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Experimental Studies on the Auricular Waves.

By

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INTRODUCTION

Since Keith and Flack¹⁾ studied the anatomic structure of the sinus node for the first time in 1907, many reports concerning the propagation of the activation wave in the auricular myocardium and the activity of the sinus node as the pace maker of the cardiac cycle have been written on the mammalian heart. Wybauw²⁾ studied the electrical activity of the sinus node. He, using the bipolar lead electrocardiogram, demonstrated on canine hearts the region of initial negativity of the auricular surface. Lewis^{3,4,5)} and Eyster & Meek^{6,7,8)} assumed that the activation of the auricular muscle originated from this region. This region of initial negativity is the same region where Keith and Flack first located a sinus node anatomically. Lewis was of the opinion that this point of initial negativity coincided with the thickest portion of the sinus node of the canine heart. Lewis⁹⁾ concluded in his investigation that the activation wave proceeds from the sinus node in a radial manner, at an average rate of 1000mm/sec. They did not distinguish any specific route of impulse conduction in the auricle. His records were obtained by means of distant bipolar lead electrocardiogram (five to eight millimeters of electrodes distance), through which it was extremely difficult to determine the precise moment of arrival of the activation wave to the muscles in contact with the electrode. In contrast, Rothberger & Scherf¹⁰⁾ and Condrelli¹¹⁾ proved anatomically and functionally three specific systems for the conduction of impulses in the canine auricles. Condrelli¹²⁾ recorded experimentally the electrocardiogram from canine auricles, in which one auricle showed the auricular fibrillation and the other auricle showed no auricular fibrillation. Laufer¹³⁾, Maekawa & Takayasu¹⁴⁾ also reported clinical cases in which the con-

duction disturbance of activation waves occurred between both auricles. They reported a case in which the right atrium showed a normal sinus rhythm, while the left atrium showed simultaneous flutter. According to Condrelli¹¹⁾, the sinus node is located on the auricular portion near the vena cava, i. e., upward, and posterior portion of the right atrium, and has three specific route for the conduction of impulse. One of them is connected with A-V node via the Torus Loweri and the atrial septum. One of the remaining two is extended into the left atrium via Bachmann's bundle. The last one is spread immediately into the right atrium. In 1937 Takayasu^{15,16,17)} analyzed the splitting of the normal P wave, mitral P wave, and pulmonary P wave of the electrocardiogram recorded from the human heart by means of the cathode ray oscillograph. He confirmed from the findings of the activation time at various points of both auricles, that there are three specific tracts for the conduction of stimuli in the auricles. In 1951 he¹⁸⁾ measured the arrival time of the activation wave at each lead-point. This was done by means of a direct lead unipolar electrocardiogram. In 1952 and 1953, he^{19,20)} studied the changes in conductivity after ligation of the Bachmann's bundle, or after cutting several localized portions of the atrial septum of the canine heart. On the other hand, Sodi-Pallares²¹⁾ conducted research on the activation time, direction of the propagation of the activation, and the features of the unipolar direct lead electrocardiogram at each point of the auricular surfaces, and the interatrial septal surface of the canine heart. He concluded that the specific ways of conduction were not recognized, but that there were two regions of the intracaval and interauricular band zone where the stimuli were easily transmitted. In 1955, Takayasu²²⁾ concluded from his experimental results examining

the activation time at each leadpoint of auricular surface, before and after ligation of the Bachmann's bundle, and after cutting the atrial septum, that the activation wave in the atrium did not proceed in a radial manner as advocated by Lewis⁹⁾, but propagated as Condrelli¹¹⁾ suggested by three specific route of conduction, i. e., sino-Tawara, sino-left, and sino-right conduction systems. These findings were also examined by various histological researches. In the experiments discussed in this thesis, research on activation time was conducted by means of a special contiguous bipolar electrode (Fig. 1) which was inserted into the canine auricle.

The following items were given special consideration in the above mentioned experiment.

(1) The activation time, the direction of the propagation of the activation, and the features of unipolar direct lead auricular electrocardiograms of the normal canine hearts.

(2) The changes in arrival time of the activation wave at each lead point after ligation or cauterization of Bachmann's bundle, lower part of Taenia terminalis, and cava Venous appendage angle.

(3) Process of the auricular activation wave in artificial extrasystole provoked in left auricular appendage by inductorium stimulation.

PART I. The activation time, the direction of the propagation of the activation, and the features of unipolar direct lead auricular electrocardiograms of normal canine hearts.

MATERIAL AND METHOD

The present study was done on fifteen mongrel dogs, well nourished and weighing about twelve to seventeen kilograms. The anesthetic used was 10% oltopan-sodium introduced by intravenous injection, at a dose of 0.3cc per kilogram of body weight.

Technique: The chest was opened under artificial respiration. The heart was exposed by amputating the sternum and second to fourth ribs in contact with sternum at approximately four centimeters length at their sternal ends. The pericardial sac was opened lengthwise at two regions, one region is near the right atrium and the other region is over the left atrium. The operation field was covered with gauze soaked in Ringer's solution after electrodes were inserted into the auricular wall and fixed.

Anatomic control: As soon as the heart was exposed, the outline of the atriums was sketched and the points explored were marked in sequence on this sketch. In order to control the location of the electrodes, they were left in their place until the completion of the experiment.

Electrodes: The unipolar electrodes and contiguous bipolar lead electrodes were of special design (Fig. 1). In the present study, the unipolar lead

Fig. 1 Illustrates the contiguous bipolar electrodes and their arrangements to the auricular wall. Each head of the electrode is made of a small hook soldered. The distance between the electrodes is 1.0 mm.

from the region near the sinus node was used as a reference lead. The contiguous bipolar lead electrodes consisted of two very fine insulated copper wires of one hundred μ in diameter. These were equipped with a small hook soldered at each tip. These two wires were held in injection needles through their respective holes as illustrated in Fig. 1, and were then inserted into the auricular cavity through the atrial wall. The small hook soldered at the tip of each wire was then fixed on the endocardial surface after the injection needles had been withdrawn. The diameter of the needle was 1/4 mm. The author calls an electrode of this type the contiguous bipolar lead electrode for endocardial experiments. One of these electrodes was available as an electrode for unipolar lead use. The indifferent electrode for unipolar lead was connected to Wilson's central terminal.

