Mechanism of the Auricular Arrhythmias

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The four auricular arrhythmias, premature systoles, paroxysmal tachycardia, flutter, and fibrillation, have been investigated in over 200 dogs by three methods: (1) high speed cinematography, (2) cathode-ray oscillography, and (3) multiple-channel electrocardiography. The hitherto unexplored body of the left auricle has been surgically exposed and thoroughly studied. Results indicate that all four arrhythmias are of unitary origin and may occur from one ectopic focus. The resulting arrhythmia depends largely upon the rate of discharge from that focus. There is no circus movement. Corroborative observations have been made on the arrhythmias in man. This conception of the auricular arrhythmias simplifies the understanding of their mechanism.

Since the classic studies of Lewis and his associates on the nature of the auricular arrhythmias, comparatively little has been published on this phase of the subject. Lewis believed that his experiments established the following concepts concerning the mechanisms of the auricular arrhythmias:

1. That auricular flutter is due to a regular circus movement in the auricles which sweeps around the openings of the venae cavae. The main impulse usually travels in a counterclockwise direction up the right auricle, circling the superior vena cava, then down the left auricle, and around the inferior vena cava, thus completing the circus. Regular daughter waves are sent off the main wave to the remainder of the auricles.

2. Auricular fibrillation is due to a circus movement of the same general type as that of auricular flutter, but in this instance the impulse pursues a tortuous and redundant path around the venae cavae and sends off irregular daughter waves to the remainder of the auricles.

3. Paroxysmal auricular tachycardia is of a different nature. Lewis believed that a rapidly discharging ectopic focus in the auricle is responsible for the arrhythmia; no circus movement is present. Other investigators, however, do favor circus movement as the mechanism of auricular tachycardia.

The evidence that Lewis gathered with regard to circus movement appeared so complete that his conclusions have been accepted as fact in most modern textbooks of physiology, cardiology, and medicine.

For the past three years we have been taking high-speed, colored cinematographs of the auricles of the intact dog's heart. A Western Electric Fastax 16-mm. camera was used with which films were taken at speeds up to 2,000 frames per second. When films taken at 2,000 frames per second are projected at eight frames per second, the motion of the auricles is slowed 250 times. Auricular events that occur in one second take four minutes to view on the screen. A magnifying lens is used which enlarges the auricle 100 or more times on projection. By careful photographic technic, and lighting the field with twelve RSP-2 photospot incandescent lamps, excellent pictures are obtained showing the most minute detail of auricular activity. By means of such pictures, the auricular contraction wave can actually be seen for the first time.

We have now studied over 75,000 feet of film from experiments on over 200 dogs with various experimentally induced auricular arrhythmias. The arrhythmias were produced by the aconitine3,4 and by the postelectrical stimulation methods. Simultaneous electrocardiograms were taken in all experiments. The electrocardiographic studies were usually done by means of a Technicon Cardiograph, a direct writing machine, which records three leads simultaneously. It is thus possible to make immediate and accurate observations upon the intrinsic deflections inscribed simultaneously.
by two or three electrodes placed on the auricles at strategic points, and at known distances from a firing focus. Thus, small differences in time of arrival of the cardiac impulse beneath each electrode can be accurately determined and the exact direction as well as the speed of the impulse can be definitely established. A dual-beam, cathode-ray oscillograph was employed in a number of experiments. This apparatus is especially valuable because (1) the electrical events at two points can be investigated simultaneously, (2) mechanical resistance is completely eliminated, and (3) the degree of amplification in time and magnitude of the impulse is much greater than that of standard equipment.

Auricular Fibrillation

Motion Picture Observations: In the motion pictures, auricular fibrillation is characterized by two phenomena: (1) Minute irregular contractions that are continuously present; these we term M contractions. (2) Large rhythmic, wavelike contractions; these we term L contractions.

The M contractions cannot be seen with the unaided eye, but reveal themselves on the high-speed films. They occur constantly throughout the auricular musculature and involve an area of auricular wall approximately .03 to 3 mm. in diameter. Superimposed on this sea of small contractions are large, moderately vigorous contractions that sweep across the auricle in a fairly regular wavelike manner, at rates of about 400 to 600 per minute. The “fibrillation” of the auricle that is visible to the unaided eye is due to these large contractions. These contractions are fairly uniform in time, but not in strength. They do not pursue a circus path, and no daughter waves are seen on the films. It can be stated that careful visual investigation by slow-motion pictures of both auricles, individually and together, provide conclusive evidence against circus movement in auricular fibrillation.

A variety of specific experiments designed to block the hypothetical circus path (burning, cutting, and the like) has been performed. These experiments have confirmed the visual evidence for the absence of circus movement.

