deformity of P wave or Ta wave was used.

(2) P wave and Ta wave were always opposite in direction and the area of both waves was almost equal, i.e. atrial gradient was nearly zero. Consequently it is considered that the activation process and recovery process of atrial excitation have the same direction, and furthermore, the time sequence of both processes is very similar.

(3) There was good correlation between the amplitude of P wave and Ta wave. \[ Ta = 0.21 \times P + 3 \times (μV.) \], i.e., the amplitude of Ta wave is about 24% of that of P wave.

(4) Duration of Ta wave was 2.4 times that of P wave on the average.

(5) There was good correlation between atrial rate (P-P interval) and P+Ta time. \[ P+Ta = 0.31 \times (P-P) + 77 \times (\text{msec.}) \]

(6) Ta wave extends to ST segment over QRS complex for 0.06 msec. on the average. Consequently, determination of PR segment or ST segment deviation, especially in their depression must carefully be made. When the depression of PR segment or ST segment does not exceed the amplitude of Ta wave expected from that of P wave, the depression might safely be considered to be the influence of Ta wave. When the ST depression exceeds the amplitude of Ta wave expected from that of P wave, it may be the real ST depression.

(7) ST segment does not return to the isoelectric line completely even in normal condition, but deviates in the opposite direction of P wave by the influence of Ta wave.

Additional Indexing Words:
High fidelity direct current amplifier A-V block Atrial gradient Activation process and recovery process of atrial excitation False ST depression Real ST depression Atrial premature beat Atrial infarction

In the electrocardiogram the ventricular complex consists of QRS wave and T wave, which represent the activation process and the recovery process of the electrical excitation in the ventricle respectively.
It is well known that P wave represents the activation process of the excitation of the auricle or, in the electrophysiologic sense, it indicates the depolarization of the muscle.

On the other hand, the deflection in the auricular complex which corresponds to the T wave of the ventricular complex is called Ta wave (atrial T wave). Ta wave represents the recovery process of the excitation of the auricle or, in the electrophysiologic sense, the repolarization that follows the depolarization of the muscle.

But Ta wave is generally hard to recognize because its deflection is very small and it coincides with the phase of the activation of the ventricular muscle and so it merges with graphic elements of the ventricular complex. Consequently, Ta wave has not attracted much attention in the electrocardiographic study till recent years.

But Ta wave is very important in the diagnosis of auricular infarction. Furthermore, because it may cause the false depression of PQ segment, J point or ST segment, there is a certain danger of leading to an erroneous diagnosis of coronary insufficiency or myocardial damage.

Accordingly, the clinical significance of Ta wave has been paid special attention in recent years.

In the present study, P wave and Ta wave could be recorded in large amplitude, using high fidelity direct current amplifier which did not make untoward deformity of these waves. The author reports the systemic investigation of normal Ta wave and touches on the electrophysiological and clinical significances of Ta wave.

### METHODS

Experiments were performed on 35 mongrel adult dogs which ranged in weight from 11 to 15 Kg. They were anesthetized with pentobarbital sodium (Nembutal) 30 mg./Kg. intravenously and under artificial respiration the chest was opened. The skin and muscle over the right 4th intercostal space were incised along the rib and the sternal edge of the 4th and 5th ribs were separated. The pericardial sac was incised for about 6 cm. along the long heart axis, keeping the sac fixed to the thoracic wall, so it appeared like a cradle of the heart.

