The Mechanism of Auricular Fibrillation and Flutter

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The Diagnosis of auricular fibrillation at the bedside is based on detecting the complete irregularity of the heart beat: the “pulsus irregularis, inequalis, deficient and intermittens,” apparently first described by Bouilland in 1836.1 The diagnosis is indirect and therefore occasionally fallacious since (a) a “delirium cordis” may also result from the action and interaction of multiple ectopic impulses, and (b) the coexistence of a complete auriculoventricular block (for example in digitalis intoxication) will give rise to a regular heart beat in the face of the auricular disorder. The direct demonstration of auricular fibrillation (other than by inspection in open chest experiments or during operations) depends on the graphic demonstration of the disturbed auricular function by pulse tracings or by the electrocardiogram. In these, evidence of coordinated auricular activity is replaced by rapid irregular undulations of the base line with various and simultaneous frequencies ranging from 300 to over 1000 cycles per minute. It appears that individual cell units maintain their characteristic properties, displaying membrane, and action, potentials with depolarization and recovery in a reasonably orderly manner.2 It is likely, therefore, that initiated by the electrical events, small areas of auricular tissue will undergo mechanical contraction and relaxation (“microsystole” of Wenckebach, “functional fragmentation” of Lewis). These are incoordinate and incoherent and, therefore, of no mechanical consequence as far as propulsion of blood is concerned: the auricles are in functional diastole. The disorder may be described as an “auricular dysrhythmia,” to borrow a neurologic phrase.

Auricular flutter, on the other hand, clearly represents a rapid series of coordinate auricular contractions that are mechanically effective. Systolic auricular contractions and diastolic relaxation may be demonstrated by fluoroscopy, kymo- and electrokymography. The condition first observed in man by Ritchie (1905)3 and by Hertz and Goodhart,4 was clearly described by Jolly and Ritchie in 1910.6 The recognition of this disorder at the bedside is difficult. It may become indistinguishable from auricular fibrillation if the ventricular rate is irregular. At regular rates, the steplike increase in rate on exercise or decrease on carotid sinus pressure with a temporary complete irregularity during the rising or falling phase in the ventricular rate is characteristic.6 Again, the direct demonstration of auricular activity is essential for the diagnosis either by direct inspection of the jugular veins displaying the transmitted auricular waves or by graphic methods.* The electrocardiogram of auricular flutter displays ceaseless, uniform, rapid activity of the auricles varying from 250 to 350 beats per minute in untreated subjects. Auricular flutter, though occasionally maintained for years, is usually an unstable condition: a slight increase in the driving rate of the auricles causes a notable mechanical and electrical auricular irregularity, clearly intermediate between fibrillation and flutter (“impure flutter”). Slowing of the flutter rate, for in-

* The presence of auricular sounds in flutter, first mentioned by Lewis,29 may be termed an “auricular gallop.” In many, if not all, instances they are to be considered extra-cardiac in origin and, like the systolic click (systolic ventricular gallop) are presumably caused by pleuropericardial adhesions. At any rate, they are not likely the result of vibrations set up in the fluttering auricle and since they occur during ventricular systole, are not caused by the inrushing blood pounding against the ventricular wall. They are therefore, very different from true ventricular gallop rhythms and from the ventricular sounds caused by auricular contraction in complete A-V block.
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stance by quinidine or Pronestyl, results in electrocardiographic complexes that resemble normal P waves with definite isoelectric (that is, quiet), interauricular intervals. This type of record has recently been redescribed as “paroxysmal tachycardia with A-V block.” 7 A clear differentiation between this disorder and regular flutter may be difficult, but since there are certain unresolved pharmacologic differences between them, it seems unwise at present to consider them identical and the terms interchangeable, as Prinzmetal proposes. 8 Nevertheless, the gradual transition from single ectopic beats to paroxysmal tachycardia to auricular flutter and to auricular fibrillation, first stressed in general terms by Hering in 1900, 9 can frequently be observed in one and the same patient. The undoubted inter-relationship between these irregularities is of no particular value in elucidating the nature of the dysrhythmia.