Electrocardiographic Studies: We have investigated the electrical activity in auricular fibrillation by the electrocardiographic and oscillographic methods described above. In the ordinary electrocardiogram the familiar “f” waves probably represent the L contractions. In direct auricular leads small, rapid, irregular deflections are also present. These small waves of electrical activity may be related to the M contractions seen on the motion pictures, and are therefore also termed M waves. On the ordinary direct-lead electrocardiogram the M waves number roughly 800 to 1,600 per minute; on the more sensitive oscillogram they number as high as 10,000 to 40,000 per minute. The M waves are seen only in auricular fibrillation and indirect evidence has been obtained which indicates that these small, irregular waves are largely responsible for the irregular ventricular responses characteristic of this arrhythmia. In ordinary electrocardiograms of auricular fibrillation, the M waves cause tiny, rough irregularities in the “f” waves. The M waves are not present in the auricular waves of other rhythms.

Simultaneous records from two closely adjacent bipolar electrodes placed at numerous selected points on the auricular surfaces show that all waves are arrhythmic and are usually unrelated in frequency or amplitude. There is no evidence of a circus movement.

Oscillographic records made through esophageal leads in man with auricular fibrillation demonstrate electrical activity similar to that found in the experimental animal.

It was shown that digitalis greatly reduces both the amplitude and frequency of all types of electrical activity in the fibrillating auricles. The decreased electrical bombardment of the auriculo-ventricular node offers an explanation for the decreased ventricular rate in auricular fibrillation after digitalis administration.

It is concluded that in both man and animals, auricular fibrillation is a chaotic heterorhythmic disturbance. There is no circus movement.

Auricular Flutter

Motion Picture Observations: Auricular flutter is due to regularly recurring contraction waves
discharged from an ectopic focus at such a rapid rate that varying degrees of A-V block are produced. The flutter waves are similar to the large waves seen in auricular fibrillation but are more regular and generally more vigorous. No M contractions are seen in auricular flutter. No circus movement exists. Our evidence is as follows:

1. No circus movement of the wave of contraction in auricular flutter can be seen in the films. No daughter waves are present.

2. Interruption of the Lewis circus pathway, by burning or cutting, does not in any way interfere with the auricular flutter, either photographically or electrocardiographically.

3. Slow-motion pictures were taken of both auricles simultaneously during auricular flutter. The films were projected so that motion was slowed 250 times. If a circus movement were present, at rates of conduction as calculated by Lewis, on films taken at this speed one appendix should be seen to contract many seconds before the other. However, it can be clearly seen on the films that both appendices contract at nearly the same instant.

4. When auricular flutter is produced by applying aconitine locally at the center of the wall of the right auricle, the contraction waves are seen to originate at the ectopic focus in a perfectly rhythmic manner, and to spread over the auricles in all directions at once. It thus appears clear that the mechanism of auricular flutter is not a circus movement; for if it were, the contraction wave would have to pursue a unidirectional path around the cavae. The films show that this is not the case; each contraction wave takes its origin at the ectopic focus, and, instead of traversing the auricle in a single direction, actually spreads from the focus simultaneously in all directions. The visual observations disprove the circus movement theory of auricular flutter and establish the true nature of this auricular arrhythmia.

Electrocardiographic Studies: Lewis's evidence for circus movement in flutter is based on timing the intrinsic deflections with paired auricular electrodes. By the technic in general use at that time, the body of the left auricle could not be exposed sufficiently to allow adequate electrographic investigation in that region. As a result, a considerable gap in Lewis's hypothetical circus path exists. Since we have been able to obtain wide exposure of the body of the left auricle, it seemed advisable to analyze the intrinsic deflections in both auricles, including the heretofore unexplored gap (the body of the left auricle), and thus test our cinematographic conclusions by Lewis's own method.

By means of extensive dissection, we have explored both auricles, including the body of the left auricle, in 30 dogs, with completely consistent results, as described in the following experiments.

Experiment 1: Aconitine was placed in the natural crevice between the inferior vena cava and the pulmonary vein from the left lower lobe. This point approximates the caudal attachment of the interauricular septum to the wall of the auricles. Electrodes were placed equidistant (2 cm.) from the aconitine focus, one electrode on the body of each auricle respectively.

If a circus movement is present, the impulse should arrive at the electrode of one auricle an appreciable time (at least 0.15 second) before it reaches the other. We have found that this was not the case; the impulse arrived at both electrodes nearly simultaneously (within 0.005 second).

Experiment 2: Aconitine was placed at the same point as in Experiment 1. Paired fixed electrodes were placed on the body of the right auricle—one at a point 1 cm. from the aconitine focus, the other at a point 4 cm. from the focus. We have found that the impulse arrived at the more distal electrode at a significantly later time than at the proximal electrode.

The same procedure was now repeated on the left auricle in the same animal. The paired electrodes were transferred to analogous positions on the body of the left auricle. The timing of the respective intrinsic deflections clearly demonstrated that the course of the impulse in the left auricle was in the same direction as in the right, i.e., away from the aconitine focus.