Recording method: The electrodes fixed on the endocardium were connected to the lead selector in order to pick up a voluntary lead. Electrocardiograms were registered on the oscillograph paper by means of Sanyei's three channel electro-cardiograph at a speed of 20 cm. per second. A signal

was given at the top of the film at one hundred and twenty millisecond intervals, and beneath it three electrocardiograms, contiguous bipolar lead and unipolar lead electrocardiograms, were recorded simultaneously. In the contiguous bipolar lead electrocardiogram, the sharp deflection was recorded in accordance with the activation of the muscle in contact with the electrodes. Accordingly the activation time of the muscle region in contact with the electrodes was demonstrated sharply by the peak of this deflection. According to Lewis and Wilson, it was considered that a double layer was induced at the margin of the resting muscles and muscles activated, and if the activation front arrived at both electrodes at the same time, the

potential difference would not appear at that instance. In such a case, the results obtained are impossible or very difficult to analyse. Due to this difficulty, electrodes for the contiguous bipolar lead were set up in such a way that both electrodes were situated along the longitudinal line of the auricle or parallel with the muscle fiber. The electrocardiograms were recorded after vanishing the injury current which had been induced by insertion of the electrode. It was approximately five minutes for the diminution of the injury current. One of the electrocardiograms recorded was shown in Fig. 2. The contiguous bipolar lead electrocardiogram showed a very sharp deflection and the peak of this deflection did not necessarily

Fig. 2 Illustrates some examples of simultaneous lead electrocardiograms of contiguous bipolar, direct unipolar and reference leads. The upper in the electrocardiograms is the contiguous bipolar lead, the middle, the direct unipolar lead and the lower, the reference lead. From left to right—An example of simultaneous leads on the posterior surface of the right auricle, on the right septal surface and on the left posterior surface.

Fig. 3 Illustrates some examples of simultaneous contiguous bipolar lead electrocardiograms. The lower of the electrocardiograms is a reference lead.

correspond exactly with the peak or nadir of the unipolar lead electrocardiogram, but rather with the steep component of the intrinsic deflection of the unipolar complex, as Durrer had stated²³⁾. Due to the above mentioned reactions, the time of arrival of the activation wave was studied using the peak of the contiguous bipolar lead electrocardiogram (Fig. 3). In Fig. 3, the upper and middle tracings are electrocardiograms of a contiguous bipolar lead from points A and B of the posterior endocardium of the right atrium, from points C and D of the right surface of the interatrial septum, and from points E and F of the posterior endocardium of the left atrium respectively. The lower tracing is a reference lead. The peak of the main deflection of the contiguous bipolar lead was regarded as the time of arrival of the activation wave. The reason for this will be described in the discussion of this current study.

RESULTS

Concerning the features of unipolar lead electrocardiograms in each point. The right atrium: In a very small region of the atrium, unipolar lead electrocardiograms were nearly always completely negative in every lead. This region coincided with the region of the sinus node. In other regions near the sinus node, a small initial positive deflection rS type was registered each time in every lead. Some examples of features of the unipolar leads from the auricular endocardium were shown in Fig. 4. At the sulcus terminals, the complexes recorded were of rS type. The magnitude of the initial positivity tended to increase as the exploring electrode approached the inferior venae cavae, nevertheless, the feature remained the rS type. At the lower portion of the inferior venae cavae, i. e., at a portion near the atrio-ventricular groove, RS type waves were recorded. However, in any lead from this region large R waves were not as predominant as S waves. At the middle zone of the right atrium, the morphology of the unipolar leads were rS type, showing the predominant S type wave. However, the feature in the leads from the upper portion or lower portion of this area changed from predominant S waves to predominant R type waves. At the auricular appendage, the complexes were diphasic RS type waves, but R deflection was equal, or lesser in magnitude than S deflection. At the tip of the appendage, the feature changed from

Fig. 4 Illustrates the features of the unipolar lead electrocardiograms at various lead-points on the auricular surfaces.

The upper: The anterior aspect of auricular surfaces.

The middle: The posterior aspect of auricular surfaces.

The lower: The right spetal aspect.

diphasic to a tall R deflection followed by a small s deflection. In all instances pure monophasic positive R type waves were not registered from the posterior right atrium. At the anterior wall of the right atrium, the complex of the unipolar lead electrocardiograms obtained from the marginal zone of the superior venae cavae, near the sinus node, were always the rS type waves. As the exploring electrode approached the anterior wall of the appendage, the feature became a diphasic wave with a predominant positive wave. Thus, at the tip of the appendage, the predominant R wave with a small s deflection or Rs type were always found. At this region too, the pure monophasic positive R wave was not observed.

On the left atrium: At the marginal zone for both venae pulmonaris, the complex recorded was rS type with a somewhat predominant S wave. At the mid zone of the posterior portion of the left atrium, the feature of the unipolar lead was diphasic RS type. As the exploring electrode approached the atrio-ventricular groove the complex obtained became Rs type with a predominant R wave. But in this region too, the pure monophasic R wave was not observed. As the exploring

electrode gradually approached the tip of the appendage, the complexes registered tended to show a larger R deflection than S deflection. According to the present data, at the tip of the left appendage, the pure positive R wave was registered in two cases out of fifteen. At the anterior region of the left atrium, the complexes did not show a more evident change than that at the posterior region. As the exploring electrode gradually approached the tip of the appendage from the aspect of Bachmann's bundle, the complexes registered changed from rS type to Rs type. But in this region too, the pure monophasic positive R wave was not observed.

