

EXPERIMENTS ON THE ORIGIN OF AURICULAR FLUTTER AND FIBRILLATION

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Little is known about the normal and even less about abnormal stimulus formation. For auricular flutter and fibrillation the fascinating circus movement theory finds almost general acceptance as an explanation. Textbooks of physiology and electrocardiography mention the circus movement as the established mechanism of flutter and fibrillation in spite of certain serious objections and short-comings. In recent years the circus movement was adopted by some authors even to explain paroxysmal auricular tachycardias.^{1,3} This position is opposed by those who assume a heterotopic stimulus formation in a center as the responsible mechanism.

The experimental approach to these problems always suffered from the difficulties encountered in the experimental production of a paroxysmal auricular tachycardia. It is possible to create this disturbance by the injection of certain substances, such as barium salts, digitalis or strophanthin, or by ligation of a coronary artery. Under these conditions, however, the tachycardia usually is not as regular as the clinical tachycardias; all too often ventricular extrasystoles and ventricular fibrillation appear.

To study the abnormal stimulus formation, for the last eight years we have employed with success a method which was introduced over 60 years ago, that is, the topical application of different substances on the cardiac surface. In 1884, Langendorff studied arrhythmias produced by the application of crystal of sodium chloride, certain acids, and other substances on the isolated apex of the ventricle of the frog heart.⁶ He found that the resulting disturbances of rhythm are not simply due to tissue irritation or local currents evoked by tissue injury. Different substances elicited different arrhythmias so that a specific drug reaction had to be assumed. Later, this method of investigation was rarely used except in the studies by Kisch on the frog heart.

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This author was, however, mainly concerned with the influences of different ions on the normal stimulus formation in the sinus.⁴

It was demonstrated that barium and sodium chloride, applied topically on the epicardial surface of the heart or by subepicardial injection provoke extrasystoles with fixed coupling and various other extrasystolic arrhythmias as well as paroxysmal tachycardias.¹³ Later it was shown that preparations of digitalis and strophanthin, administered in the same way, elicited the same response.¹⁶ The latter drug was employed independently by Kisch, with the same results.⁵ Proof that these arrhythmias actually originated in the area of application was furnished experimentally: if extrasystoles were present, the application of the thermode was followed by an increased number of extrasystoles. If, however, extrasystoles had disappeared, they reappeared when the thermode was applied.¹⁸

The experimental study of auricular flutter is more difficult. The classic investigations of this arrhythmia had to be made on short runs, of flutter which persisted briefly following faradization of the auricle. More persistent flutter follows the administration of physostigmin, muscarin or acetylcholine, but these substances change the condition of the whole heart in a way which makes evaluation of the experimental results difficult. They usually lead to fibrillation and not to flutter.

Some years ago we succeeded in producing more persistent auricular flutter by faradization of the dog's auricle. According to Lewis the circulation central wave usually uses a path around the large veins up or down the taenia terminalis of the right auricle in which the sinus node is situated. During auricular flutter a broad ligature was applied over this structure in 17 animals and in 16 of these 17 experiments the auricular flutter persisted without any change in the electrocardiogram. Since in transient auricular flutter any manipulation of the auricles may bring the flutter to a sudden end, these results were considered significant,

One of the most reliable methods for the experimental production of extrasystoles with fixed coupling is the cautious intravenous administration of minute amounts of aconitine and vagus stimulation.²⁰ When the electrocardiogram still is unchanged in respect to rhythm rate and form of the waves, brief stimulation of the vagus nerve in the neck leads to the appearance of a bigeminy or trigeminy with a constant form. This disturbance of rhythm lasts sometimes for more than one hour and permitted for the first time the experimental study of bigeminy with fixed coupling.

The same substance, aconitine, was used in a new series of experiments. The heart of a dog was exposed in the usual way during membutal anesthesia. A 0,05 per cent solution was prepared from crystalline aconitine and 0,05 cc. was injected into the appendix of

the left or right auricle. The vagi were severed in the neck. Every precaution must be taken not to inject the aconitine into the cavity of the heart because this leads immediately to ventricular fibrillation. The electrocardiogram was always recorded in lead II.

Within 90 seconds following the injection of aconitine a regular tachycardia appeared with a rate between approximately 200 and 300 beats per minute. The tachycardia lasted for as long as 90 minutes with regular rhythm and permitted certain observations to be made.

Faradic stimulation of the right or left vagus nerve in the neck during the tachycardia always led to a remarkable acceleration of the auricles in all experiments. This acceleration occasionally amounted to more than twice the original rate. It appeared immediately after stimulation was started and the former rate returned within a few beats, after the stimulation ended. The auricular waves remain unchanged during the stimulation or show only those slight changes which one would expect with an increase of rate.