This finding is of crucial importance since, if the impulse were pursuing Lewis's circus path, it should on its "return journey" travel toward the aconitine focus instead of away from it.

Thus, by Lewis's own method, when the course of the impulse over the heretofore unexplored gap (body of left auricle) is charted, the circus movement theory is shown to be invalid and the true nature of flutter is revealed.

Experiment 3: As described for the similar motion-picture experiment, aconitine was placed in the center of the body of the right auricle. After flutter
was produced, an electrode was placed on each side of the focus and equidistant from it. It was found again that the impulse arrived at each electrode simultaneously.

The observations of this experiment confirmed those seen on the motion-picture and in the other electrocardiographic experiments that the impulse travels away from the focus into all directions simultaneously.

The configuration of the auricular flutter wave of the electrocardiogram has been elucidated by these studies. It is found to consist of a P' wave followed by an oppositely directed, prominent Ta wave. The Ta wave also occurs in rapid paroxysmal auricular tachycardias. These observations on the configuration of the auricular complex have been found to be true in both animals and man.

These observations on the mechanism of flutter have been confirmed in the human. It has been found that there is an intrinsic deflection in esophageal and precordial leads in patients with auricular flutter. By timing the intrinsic deflection and observing the general configuration of the auricular complex, ectopic foci have been demonstrated in humans with flutter. The ectopic focus is characterized by a completely negative deflection. As the impulse travels away from this focus, the intrinsic deflection is preceded by a positive component which becomes larger as the distance from the focus is increased. Utilizing both the configuration of the auricular deflection and the timing of the intrinsic deflection, it has been found that the impulse spreads concentrically from the ectopic focus. There is no circus movement.

**Auricular Tachycardia**

Films of paroxysmal auricular tachycardia and of auricular flutter reveal that the contraction waves in the two rhythms are similar. The two arrhythmias differ in at least three respects: (1) The auricular rate (the rate of discharge from the ectopic focus) in tachycardia is slower than in flutter. (2) As a result of this slower rate each auricular wave is followed by a ventricular response, that is, no A-V block exists in auricular tachycardia. (3) The propagation of the individual tachycardia wave is faster than that of the flutter wave. As in flutter, each tachycardia contraction wave originates in an ectopic focus in the auricular musculature and proceeds to invade the auricles in all directions simultaneously.

**Unitary Nature of the Auricular Arrhythmias**

From our cinematographic, electrocardiographic and oscillographic observations the same basic mechanism would appear to be responsible for auricular premature systoles, auricular paroxysmal tachycardia, auricular flutter, and auricular fibrillation, that is, a single ectopic focus.

Evidence in favor of this viewpoint can be obtained from the following experiment, using a modification of Scherf's aconitine method. A drop of a 0.2 per cent solution of aconitine in benzene is placed on a small area (swabbed dry) of the wall of the auricle. Auricular fibrillation usually results after a few minutes. When the ectopic focus is cooled by spraying with ethyl chloride, the rhythm often changes in the following order: from auricular fibrillation to auricular flutter, auricular tachycardia, and sinus rhythm with auricular premature systoles. When the cooling is stopped and the point of application of the aconitine is allowed to come towards body temperature, a return of the arrhythmias in reverse order is usually observed. By manipulating the amount of aconitine applied and/or the temperature of the focus, it is often possible to maintain any desired arrhythmia for a relatively long period of time. Study of the slow-motion picture films of these experiments reveals that the contraction waves in auricular premature systoles, auricular tachycardia, and auricular flutter are indistinguishable except for their rate and speed of conduction. When the rate of discharge of impulses from an ectopic focus exceeds a certain critical level varying from 300 to 600 per minute in different animals, auricular flutter gives way to auricular fibrillation.

The same sequence of auricular arrhythmias as described above may be produced by electrical stimulation instead of the local application of aconitine. The rate of discharge of impulses can thus be controlled at will. As the rate is increased from 100 to 600 stimuli per minute, it is often possible to produce the cinematographic...
appearance of auricular premature systoles, auricular tachycardia, auricular flutter, and auricular fibrillation in the order named. The arrhythmias produced by electrical stimulation appear identical photographically with those produced by aconitine.

It is noteworthy that the same close relationship and transitions described above for the experimentally produced auricular arrhythmias have also been frequently observed in man, spontaneously, and after medication, after certain surgical procedures, after trauma to the heart, and following certain infections.

The present conception of the action of antiarrhythmic drugs, such as quinidine and digitals, is that they act largely through their supposed effect on the gap between the head and the tail of the circus movement. In the light of the above observations such a concept is untenable.

The observations on the auricular arrhythmias described in this paper will be included in a monograph now in preparation which will cover the entire subject in full detail.

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