On the right surface of the atrial septum: In the postero superior region of the atrium near the superior venae cavae, the complex found was of rS type with predominant S deflection, and RS type waves were not found. In the mid region, the morphology observed was also rS type, but the R wave was somewhat augmented. As the exploring electrode approached the inferior areas, the morphology recorded was also of the rS type, and in only a few experiments was the diphasic RS type recorded. The predominant R wave followed by a small s wave was not observed anywhere in this region.

Fig. 5. Illustrates the measures of the time of arrival of the activation wave at various lead-points of the auricular surfaces.

Measurements of the arrival times of the activation wave at various lead points are shown on Fig. 5. (Measures were obtained by the contiguous bipolar lead method)

On the right atrium: At a point near the sinus node, the activation wave reached the above mentioned point very rapidly. Travel time was approximately 0.0010 seconds. (measured from the onset of the P wave of the reference lead) When the exploring electrode was removed from this point, and attached to the sulcus terminalis and to the lower part of the inferior venae cavae, the activation wave reached this point in about 0.015 to 0.018 seconds. At the mid portion of the right atrium, the activation time was somewhat delayed, and at the appendage, it was approximately 0.016 to 0.026 seconds. At the tip of the appendage, the arrival time of the activation wave became increasingly delayed. However, anywhere along this region travel time was not longer than 0.030 seconds. At the anterior portion near the sinus node, i. e., at the marginal zone of the superior venae cavae, the activation time was approximately 0.017 to 0.018 seconds. The activation time became gradually prolonged as the exploring electrode approached the tip of the auricle. At the marginal zone of the appendage, it was approximately 0.023 to 0.024 seconds, and at the tip of the auricle approximately 0.026 to 0.033 seconds. In the latter region, the activation wave reached each point within the above mentioned time. The most prolonged point of arrival time of the activation wave, where arrival time was approximately 0.033 seconds, was not the tip of the appendage, but the inferior point a few millimeters distant from the tip of the appendage.

On the left atrium: In general the activation wave reached the left atrium somewhat later than the right atrium. Even at the earliest point of activation in the left atrium, activation time was somewhat more prolonged than that of the latest point in the right atrium. At the Bachmann's bundle, the activation time was about 0.030 to 0.033 seconds. As the exploring electrode approached the marginal zone of the left auricular appendage, the activation time increased gradually amounted to 0.035 to 0.036 seconds. At the tip of the anterior portion of the left auricular appendage, the activation times were increased amounted to 0.039 to 0.045 seconds. In this region,

the most delayed activation time was noted at the upper region of the tip. At the posterior wall of the left atrium, near the insertions of both pulmonary veins, the activation time amounted to approximately 0.041 seconds. In respect to this value it was found that the electrode was hanging to the vascular tissue, and was not useable for the determination of the activation time. At the insertion of both pulmonary veins, the activation time was about 0.033 to 0.035 seconds. At the mid zone of the posterior wall, where the Bachmann's bundle ramifies into the tissue of the appendage, the activation time was from 0.036 to 0.038 seconds. The nearer the exploring electrode approached to the tip of the appendage, the longer the activation time. In this region, the activation time amounted to about 0.044 to 0.049 seconds. The area, where the latest activation time, amounting to 0.049 seconds was observed, was the upper regional tip of the appendage. A fact worth noting was that the feature of unipolar lead electrocardiogram at this point was a pure monophasic positive R type wave.

On the atrial septum: Generally speaking the activation time in this region was comparatively delayed. Even in the upper region near the sinus node the activation time amounted to 0.018 seconds. The activation time was prolonged gradually according to the displacement of the electrode in regard to the anterior and lower zone. In the mid region, the activation occurred at about 0.024 seconds, and in the lower zone the activation time was from 0.028 to 0.031 seconds. According to these results, activation seems to take place in the order of top to bottom, and from back to front.

Next, the conduction rate of the activation wave in each region of the auricular muscles was measured according to the relationship between the activation times at each point, and the distances between each lead point.

The value obtained from the portion nearest the sinus node was highest and ranged from 800 to 1200 mm/sec. The conduction rate in the right auricular appendage ranged from 500 to 800 mm/sec. However, the conduction rate in the left atrium was rather less than that in the right atrium and ranged from 250 to 400 mm/sec. In the region near the vascular tissue of each artery or vein, the conduction rate was very small and amounted to approximately 200 mm/seconds. The results ob-

tained coincided with those of Lewis⁹⁾ and Sodi-Pallares²¹⁾.

PART II. The changes in arrival time of the activation wave at each lead point after ligation or cauterization of Bachmann's bundle, lower part of the Taenia terminalis, and the cava venous appendage angle.

MATERIAL AND METHOD

In these experiments, seventeen mongrel dog were prepared in the same manner as in part I. After the heart was exposed, the following manipulations were performed to facilitate experimentation, i. e., the pericardial sac was opened till the root of the vena cava superior and the adhesion to the Aorta and the front region of the auricles was easily peeled off with the fingers. By doing this the Bachmann's bundle was readily exposed. Bachmann's bundle, the Taenia terminalis or cava venous appendage angle was ligated about 1.5 cm wide perpendicular to the muscle fiber, by use of a round surgical sewing needle.

RESULTS

Fig. 6 Illustrates the delay of the activation time after the ligation of Bachmann's bundle at each representative point of the auricular surfaces. Mark A and B show the E.C.G. of before and after ligation of Bachmann's bundle respectively.