A-V block was made by the ligation of the atrioventricular node artery at the region of the "U"-turn of right coronary artery in the posterior portion, or in some cases by destruction of A-V node with the needle inserted from the posterior right atrium. The heart was immediately replaced in the pericardial sac, the separated ribs being fixed, and the chest was closed. Under this condition, standard limb leads, unipolar limb leads, chest lead (V_{1}) and esophageal lead were recorded within a few sec. after respiration was halted. The employed amplifier was a biophysical ME Amplifier BE-All (Yokokawa Electric Co. Ltd.) and this was connected to EMO 62, 6-channel photocorder. High cut filter of 100 cps was used and the
recording was done at the paper speed of 10 cm./sec. The electrocardiogram was taken at the sensitivity of 100 \( \mu \text{V.} = 3 \text{ cm.} \) in standard and unipolar limb leads and chest lead, 100 \( \mu \text{V.} = 1.5 \text{ cm.} \) in esophageal lead. A bipolar electrode was used for atrial premature beat. The stimuli, a square wave current of 4 msec. in duration and 4 to 7 V. in strength, was given after the refractory period of normal sinus rhythm was over. The site of the stimulation was the coronary sinus region. In measuring the area of P wave and Ta wave, the waves which were traced on the paper were cut out and weighed by the direct-reading micro-balance. The "weight" of each wave was converted into the area.

**Results**

Typical normal patterns of P wave and Ta wave are shown in Fig. 1 and 2. To record P wave as prominently as possible, QRS complexes and T waves were in part scaled out. The upper row shows P wave, QRS complex and T wave before A-V block was produced. The lower row shows P wave and Ta wave after the production of A-V block.

P wave deflections were always positive in leads I, II, III, and aVF, and negative in aVR, aVL and V1. In esophageal lead, P wave deflection changed to positive or negative, according to the distance of the tip of electrode from the teeth.

In every lead, P wave was followed by a "dome-shaped" Ta wave distinctly. Ta wave deflections were always negative in leads I, II, III and aVF, and positive in aVR, aVL, and V1, namely P wave and Ta wave were in-

![Figure 1](image-url)
variably opposite in direction.

In esophageal lead, Ta wave changed to negative or positive according to the distance of the tip of electrode from the teeth. By applying artificial stimulation to the coronary sinus region, a positive P wave in lead II turned to negative with the Ta wave change from negative to positive (Fig. 3).

Fig. 2. Upper row shows P, QRS, T wave before A-V block was produced. Lower row shows P, Ta wave after A-V block was produced.

Fig. 3. Positive P and negative Ta wave changed to negative P and positive Ta wave by applying artificial stimulation to coronary sinus region in lead II.
Fig 4. The distribution of the amplitude of P wave and Ta waves in each lead.

Table I. The Amplitude of P and Ta Waves and Ta/P in μV.

<table>
<thead>
<tr>
<th>Lead</th>
<th>P</th>
<th>Ta</th>
<th>Ta/P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>S.D.</td>
<td>Max.</td>
<td>Min.</td>
</tr>
<tr>
<td>I</td>
<td>111</td>
<td>37</td>
<td>178</td>
</tr>
<tr>
<td>II</td>
<td>286</td>
<td>72</td>
<td>392</td>
</tr>
<tr>
<td>III</td>
<td>231</td>
<td>76</td>
<td>343</td>
</tr>
<tr>
<td>aVR</td>
<td>124</td>
<td>29</td>
<td>160</td>
</tr>
<tr>
<td>aVL</td>
<td>77</td>
<td>23</td>
<td>110</td>
</tr>
<tr>
<td>aVF</td>
<td>178</td>
<td>43</td>
<td>251</td>
</tr>
<tr>
<td>V1</td>
<td>83</td>
<td>14</td>
<td>108</td>
</tr>
<tr>
<td>Es0</td>
<td>311</td>
<td>87</td>
<td>430</td>
</tr>
</tbody>
</table>
Fig. 4 shows the distribution of the amplitude of P wave and Ta wave in each lead. The mean amplitude of Ta wave was 27 μV. in lead I, 60 μV. in lead II, 54 μV. in lead III, 26 μV. in aVR, 20 μV. in aVL, 33 μV. in aVF, 27 μV. in V1 and 78 μV. in esophageal lead. The mean amplitude of P wave ranged from 77 μV. to 311 μV. according to leads (Table I). An obvious positive correlation was observed between the amplitude of P wave and Ta wave in every lead (Fig. 5). It was given by the equation:

\[ Ta = 0.21P + 3 (\mu V) \]

and correlation coefficient was 0.85.