In recent years, a few advances in electrocardiographic technics have been made that are of some concern to the clinical evaluation of the auricular irregularities. Due to the proximity of certain precordial regions (third and fourth intercostal space at the right sternal border) to the auricular mass, so called unipolar leads from this area (V1 position or one intercostal space higher) demonstrate auricular activity far better than the standard bipolar limb leads, although the direction of the spread of auricular excitation is still best demonstrated in leads I and II (excluding esophageal exploration). As far as P waves are concerned, these leads are not, however, directly comparable to direct leads from the cardiac surface and do not lend themselves easily to accurate measurements of the direction that an impulse assumes over auricular tissue. On the other hand, the use of esophageal leads, at a level of 30 to 40 cm. from the teeth, and of endocardial electrocardiograms places the exploring electrode in close proximity to the auricular musculature and permits an accurate tracing of the spread of auricular excitation. Such leads demonstrate the fundamental pattern of a surface electrogram of muscle tissue with a distinct multiphasic QRS-type deflection (PQRS). The maximum positivity recorded by the exploring contact (the peak of P) closely coincides with the arrival of the impulse at the explored site. This point (the peak of the auricular R wave) has been termed by Lewis the onset of the “intrinsic” deflection, and its usefulness for estimating the time course of an action current as a tool in general electrophysiology considerably antedates Lewis’ experiments. Its value cannot be disputed as long as measurements of the onset of the intrinsic deflection are confined to direct leads from the surface of the heart or leads reasonably similar to them (semidirect leads).

In auricular flutter the individual components of the flutter cycle, as seen in such semidirect esophageal or endocardial leads, differ in no important aspect from the auricular complexes during normal sinus rhythm, another indication of the orderly manner in which individual units pass in and out of the excitatory state, and a demonstration that the differences in the configuration of the P in standard limb leads in flutter as compared with paroxysmal tachycardia and normal rhythm are more apparent than real. In auricular fibrillation the demonstration of an intrinsic auricular deflection can usually be made only from endocardial leads. Clinical auricular fibrillation and auricular flutter have, therefore, become accessible to detailed electrophyslogic analysis which until recently was confined to animal experiments.

THE NATURE OF AURICULAR FIBRILLATION AND AURICULAR FLUTTER IN MAN

Cushny in 1899, 10 and, 10 years later, Rothberger and Winterberg 11 and Lewis 12 were the first to stress the similarity between the clinical syndrome of perpetual cardiac irregularity and the disorder following rapid electrical stimulation of cardiac tissue in dogs, first studied in Ludwig’s laboratory and later extensively analyzed by McWilliam. 13 The overt identity of these two conditions has been accepted, and the results concerning the possible mechanisms of experimental auricular disorders following faradic stimulation, topical or intravenous application of aconitine, acetylcholine, barium chloride, fagarine, veratrine, and other substances in the experimental animal have been
transferred—perhaps too readily—to the spontaneously occurring disorder in man.

Over the years, three etiologic concepts have found widespread support: (1) *Multiple heterotopic impulse formation*, which assumes a complete, functional independence of small areas each responding to its own abnormal impulse center;9, 12, 14, 15 (2) *single heterotopic tachysystole*, by which a single ectopic auricular focus of high inherent frequency will discharge repetitively and, therefore, act as auricular pacemaker;5, 16, 17, 18 (3) *circus movement*,19-24 where either one impulse traverses auricular tissue in a circular fashion with tangential offshoots (Lewis), or where multiple small circuits may be established.19, 21