Results of ligation and cauterization of the Bachmann's bundle: The ligation of Bachmann's bundle was performed at a point in the middle of the bundle. The activation time at point 1, a point of the anterior right portion of the atrium, did not show the prolongation after the ligation (Fig. 6). But at points 2 and 3, two points of the left atrium, the activation times were prolonged considerably after ligation. Delays amounted to 0.010 and 0.049 seconds respectively. From the exploring electrode inserted into the atrial septum, point 4 in Fig. 6, no effects upon the ligation of Bachmann's bundle were recorded. The results of these experiments are shown in Fig. 7. In this figure, cauterization

Table I a.

Comparison of the delay of the activation time at each lead-point.			
Point Explored	Before	After Ligation	After Cauterization
1	0	+0.025"	+0.033"
2	0	+0.024"	+0.032"
3	0	+0.010"	+0.018"
4	0	+0.003"	+0.012"
5	0	+0.007"	+0.017"
6	0	+0.004"	+0.015"
7	0	+0.011"	+0.020"
8	0	+0.002"	+0.015"
9	0	+0.004"	+0.010"
10	0	+0.003"	+0.010"
11	0	+0.003"	+0.005"
12	0	+0.001"	+0.003"
13	0	+0.001"	+0.001"
14	0	+0.002"	+0.002"
15	0	+0.002"	+0.002"
16	0	+0.002"	+0.002"

— The point of ligation of Bachmann's bundle.

Table I b.

Comparison of the delay of the activation time at each lead-point.			
Point Explored	Before	After Ligation	After Cauterization
1	0	+0.009"	+0.037"
2	0	+0.006"	+0.036"
3	0	+0.005"	+0.022"
4	0	+0.015"	+0.015"
5	0	+0.015"	+0.015"
6	0	+0.015"	+0.015"
7	0	+0.011"	+0.011"
8	0	+0.002"	+0.002"
9	0	+0.001"	+0.001"
10	0	+0.001"	+0.001"
11	0	+0.001"	+0.001"
12	0	+0.001"	+0.001"
13	0	+0.002"	+0.002"
14	0	+0.001"	+0.001"
15	0	+0.001"	+0.001"

was overlapped to the ligation in the same place. The activation times at the points from 1 to 11 of the anterior wall of the left atrium were delayed by ligation of Bachmann's bundle, and they were more prolonged after overlapping of the cauterization. In contrast, the influence of the ligation or cauterization was not observed at points 12 to 16 of the anterior wall of the right atrium. The tendency was the same in the posterior wall of the left atrium, remarkable prolongation of activation time was found after the same procedure. However, at point 9 of the atrial septum and point 8 of the postero-inferior portion of the left atrium, no remarkable changes in the activation time were observed. At points 10 to 15 of the posterior wall of the right atrium, the activation time scarcely

prolonged after ligation and cauterization.

The results of ligation of the lower portion of the Taenia terminalis: The ligation of the Taenia terminalis was performed at the lower part of the terminalis at a width of about 1.5 cm., as shown in Fig. 8. In Fig. 8 the result obtained in each

Table II a.

Comparison of the delay of the activation time at each lead-point.		
Point Explored	Before	After Ligation
1	0	+0.011"
2	0	+0.001"
3	0	+0.001"
4	0	+0.001"
5	0	+0.001"
6	0	+0.001"
7	0	+0.001"
8	0	+0.001"
9	0	+0.001"
10	0	+0.001"
11	0	+0.001"
12	0	+0.001"
13	0	+0.001"
14	0	+0.001"
15	0	+0.003"
16	0	+0.017"
17	0	+0.031"
18	0	+0.036"
19	0	+0.023"
20	0	+0.007"

Fig. 8 Illustrates the delay of the activation time at each lead-point of right auricular endocardial surface after the ligation of the lower part of the Taenia terminalis.

experiment are illustrated. In this figure, we can see that no remarkable changes occurred at points 1 to 14 after ligation, but at points 15 to 20, the activation time was significantly delayed after ligation of the lower portion of the Taenia terminalis.

Results of the ligation of the Cava Venous appendage angle: In these experiments, the ligation was performed at the Cava Venous appendage angle under the same procedure as used on Bachmann's bundle and the Taenia terminalis. The result in each experiment are shown in Fig. 9. The acti-

Table II b.

Comparison of the delay of the activation time at each lead-point.		
Point Explored	Before	After Ligation
1	0	+0.008"
2	0	+0.007"
3	0	+0.004"
4	0	+0.004"
5	0	+0.003"
6	0	+0.002"
7	0	+0.001"
8	0	+0.001"
9	0	+0.001"
10	0	+0.001"
11	0	+0.001"
12	0	+0.002"
13	0	+0.002"
14	0	+0.001"
15	0	+0.007"

Fig. 9 Illustrates the delay of the activation time at each lead-point of right auricular endocardial surface after the ligation of the Cava venous appendage angle.

vation time was delayed at each point of the anterior surface of the atrium after ligation of the Cava Venous appendage angle, but remarkable changes were not observed at points 9, 10 and 11 (points

of the posterior surface of right atrium). (Table 2b) When the Torus Lowerii was cut through at the front of the Fossa Ovari, A-V block was induced immediately.

PART III. Process of the auricular activation wave in artificial extra systole, provoked in the left auricular appendage by inductorium stimulation.

MATERIAL AND METHOD

For this experiment seventeen mongrel dogs, which were used for the experiments in Part II, were made use of at the same time.

Artificial stimulation was given to the endocardium of the left auricular appendage by means of the inductorium stimulation. The process of the propagation of the activation wave in artificial extra systole was studied in both conditions before and after the ligation of Bachmann's bundle and the lower portion of the Taenia terminalis. One of these experiments was shown in Fig. 10. Point "N" is the point that was stimulated.

Fig. 10 Illustrates the changes of the electrocardiograms in artificial auricular premature beat provoked at point "N" of the anterior endocardial surface of the left auricular appendage. A: Changes before the ligation of the Bachmann's bundle and B, that after the ligation. The upper of the electrocardiograms is the contiguous bipolar lead, the middle, the direct unipolar lead and the lower, a reference lead. The arrows in the electrocardiograms are the onset of the escaped current in artificial stimulation.