The ratio of amplitude of Ta wave and P wave (Ta/P) was 0.24 on the average. Fig. 6 shows the distribution of the duration of P wave and Ta wave in each lead. In measuring the duration of P wave and Ta wave, the points at which the isoelectric line crosses them were arbitrarily determined as the initiation and the termination of each wave.

The mean duration of Ta wave was 136 msec. in lead I, 146 msec. in lead II, 148 msec. in lead III, 150 msec. in aVR, 152 msec. in aVL, 148 msec. in aVF, 151 msec. in V1 and 145 msec. in esophageal lead. The mean duration of P wave ranged from 56 msec. to 68 msec. according to leads (Table II).

The ratio of the duration of Ta wave and P wave (Ta/P) was 2.4 on the average. (P+Ta) time ranged from 140 msec. to 300 msec. and the mean value was 210 msec. In every lead there was good correlation between the atrial rate, P-P interval, and P+Ta time, which represents the electrical duration of atrial excitation (Fig. 7).

The increase of P-P interval, i.e., the decrease of atrial rate, brought
about the increase of P+Ta time. It was given by the equation,

$$P + Ta = 0.31(P-P) + 77 \text{ (msec.)}$$

and the correlation coefficient was 0.82.

The areas of P wave and Ta wave were measured in every lead, after
determining that the upward deflection was positive and the downward deflection was negative, and the algebraic sum of the two waves in each lead, which represents the component of atrial gradient, was calculated.

The area of P wave ranged from 2.0 µV. sec. to 8.7 µV. sec. and that of Ta wave ranged from 2.2 µV. sec. to 9.4 µV. sec. and the component of atrial gradient ranged from −0.7 µV. sec. to 1.5 µV. sec. according to leads (Table III). In Fig. 8 is seen an illustrates of the areas of P wave and Ta wave and the component of the atrial gradient in each lead. P wave and Ta wave in the atrial premature beat were also opposite in direction and the area of the two waves was almost equal. Accordingly, the atrial gradient in the atrial premature beat was also nearly zero as in normal sinus rhythm. On the other hand, by the production of atrial infarction with cautereization, the transitional

| Table III. The Area of P and Ta Waves and the Component of Atrial Gradient in µV. sec. |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Lead  | P area (µV. sec.) | Ta area (µV. sec.) | Atrial Gradient Component |
| I     | 2.7  | 0.9  | 4.8  | 1.4  | −2.5 | 0.7  | −1.2 | −3.7 | 0.2  | 0.6  | 1.1  | −0.7 |
| II    | 8.4  | 2.7  | 12.6 | 5.6  | −6.9 | 1.5  | −5.4 | −9.1 | 1.5  | 1.3  | 3.5  | 0    |
| III   | 5.7  | 1.5  | 7.4  | 3.7  | −5.1 | 1.4  | −3.4 | −6.7 | 0.6  | 0.5  | 0.7  | 0.3  |
| aVr   | −2.9 | 1.1  | −0.9 | −4.1 | 2.8  | 1.2  | 4.4  | 1.1  | −0.1 | 0.5  | 0.4  | −0.8 |
| aVl   | −2.2 | 0.4  | −1.7 | −2.7 | 2.2  | 0.4  | 2.8  | 1.7  | 0    | 0.5  | 0.8  | −0.5 |
| aVP   | 5.0  | 1.4  | 6.4  | 3.6  | −4.5 | 1.0  | −2.7 | −5.6 | 0.5  | 0.7  | 1.5  | −0.7 |
| V1    | −2.0 | 0.8  | −1.3 | −3.7 | 2.5  | 0.9  | 4.2  | 1.7  | 0.5  | 0.2  | 0.8  | 0.1  |
| Eso   | 8.7  | 2.1  | 12.2 | 4.6  | −9.4 | 3.4  | −5.2 | −14.8| −0.7 | 0.4  | 1.7  | −4.4 |
Fig. 8. The area of P and Ta waves and the component of the atrial gradient in each lead.