Fig. 1 shows in the beginning an auricular tachycardia with a rate of 300 beats per minute. Stimulation of the right vagus nerve causes a complete inhibition of the a-V conduction and an increase of the auricular rate to 375. With the cessation of stimulation the former rate of the auricle quickly returns.

Clamping the appendix of the auricle proximal to the area into which the injection of aconitine had been made caused an immediate disappearance of the tachycardia and the return of sinus rhythm. Removal of the clamp allowed the tachycardia to return within a few seconds.

Cooling of the site of origin of the tachycardia, that is, the area into which the injection had been made, caused the tachycardia to vanish for the duration of the cooling. The tachycardia always recurred immediately after the thermode was removed.

Fig. 2 shows a tracing from the same experiment as Fig. 1. It was actually taken shortly after that of Fig. 1 was obtained. It shows an auricular tachycardia with a rate of 300, the disappearance of the tachycardia during the cooling, and its reappearance after cooling was terminated.

Of importance for the evaluation of these experiments is the decision as to whether we are dealing with an auricular tachycardia of the "essential paroxysmal tachycardia" type or whether the tracings show auricular flutter. This differentiation is difficult and often impossible. The tracings show auricular waves which are similar to those published repeatedly as samples of auricular flutter in the dog. According to Lewis the auricular rate during flutter in the dog varies between 345 and 580.¹⁰ The rate in the experiments just demonstrated was below this range before the vagus stimulation and well within it

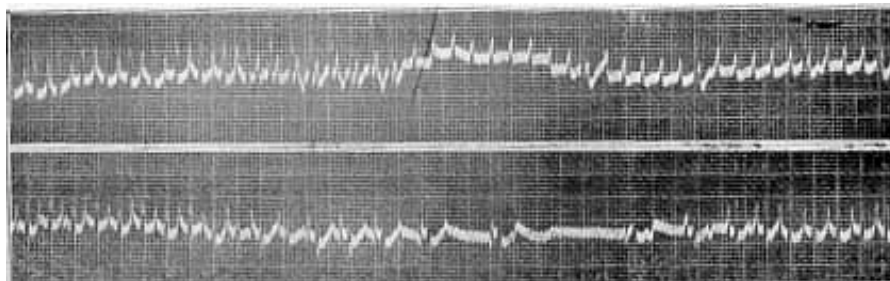


Fig. 1 - Effect of vagal stimulation during an auricular tachycardia (auricular flutter).

Fig. 2 - Cooling of the focus of injection of aconitine interrupts the tachycardia for the duration of the cooling.

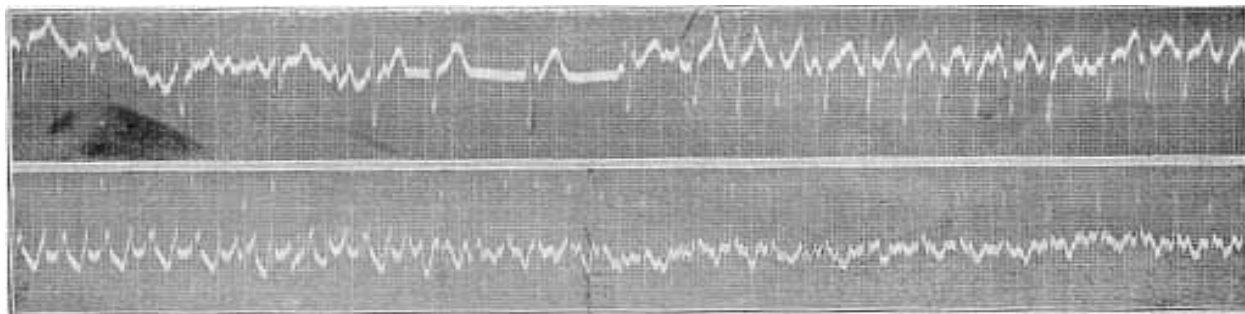


Fig. 3 - Cooling of the area of the injection of aconitine stops auricular fibrillation for the duration of the cooling.

Fig. 4 - Auricular flutter with 2: 1 block changes into auricular fibrillation.

during the stimulation. The form of the wave is compatible with flutter despite the presence of isoelectric lines between the single auricular waves. The contiguity of the waves need not be present, at least in some leads, in flutter. Against the diagnosis of auricular paroxysmal tachycardia due to a rapid stimulus formation in a center is the observation that even strongest faradic stimulation of the vagus never abolished the tachycardia. We know, however, that carotid pressure abolishes only a certain percentage of auricular tachycardias and our inability to do so in these experiments may be due to the special conditions.