Point 1 is a point on the marginal zone of the anterior wall of the left atrium. This point is near the end of Bachmann's bundle. Point 2 is a point on the mid region of the anterior right auricular appendage. Point 4 and 5 are points on the downward portion of the atrial septum, and on the posteriorly inferior portion of the right atrium near the atrioventricular groove. The time of arrival of the activations were measured with the time interval from the escaped current to the peak of the main deflection of the contiguous bipolar lead tracing. The latency of the auricular muscle was assumed to be identical in all regions.

RESULTS

An example of these experiments was illustrated in Fig. 10. These results indicate that the activation time at point 1 in artificial auricular extra systole was not changed by ligation of Bachmann's bundle, but that at point 2 activation time was delayed remarkably after ligation. Those at point 3 (a posterior point of left atrium) point 4 (a point of atrial septum), and point 5 (a point of posteriorly inferior portion of the right atrium) were not significantly prolonged after the ligation. The results in each experiment are shown in Figs. 11 and 12. In the experiment illustrated in Fig. 11, the stimulation point was placed at a point of the anterior wall on the left auricular appendage. The activation time at point 1 in extra systole, before ligation of Bachmann's bundle was about 0.023 seconds, but that after ligation or cauterization, the activation time was about 0.022 seconds,

Fig. 11 Illustrates the changes of the activation time in artificial extra systole at each lead-point of the auricles before and after ligation or cauterization of the Bachmann's bundle. Artificial stimulation was given at the point "N" of the anterior endocardial surface of the left auricular appendage.

Fig. 12 Illustrates the changes of the activation time. Artificial stimulation was given at the point of posterior endocardial surface of the left auricular appendage. (compare with Fig. 11)

0.026 seconds respectively. No notable changes were observed. On the contrary, activation time at point 2 (a point on the anterior portion of the right atrium) in the extra systole was about 0.035 seconds before ligation, but after ligation, it was about 0.079 seconds, and still more prolongation was found when cauterization was overlapped to the ligation. The values measured were 0.082 seconds. At each point of the posterior wall of the left auricle, the activation time in extra systole was not significantly changed by the ligation or the cauterization. In like manner, the activation time in the extra systole at point 4 (a point of the postero-inferior portion of the left atrium), point 5 (a point of the inferior portion of the atrial septum), and point 6 (a point of the postero-inferior portion of the right atrium near the atrio-ventricular groove) remained almost unchanged after these manipulations. On the contrary, the activation time in the extra systole at point 7 (a point near the insertion of the superior venae cavae) was about 0.040 seconds before ligation of Bachmann's bundle, but after ligation and cauterization, the activation time was delayed about 0.077 seconds and 0.081 seconds respectively. Next, the activation time at point 1 (a point near the anterior insertion of the left auricular appendage) in the extra systole provoked at the posterior left auricular appendage was about 0.016 seconds, and that after ligation of Bachmann's bundle was 0.023 seconds, and that after cauterization was 0.022 seconds. From these values, no significant delay was observed. (Fig. 12).

In contrast, the activation times in the extra systole at points 2 and 3 (two points of anterior

portion of the insertion of the right auricular appendage, 0.5 mm apart from each other) were about 0.057 seconds and 0.059 seconds respectively before ligation of Bachmann's bundle. After ligation they were 0.067 seconds and 0.070 seconds respectively. The longer prolongation was observed in points 2 and 3 after cauterization was overlapped to ligation and the values measured were 0.079 seconds and 0.083 seconds respectively. Noteworthy was that the delay of the activation time was more significant at point 3 than at point 2. On the other hand, the activation time in an extra systole at points 1, 2 and 3 (three points of the posterior insertion of the left auricular appendage) were not significantly changed by ligation or cauterization of Bachmann's bundle. In like manner, the activation times at point 4 (a point of inferior portion of the atrial septum), and points 5 and 6 (two points of the postero-inferior portion of the right atrium near the insertion of the inferior venae cavae) were not remarkably delayed after ligation and cauterization. In contrast, the activation time in the extra systole at point 7 (a point near the insertion of the superior venae cavae) was about 0.049 seconds, but it was significantly delayed after ligation, and still greater prolongation was observed after cauterization overlapped. The values obtained were 0.065 seconds, and 0.077 seconds respectively.

Point Explored	Activation time in extrasystole provoked at the point "N"	
	Before Ligation	After Ligation
1	0.060"	0.062"
2	0.067"	0.068"
3	0.060"	0.062"
4	0.071"	0.091"
5	0.085"	0.094"
6	0.081"	0.091"

N : Stimulus point.

Fig. 13 Illustrates the changes of the activation time in artificial extra systole at each lead-point of the right auricle before and after ligation of the lower part of the Taenia terminalis. Artificial stimulation was given at the point "N" of the anterior endocardial surface of the left auricular appendage.

When the artificial auricular extra systole was provoked at the anterior left auricular appendage as shown in Fig. 13, the activation times at points 1 to 3 was about 0.062 seconds, 0.068 seconds, and 0.062 seconds respectively after ligation of the lower part of the Taenia terminalis. These values were not prolonged compared with those before ligation of Bachmann's bundle. But at points 4 to 6 (three points on the postero-inferior portion of the right atrium near the venae cava inferior), the activation times in an extra systole were about 0.071 seconds, 0.085 seconds, and 0.081 seconds respectively before the ligation, but after the ligation of the lower part of Taenia terminalis they changed to 0.091 seconds, 0.094 seconds, and 0.091 seconds respectively. From these values, remarkable prolongations were observed after ligation of the Taenia terminalis. The conduction rate of the activation wave in Bachmann's bundle was about 1000 mm/seconds in normal heart beat, but that in an artificial auricular extra systole evoked in the left auricular appendage was about 700 mm/seconds.