Fig. 9. Upper row shows normal P and Ta waves. Lower row shows the change of P and Ta waves after the production of atrial infarction.
portion of P wave and Ta wave became markedly elevated when P wave was positive and markedly depressed when P wave was negative in some leads.

Consequently, differentiation of P wave and Ta wave became difficult as Ta wave became very small or disappeared (Fig. 9). As a result, there appeared a large atrial gradient.

**DISCUSSION**

It has long been known that the P wave recorded by the ordinary electrocardiogram did not represent all electrical changes of the atrium. Kraus and Nicolai\(^1\) (1907), Samojloff\(^2\) (1909) and Straub\(^3\) (1910) described the “after-wave” following P wave. Hering\(^4\) (1912) was the first to prove that it was a part of atrial electrical activation. He designated the wave as Ta wave and considered it to correspond to T wave in ventricular complex. Thereafter, investigations on Ta waves were performed in isolated heart preparations from various animals.

Since the studies by Abramson et al.\(^5\) (1938), electrical phenomenon of the atrium, especially atrial repolarization, has attracted the attention of several investigators. In recent years, Ta wave was studied clinically by Wasserburger,\(^6,7\) Gross,\(^8\) Berkun,\(^9,10\) and Tranchesi.\(^11\) But the deflection of Ta wave was very small in these investigations, because the recordings were done by ordinary electrocardiogram. Accordingly, it might have been that those data lacked accuracy and its analysis was insufficient.

In the present study, P wave and Ta wave were recorded in very large amplitude without untoward deformity using a direct current amplifier and highly reliable results were obtained. The direct current amplifier in which a semiconductor chopper was included had very high sensitivity, with drifting of baseline and noise being small, and as the time constant was infinite, it yielded no deformity of the waves.

Preliminary study showed that P wave and Ta wave recorded either with a high cut filter of 100 cps, or without a filter, scarcely changed in form or amplitude. So a high cut filter of 100 cps, was used to minimize the intervention of the electromyogram or other untoward noise. To study the influence of thoracotomy upon P wave, electrocardiograms before and after the thoracotomy were compared. P wave scarcely changed in any lead before and after the procedure, in amplitude duration, as well as polarity and form. In addition, the P wave hardly changed at all before and after the manipulation for the production of A-V block. Consequently, Ta wave was also considered to be recorded under almost normal conditions. The reason why P wave did not change might be attributed to the following factors:
As the atrium is small and is situated deeply in the pericardial sac, it is scarcely influenced by various changes such as temperature, intrathoracic pressure, positional relationship between heart and lung, air volume in the lung and heart position.

Besides in opening the chest, only 2 ribs at sternal edge were separated. This kept the deformity of thoracic cage to a minimum and was also considered to do much to the constancy of P wave. Soon after the steep deflection of P wave, Ta wave was slowly described in opposite direction. Also, when various regions of the atrium were stimulated artificially, P wave and Ta wave were always described in opposite direction.

In ventricular complex QRS wave and T wave are usually described in the same direction. This is explained by the following mechanism. Though the activation process of the ventricular muscle spreads from inside to outside, the recovery process spreads from outside to inside, because of the pressure gradient and/or the difference of temperature between inside and outside of the ventricular cavity.

We demonstrated that in the atrium, P wave and Ta wave have the opposite direction as in single cardiac muscle fiber. Consequently, it should be considered that in the atrium, activation process (depolarization) and recovery process (repolarization) have a similar direction. This is attributed to the fact that the intra-atrial pressure is not so high as that of intra-ventricular pressure and as the atrial muscle is very thin, there is almost no difference of temperature between the inside and outside of the atrial cavity.