The first concept (multiple ectopic foci), championed for many years by Lewis, is so closely related to the second that a clear separation of both need not be considered once it is assumed that each center may act as pacemaker by virtue of its frequency. It was based on Engelmann’s original assumption that an increase in excitability of cardiac musculature preceded the onset of fibrillation. It was generally abandoned when it became obvious that a decrease in excitability was an important factor in the causation and maintenance of the disorder. The second and third concept have been considered—perhaps again without full justification—as mutually exclusive, with the British school largely proposing some form of circular excitation, and the Viennese school holding out for a unicellular (?) ectopic focus. Recent experimental work by Rosenblueth and his group, and on the other hand, by Scherf and by Prinzmetal and their associates have renewed the discussion which may be of practical importance from a therapeutic standpoint. The pharmacologic implications of the nature of these and related disorders have recently been presented admirably by Dawes.25

**Is a Circus Movement Possible in Excitable Tissue? Yes**

A circus movement (that is, re-entry of an impulse into an area of tissue that had responded to the same excitation wave before) is an experimentally proven fact. “If a closed circuit of muscle is provided... it is possible to start a wave in this circuit which will continue to propagate itself round and round the circuit for an indefinite number of times.” Such a cyclic recurring impulse propagation has been demonstrated for the following tissues: muscular ring of the jelly fish,26 cardiac musculature of the turtle,19, 20, 26, 27 heart of the electric ray, and frog,19, 21 and large marine loggerhead turtle.21 Common to all these experiments was the simplicity in design of the experiments which allowed the direct demonstration of the “merry-go-round” of either a contraction wave19, 21, 26 or of an excitation wave.19, 23 Furthermore: Two conditions were always present in these experiments: (1) the wave of excitation or contraction was always smaller than the circus path, and (2) the conduction of the impulse or the contraction was forced to proceed in one direction only. This was accomplished by preventing the natural bidirectional spread from the point of stimulation over both limbs of the circle through clamping or otherwise temporarily impeding conduction of a region adjacent to one side of the stimulus point. Thus, an impulse will be prevented from entering one limb of the path but can freely proceed over the other. It will eventually reach the obstructed area, arriving there from the opposite side, and, if the obstruction has by now been released and the tissue has become responsive again, the impulse will traverse through the previously clamped region to the point of origin (the site of the original stimulation), pass beyond this and continue perpetually over the prescribed circus path. Of particular interest in these earlier experiments are the observations by Garrey21 that (1) during repetitive stimulation several such “circus contractions” followed each other, one upon the heel of the other, and that (2) local areas of refractory tissue caused the apparently complete disappearance of the circus contractions, only to have these reappear beyond the blocking area. Garrey assumed that these phenomena were created by failure of conduction in superficial muscle layers forcing the circus contractions through deeper layers from which they emerged beyond the obstructed region.

The important feature of these experiments
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is the presence of local differences in the length of the excitable state and, therefore, in the refractory period resulting in an unequal rate of tissue recovery which is delayed in one limb beyond the time interval separating repetitive stimulating impulses and thereby forces impulse conduction in one direction only. This phenomenon was later shown for heart muscle strips of the turtle by Schmitt and Erlanger who demonstrated the presence of unidirectional impulse conduction and proved it to be a state of conduction impairment preceding complete blockage. Re-entry on this basis, and, therefore, circus movement, is possible even in longitudinal muscle fibers providing they have a certain width: fibrillation is not possible in narrow muscle bridges and for this reason fibrillatory activity is not conducted from auricle to ventricle across the A-V bundle.

McWilliam, Mines, Garrey, and Erlanger all commented on the possibility that such mechanisms may be at play in certain types of extrasystolic disorders, paroxysmal tachycardia, and in fibrillation of the auricles and ventricles. We know that the mammalian heart in situ responds to the same fundamental laws as other excitable tissues. The experiments cited were conducted under artificial circumstances, but their results suggest that re-entry phenomena with consequent circus movement is a mechanism likely to occur in the human heart under appropriate circumstances.