DISCUSSION

From the findings of the features in experimental direct unipolar lead electrocardiograms, it was confirmed that at the region near the sinus node the feature of unipolar lead electrocardiogram was negative throughout or predominantly negative, but at a point apart from this region, it was biphasic. The more apart from this region, R waves became more prominent and at the area of both auricular appendage, the feature was diphasic with predominantly positive waves. These findings were agreeable with the results of Sodi-Pallares²¹⁾ and of Lewis⁹⁾, and at the region activated last of all (the posterior portion of the left auricular appendage), the feature was completely positive. But according to Sodi-Pallares²¹⁾, the posterior region of the left auricle near the insertion of the left pulmonary veins was found to be the last zone of activation, in other words, this was the only region where a completely positive R wave was found. The present data was agreeable with the classic concept that, as Lewis⁹⁾ reported, the tip of the left auricular appendage was the region activated last of all. The reasons why different results were obtained are contemplated as follows. The posterior region of the left auricle near the left pulmonary vein is the most difficult region to approach, and it

consists anatomically of vascular tissue mixed with the auricular muscles. In an experiment, it is very difficult to fix the exploring electrode to this region from the epicardial aspect, especially at the end of the auricular muscle tissue. But according to the present method, the fixation of the electrode was extremely easy everywhere, and this region was no exception. Craib³⁶⁾ reported that, when the activation wave spread from one side of the muscle strip to the other side, purely positive deflections were recorded from the electrode attached to the end of the muscle strip. From the electrode fixed to a point on the activation course, a negative S wave may be observed as a phenomenon along the activation front. As this reaction principle on muscle strips coincides with the auricular muscle tissue reaction, the results of the above mentioned report are experimentally correct. In the above mentioned this reaction appeared to be the reason why the S type wave was registered from the electrode fixed to the endocardium at the insertion of the pulmonary vein. Therefore, the electrode at this insertion was supposed to be situated half way along the auricular muscle. Lewis⁹⁾ and Takayasu²²⁾ carried out some experiments on the propagation of the auricular activation. This was accomplished by means of direct unipolar lead electrocardiograms from the epicardium of the auricles. But, it is a matter of great importance to point out the just point of deflection as the mark of arrival time of the activation. The intrinsic deflection of the unipolar direct lead electrocardiogram are generally accepted as the mark of arrival time of the activation. The intrinsic deflections are very sharp, nevertheless, the duration times are too lengthy for an accurate determination of the arrival time of the activation. Therefore, the opinions of each investigator do not necessarily coincide with each other in regard to the point marking the arrival time of the activation wave. For an example, Lewis²⁹⁾ and Wilson³⁰⁾ took the peak of the R deflection, Wilson³¹⁾ took the nadir of the S deflection. On the other hand, Maekawa³²⁾ accepted the point crossed by the zero line. In connection with this matter, Clement³⁷⁾ and Harris²⁵⁾ reported the contiguous bipolar electrode as being the best method. Toyoshima³⁴⁾, Sodi-Pallares²¹⁾, and Durrer²³⁾, judging from the main deflection of the contiguous bipolar lead electrocardiogram, concluded that the arrival time of the activation wave coin-

cide with some instant during the duration of the intrinsic deflection of the unipolar lead electrocardiogram. However, it did not necessarily coincide with the specialized point of deflection such as at the top or the nadir. According to Itatsu^{26,27)}, the QRS complexes recorded by the contiguous bipolar electrode were very narrow, and it was possible to discuss the exact moment of muscular activation due to the short duration of the activation. He elucidated these problems theoretically and proved them by experimentation. He used the tip of the main deflection of the contiguous bipolar lead electrocardiogram as the arrival time mark of the activation wave. In the present study, the author employed the tip of the main deflection of the contiguous bipolar lead as arrival time mark of the activation. This agreed with the result of Itatsu. In the present data, the tip of the main deflection of the contiguous bipolar lead coincided with somewhere of the intrinsic deflection in direct unipolar lead electrocardiogram simultaneously recorded. This was as Toyoshima³⁴⁾ has previously concluded. The tip of the contiguous bipolar lead electrocardiogram coincided very well with the sharpest portion of the intrinsic deflection. This was in agreement with the results obtained by Durrer²³⁾ and his associate. Most of the values measured at the tip of the contiguous bipolar lead were agreeable to the values measured by Lewis⁹⁾ and Sodi-Pallares²¹⁾, and intimate relationship were observed between the features and the activation time. For example, rS type wave were recorded in the area where the activation wave arrived in the early stages of auricular activation. From the area, which was activated at a later stage of auricular activation, biphasic waves with predominant R waves were recorded. However, there was a place along the activation front where the activation time and morphology did not have the above mentioned relationship. For example, at points 1 and 2 in the posterior portion of the right atrium in Fig. 4 the activation times were nearly the same (about 0.018 second), and the features were of rS type waves and RS type waves respectively. At points 3 and 4 (a point on the posterior left auricular appendage and a point on the anterior left auricular appendage respectively), the activation times were nearly the same at approximately 0.040 seconds, but the features of the complexes were of RS type and

Rs type respectively. The opposite phenomena could also be observed. There were some points along the activation front representing similar morphology but different activation times. For example, in points a and b (two points on the posterior portion of the left atrium as illustrated in Fig. 4) the features of the complexes were RS type waves in both cases, but the activation times of each were about 0.042 seconds and 0.035 seconds respectively. The reason of this has not yet been determined. In general, the results obtained in the present experiments coincided more or less with these of Sodi-Pallares²¹⁾.