The increase of rate by vagus stimulation has been repeatedly seen during auricular flutter.^{10,15} It has not been described up to the present in paroxysmal tachycardia, but heretofore it has never been possible to study the effect of vagal stimulation in auricular flutter was used by Lewis as a important fact to support the circus movement theory. The refractor phase of the auricles is known to be shortened to more than 1/5 of the normal value during vagal stimulation. According to Lewis, this abolishes barriers in the form of islands of refractory muscle in the way of the circulating wave, permits it to follow a less sinuous path, and therefore to circulate faster. Another possibility considered by Lewis was the utilization of another and shorter path by the central wave around the orifices of the great veins.^{11,12} This is rather improbable for a shortening of the path originally used around the veins sufficient to double the rate without any change of the form of the f-waves is not conceivable.

The experience that cooling of the site of the injection of aconitine which prolongs the refractory period, leads to the immediate disappearance of the tachycardia speaks strongly against a circus movement, unless one accepts a localized circus movement at the site of the injection.¹⁹ Still more important is the fact that interruption of the cooling causes the tachycardia to reappear instantly. Without the existence of a focus of stimulus formation this observation cannot be explained. A similar observation was not reported in the experiments by other authors because they worked, exclusively on flutter following faradization of the auricles. If this type of flutter once disappears it does not recur.

The experiments were continued by injecting the aconitine solution into the region of the taenia terminalis in which sinus node is situated. This has the advantage that the larger muscle bundle and the increased amount of connective tissue makes a sub-epicardial deposition of the aconitine much easier.

Injection into this site causes a similar tachycardia as the injection into the wall of the auricular appendices. Vagus stimulation, how-

ever, caused only a moderate acceleration of the rate. This may be due to the fact that the refractory phase of the specific tissue is less influenced by the vagus. Its refractory phase is certainly much longer than that of the common auricular muscle.^{2,9}

Repeatedly auricular fibrillation was obtained by an injection at this area and this could easily be stopped by cooling.

Fig. 3 shows auricular fibrillation which appeared after the injection of a fresh solution of aconitine into the area of the sinus node. Cooling caused an immediate cessation of the fibrillation with a cardiac standstill. A few automatic and normal beats appear and then the fibrillation starts again.

Repeatedly during or following vagus stimulation and sometimes even spontaneously the auricular tachycardia changes suddenly into fibrillation. Thus Fig. 4 shows a regular auricular tachycardia at the beginning of the tracing and suddenly this changes into fibrillation. Such changes are known to occur readily in flutter and are very rare in auricular tachycardia. Therefore this phenomenon speaks strongly in favor of the assumption that we are dealing with auricular flutter in the tracings like Fig. 1 and Fig. 2.

The reoccurrence of the fibrillation with the interruption of the cooling is not compatible with the circus movement in the sense of the Lewis' hypothesis.

If the circus movement theory is rejected and that of rapid stimulus formation in a center is accepted, the increase of rate during vagus stimulation represents a paradoxical effect. It is known, however, that under certain conditions the vagus exerts stimulating effects on the heart.²⁰ It is established that during vagus stimulation the chronaxie of the heart is decreased, that is, the excitability is increased.⁸ The effect of vagus stimulation on the rate of stimulus formation has not been explored up to the present time.

There are many strong arguments against the circus movement theory of Lewis.¹⁴ By mapping out the path taken by the central wave by means of direct leads from the auricle of the dog the full path has been investigated only once and even in this single experiment this was incomplete owing to technical difficulties. Further-more, the attempt by Lewis and his coworkers to calculate during flutter the rotation of the auricular axis for 360 degrees from the form of the f-waves is not justified. These waves are certainly not due to the central wave in a small bundle of muscle but owe their origin to the spread of the centrifugal waves over the rest of the auricles.

These objections and the experiments discussed in this communication do not prove that a circus movement does not exist. Circus movement does occur in muscular rings and the occurrence of return extra-sistoles in the human heart speaks in favor of circulating waves. It

is also certain that many peculiarities of auricular fibrillation and flutter and the action of some drugs on these arrhythmias are explained better by the assumption of a circus movement than with the other theories. The experiments reported here, which are being continued, show however that it is improper to state that flutter and fibrillation are caused by a circus movement without even discussing other possibilities. In this respect the followers of Lewis go farther than Lewis himself who never forgot that he merely supplied important facts in favor of a brilliant hypothesis.

CONCLUSIONS

Topical application of aconitine to the dog's auricle caused regular rapid auricular tachycardia and fibrillation of the auricles. Since this tachycardia often changed into fibrillation it is assumed that it represents auricular flutter. Isolating the area of the injection from the rest of the auricle by a clamp restored sinus rhythm for the duration of the clamping.

Cooling of the area of injection abolished flutter and fibrillation for the duration of the cooling.

Reference is made to the fact that these observations are incompatible with the circus movement theory of Lewis.

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