The author demonstrated that the area of P wave and Ta wave was almost equal in every lead, and the atrial gradient was nearly zero. Since Wilson advocated the concept of ventricular gradient in 1931, many investigators studied this problem qualitatively and quantitatively, and this concept has been applied to the atrium. Thereafter, Lepeschkin and Berkun reported that P wave and Ta wave were opposite in direction and the area of each wave was almost equal, the atrial gradient being nearly zero. But in the former studies, both P wave and Ta wave were very small in amplitude, and there was a question about its accuracy.

In the present study, P wave and Ta wave were recorded in large amplitude and very reliable results were obtained. The results should be considered to show that the time sequence of the activation process of atrium is very like that of the recovery process or they are completely the same. Also in atrial premature beat, the atrial gradient proved to be nearly zero, suggesting there was no electrophysiological difference concerning the direction of the activation process and recovery process between atrial premature beat and sinus rhythm.
Now, in normal condition there did not exist P-Ta segment between P wave and Ta wave as Abramson\(^5\) and Tranchesi\(^11\) described. This may be attributed to the fact that the duration of action potential of atrium is short compared to that of ventricle and the "plateau" of membrane action potential is almost none or, if any, very short. The electric duration of ventricular excitation is represented by Q-T interval, and it corresponds to P + Ta time in the auricle.

In the present paper it was shown that P + Ta time ranged from 0.14 sec. to 0.30 sec., mean value being 0.21 sec., in dogs. As PR interval is 0.10 sec.\(^15\) and the duration of QRS complex is 0.05 sec.,\(^15\) P + Ta time, i.e., electric duration of atrial excitation, far exceeds PR segment or QRS complex and has influence on ST segment for 0.06 sec. on the average.

In clinical electrocardiogram, depression of PR segment under isoelectric line is often encountered. It is usually found when the amplitude of P wave is large. This fact may suggest that the depression of PR segment is attributed to the increased amplitude of Ta wave accompanied by the enlarged P wave. From the linear relationship between the amplitude of P wave and Ta wave, which was obtained in the present study, the normal amplitude of Ta wave can be nearly estimated from that of P wave. If the depression of PR segment exceeds the normal amplitude of Ta wave, we might be able to find a clue to Ta wave abnormality; that is, some pathological changes of recovery process of atrial excitation. As shown above, Ta wave has influence upon the ST segment as well as the J point of ventricular complexes. This might cause false ST depression and it might be likely to lead to an erroneous diagnosis of an ischemic change of the myocardium. Even if there exists a ST depression, when it does not exceed the amplitude of Ta wave expected from that of P wave, the ST depression is considered to be nearly the influence of Ta wave. When the depression exceeds the amplitude of normal Ta wave, it is thought as a real ST depression. The net amplitude of the depression derived from the ischemic change of the ventricular muscle must be deduced by the subtraction of the amplitude of Ta wave. But even if the depression of ST segment exceeds the amplitude of normal Ta wave, it can not be completely denied that it is the influence of abnormally enlarged Ta wave caused by some pathological conditions of the recovery process of atrial excitation in atrium.

As shown above, Ta wave extends to the ST segment for 0.06 sec. on the average, that is about half the duration of Ta wave. Consequently, even in normal conditions, ST segment does not return to the isoelectric line completely, but deviates downward or upward by the influence of negative Ta wave or positive Ta wave.

As the normal P wave is very small and Ta wave is almost unrecognizable
ATRIAL T WAVE OF ELECTROCARDIOGRAM

in clinical electrocardiogram, the ST segment seems as if it returns to the isoelectric line completely.

Only when the P wave is described in large amplitude, the influence of Ta wave becomes clear and the dome-shaped depression of ST segment appears. But ST segment is seen to deviate invariably to one degree or another, yet always in the opposite direction to the P wave deflection. It is very reasonable that the American Heart Association made much of the problem and, to avoid the influence of Ta wave, recommended the use of the junction of PR segment and QRS complex as a base-line when estimating ST deviation. Studies under way show marked changes in some leads in atrial infarction. Such specific changes are the subject of a subsequent paper.

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