According to Lewis⁹⁾ the activation waves in the auricles proceed radially from the sinus node, and according to Rothberger & Scherf¹⁰⁾, propagation of the activation wave to the left atrium is transmitted via Bachmann's bundle; this being the shortest route for the left auricle. On the other hand, according to Condrelli¹¹⁾, the activation of the atrium is transmitted through three ways, i. e., sino-Tawara, sino-left, and sino-right conduction systems. Recently, Takayasu²²⁾ confirmed the results of Condrelli¹¹⁾ by histological and experimental research on the canine heart. According to the present study, the onset of the activation of the left auricular muscle was remarkably delayed after ligation or cauterization of Bachmann's bundle, but that activation of the right atrial muscle remained unchanged. From these results, the author formed the opinion that Bachmann's bundle is the shortest route of transmission of the impulses to the left atrium as mentioned by Condrelli¹¹⁾, and Rothberger, Scherf¹⁰⁾ and Takayasu²²⁾. However, it was found that there was an area where the onset of the activation did not present any delay even after ligation or cauterization. It was located in a small region of the posterior and inferior portion of the left atrium near the atrio-ventricular groove. (Fig. 7. Table 1b. Points 8, 9, 10) From these results in the present study it was surmised, that the activation of the atrial septum was not influenced by ligation and cauterization. It was also surmised that there might be a portion, where the activation was not transmitted via Bachmann's bundle, but was transmitted directly from the postero-inferior aspect of the right atrium. The ligation was performed at the lower part of Taenia terminalis, then the arrival time of the activation wave to the portion beyond the center of ligation was re-

markedly prolonged. The same tendency was observed in all experiments (Fig. 8), i.e., the arrival times of the activation wave at points 1 through 14 (numerous points along the anterior and posterior portion of the right atrium) indicated few changes, but at points 15 through 20, which were located in the peripheral region of the ligation, they were delayed after the procedure. (Fig. 8, Table 2a) In the cases in which the ligation was performed at the cava venous appendage angle, the arrival time of the activation wave at each point along the anterior wall was uniformly prolonged. (Fig. 9, and Table 2b). However, the degree of prolongation was most distinct at the peripheral portion near the ligation, and at other points; for example, at points 8, 12 and 14 etc. (Fig. 9, Table 2b). These points were located anteriorly to inferior apart from the ligation portion, and the degrees of prolongation were considerably less than those as the points near the ligation. These results from my experiments may reveal that the activation wave coming from the sinus node reaches the peripheral region of the ligation with a marked delay. This delay is especially noticeable in the adjacent region of the ligation. On the other hand, at the small area, along the mid proportion of the posterior right atrium, no prolongation of activation times were observed after ligation of the cava venous appendage angle. (Fig. 9, points 9, 10, and 11, Table 2b). As for the propagation process of the activation wave in the right atrium, Condrelli¹¹⁾ and Scherf¹⁰⁾ reported that the activation is transmitted through the specialized muscle fiber, i.e., sino-right conduction system, which is supposed to have the same function as Bachmann's bundle. On the other hand, D. Durrer²⁴⁾ studied the direction of propagation of the activation wave with the aid of differential epicardial electrodes. He stated that the activation wave traveled with a constant speed along the right auricular surface but did not spread exactly radially, and was slightly curved. He also stated that the activation wave spread along the thick muscular ridges of the auricular wall quite rapidly, and that the activation wave was propagated to the tangential direction through auricular myocardium. To these opinion mentioned above, Sodi-Pallares²¹⁾ stated that the specific route of conduction was still undetermined. To clarify these questions, the ligation was performed by the author at a portion near the head or the tail of

the sinus node. According to the results of these experiments, the activation wave reached the caudal region beyond the ligation with some delay, and that the degree of the delay was longer as the point was moved closer to the center of the ligation. If the activation wave should propagate through a specialized muscle fibers as Condrelli¹¹⁾ mentioned, delay of the activation time would be nearly the same at each point under the control of the same specialized muscle fibers. Judging from the electrical phenomenon in the present study, and having no connection with the hisological findings, the author confirmed that the activation wave originating in the sinus node spread in short lengths through a specialized muscle fiber, such as sino-right conduction system, and then spread radially from muscle to muscle. Moreover, from the experimental findings in artificial auricular extra systole provoked at point "N", in Fig. 10, 11, 12 and 13 (left auricular appendage), it is surmised that the activation provoked in the left atrium spread to the right atrium via Bachmann's bundle, and then propagated to the anterior region via the aortic side from there to the posterior region via cava venous appendage angle, and then reached the postero-inferior of the right atrium through the Taenia terminalis. In the present experiments, the arrival time of the activation wave in artificial auricular extra systole was measured by use of the escaped current registered on the electrocardiogram. When the ligation was performed at the lower part of the Taenia terminalis, the activation time at the region beyond the ligation was markedly prolonged, but the morphology of the contiguous bipolar lead electrocardiogram before and after the ligation registered no essential differences. For this reason it was surmised that the activation wave in the auricular extra systole after they were ligated, may travel along the same aspect of the auricular region as before the ligation. However, in the case in which Bachmann's bundle was ligated, the activation time in the extra systole was remarkably delayed at the anterior and high posteriorly regions of the right atrium. The reason for the marked delay may be as follows: The activation wave in the extra systole is perhaps transmitted to the anterior region of the right atrium through the postero-inferior regions of the left and the right atrium. From this data, it was surmised that the activation wave in the artificial auricular extra systole, provoked in the

left auricle, may be propagated radially in the left atrium, and transmitted to the anterior, posterior region of the right atrium via Bachmann's bundle. When it is impossible to pass through Bachmann's bundle, due to the ligation, the activation wave perhaps reaches the anterior region of the right atrium via the postero inferior portions of the left and right atrium. The process of the propagation of the activation wave in the atrial septum did not reveal any changes after the ligation of Bachmann's bundle.

CONCLUSION

The author has performed research on the arrival time of the activation wave, on the morphology, on the conduction rate, and the process of the propagation of the activation wave in artificial auricular extra systole. This was provoked at the left auricular appendage in a canine heart, using a special electrode fixed on the atrial endocardial surface. The tip of the main deflection of the contiguous bipolar lead was used as the mark of arrival time of the activation wave.

1. The measure of the activation time in a normal heart were nearly the same as those measured by Lewis⁹⁾, and Sodi-Pallares²¹⁾ despite the fact that a different method was employed in the present study.

2. The auricular portion activated last was the tip of the left auricular appendage, and was not the region of the inferior left pulmonary vein as was used by Sodi-paralles²¹⁾.

3. The morphology of the complexes was nearly the same as that obtained by Sodi-Pallares²¹⁾, except at the tip of left auricular appendage, where the entirely positive deflection was observed. The entirely positive deflection was observed only at the region where the muscle were activated last of all.

4. Values for the conduction rate of the activation wave were agreeable with those of Sodi-paralles²¹⁾.

5. The shortest route for the conduction of the activation wave to the left atrium was via Bachmann's bundle, but the atrial septum has nothing to do with Bachmann's bundle as far as the propagation of the activation wave, and it seemed to be important to accept the fact that the activation wave propagates from the postero-inferior region of the right atrium to a small region of the

posterior-inferior portion of the left atrium.

6. The activation wave in the artificial auricular extra systole provoked at the tip of left auricular appendage reaches the right atrium via Bachmann's bundle under normal condition, but when the bundle was ligated or cauterized, it seemed to be transmitted to the right atrium through the posterior-inferior regions of the left and right atriums, and then spread into the anterior right atrium.

7. As for the process of the activation wave in the right atrium, the author could not assure wheather the activation wave was transmitted through the specific way of sino-right system as Condrelli and Scherf reported, or propagated in radial manner muscle by muscle.

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REFERENCES

- 1) Keith, A., and Flack, M.: *J. Anat. & Physiol.* **41**: 172, 1907.
- 2) Wybauw, R.: *Arch. Internat. de physiol.* **10**: 78, 1910.
- 3) Lewis, T., Oppenheimer, B. S., and Oppenheimer, A.: *Heart*, **2**: 147, 1910.
- 4) Lewis, T., *Heart*, **2**: 23, 1910.
- 5) Lewis, T., Meakins, J., and White, P. D.: *Phil. Tr. Roy. Soc., London, Series B*, **205**: 375, 1914.
- 6) Eyster, J. A. E., and Meek, W. J.: *Heart*, **5**: 119, 1913.
- 7) Meek, W. J., and Eyster, J. A. E.: *ibid.* **5**: 227, 1913.
- 8) Eyster, J. A. E., and Meek, W. J.: *Arch. Int. Med.*, **18**: 775, 1916.
- 9) Lewis, T.: London, 1925, Shaw & Sons, Ltd.
- 10) Rothberger, C. J. & Scherf, D.: *Zeitschr. f. d. ges. exp. Med.*, **53**: 792, 1926.
- 11) Condrelli.: *ibid.* **68**: 593, 156, 1929.
- 12) Condrelli.: *Atti Soc. Ital. Proger. Sci.*, **20**, 1931. Cit by Lit 17.
- 13) Laufer: *Zeitschr. f. klin. Med.*, **127**: 678, 1935.

- 14) Maekawa & Takayasu, M.: *Japanese Circulation J.* **2**: 137, 1936.
- 15) Takayasu, M.: *ibid.* **3**: 293, 1937.
- 16) Takayasu, M.: *ibid.* **3**: 381, 1937.
- 17) Takayasu, M.: *ibid.* **3**: 447, 1937.
- 18) Takayasu, M.: *ibid.* **15**: 111, 1951.
- 19) Takayasu, M.: *ibid.* **17**: 129, 1953.
- 20) Takayasu, M.: *J. Jap. Soc. Int. Med.* **41**: 20, 1952.
- 21) Puech, P., Esclarissat, M., Sodi-paralles, D., and Cisneros, F.: *Am. Heart J.* **47**: 174, 1954.
- 22) Takayasu, M.: *Japanese Circulation J.* **18**: 414, 1955.
- 23) Durrer, D. and van der Tweel, L. H.: *Am. Heart J.* **47**: 192, 1954.
- 24) Van der Kool, M. W. Durrer, D., Th van Dam, R., & van der Tweel, L. H.: *ibid.* **51**: 684, 1956.
- 25) Harris, A. S.: *Am. J. Physiol.* **134**: 319-332, 1941.
- 26) Itatsu, H.: *Japanese Circulation J.*, **18**: 1, 1954.
- 27) Itatsu, H.: *ibid.* **19**: 230, 1955.
- 28) Groedel, F. M. and Borchardt, P. R.: *Direct Electrocardiography of the Human Heart.* 1948.
- 29) Lewis, T. and Rothschild, M. A., *Phil. Tr. Roy. Soc. (London)* **206**: 181, 1915.
- 30) Wilson, F. N., Johnston, F. D. and Hill, I. G. W.: *Am. Heart J.*, **10**: 176, 1934.
- 31) Wilson, F. N., Rosenbaum, F. F. & Johnston, F. D.: *Advances in Internal Med.*, **2**: 1, 1947.
- 32) Maekawa, M., Takayasu, M.: *Igaku*, **2**: 552, 652, 735, 1947.
- 33) Sodi-paralles, D., Barbato, E. and Delmer, A.: *Am. Heart J.* **39**: 387, 1950.
- 34) Toyoshima, H., et al: *Japanese Circulation J.* **18**: 37, 1954.
- 35) Sodi-paralles, D., Rodriguez, M. Chait, L. O., and Zuckermann, R.: *Am. Heart J.*, **41**: 569, 1951.
- 36) Craib, E.: *Heart* **14**: 71, 1927.
- 37) Clement, E.: *Z. Biol.* **58**: 110, 1912.