HAS A CIRCUS MOVEMENT BEEN DEMONSTRATED AS THE CAUSE OF CLINICAL AURICULAR FIBRILLATION AND FLUTTER? NO

Sir Thomas Lewis, by inductive reasoning and by a series of limited, now classic, experiments attempted to apply the experimental results of Mines and Garrey to the mammalian heart and to clinical auricular fibrillation and flutter. He traced the excitation wave in electrically induced flutter in dogs by measuring the time interval between the summit of P in lead II and the intrinsic deflection of a direct auricular lead from the exposed heart. The results seemed to him to indicate that in flutter a circus movement would travel around the two venae cavae and that tangential offshoots from the “mother wave” would excite the remainder of auricular and ventricular muscle. A similar, but smaller circular pathway, perhaps around one vena cava only, would account for the basic mechanism of auricular fibrillation. The paths measured were not always in the same region: “In some instances...the wave would appear to circulate, not around the cavae, but in some other ring of muscle, such as that surrounding the mitral orifice; the events vary to this extent.” An additional point deserves emphasis: his measurements revealed an early completion of activation of the “auricle as a whole” during the upstroke of P in the normal heart: “activation is crowded into a very limited phase of the auricular cycle,” while in auricular flutter “...the times of activation are diffused over the whole curve” of the auricular flutter cycle. Since in his experiments the exposure of the left auricle was poor, no direct measurements could be made over a large segment of the proposed circus path. Knowing the conduction velocity, Lewis was forced to calculate the spread over this “blind area” and to estimate the time of arrival at the next measurable point (inferior vena cava). His calculations coincided with the measurements obtained.

In one human subject with auricular flutter, calculations of the instantaneous electrical axes of the flutter cycle obtained in the sagittal plane demonstrated clockwise rotation through 360 degrees for each auricular complex. Lewis recognized the limitations of his carefully conceived experiments but apparently thought that the many, to him interdependent, observations would support his conviction that all instances of auricular flutter and fibrillation were due to a circus movement. Rothberger very soon called attention to the limited number of experiments upon which Lewis’ structure had been erected and objected to the generalizations made from isolated experiments. He criticized the observations on the human heart on the ground that a relatively small circus path could not possibly dominate the auricular surface electrocardiogram and that the calculated axes would have to represent the instantaneous vectorial forces of the entire auricular mass. Lewis’ conclusions from this
example appear certainly invalid since he, in effect, traced the auricular vectorcardiogram in the sagittal plane. As the vectorial forces of normal auricular and ventricular excitation describe a "loop" in space, it is only of interest that the same general order of excitation was present in his subject—an argument more against than in favor of a circus movement and, at any rate, simply an indication that the larger part of auricular tissue was activated in a straight sequential order, a point Lewis did not deny. Similar criticism applies to recent arguments based on like measurements by Dechard, Ruskin and Herrmann.31

The second observation on Lewis' case, the long duration of the auricular excitation in flutter as compared with the duration during normal sinus mechanism seems also to be fallacious. The statement that the "activation of the auricle as a whole...is crowded into a very limited phase of the auricular cycle" is illogical and apparently neglects the activation of the left auricle. It is evident that activation of the right auricle is crowded into the "upstroke and summit of P," but this is followed by the activation of the posterior and left auricular mass, obviously accounting for the remainder of the auricular deflection.32 There seems to be no difference in the length of the excitation of the total auricular mass in the normal as compared with the fluttering auricular muscle.

Recent reports by Grishman and his co-workers33 and by Cabrera and Sodi Pallares34 have attempted to circumvent these earlier difficulties. Grishman's observations are not available for review, and the circus movement demonstrated by Cabrera and Sodi Pallares suffers from the fact that no definite intrinsic deflection can be obtained for auricular tissue in normal rhythm, as well as during auricular flutter, from the precordial region, presumably because the sheetlike auricular tissue is too far removed from the chestwall to allow the recording of truly semidirect leads.35 The combination of endocardial and esophageal leads recently explored by Wenger and Hofman-Credner,35 though again measuring only a portion of the path traversed by the impulse, seemed likewise to support the concept of a circus excitation. These observations are in direct contrast to Prinzmetal's published reports on human auricular flutter.

Lewis' experiments on the exposed auricle of the dog in flutter are of a more direct kind. Prinzmetal has republished one of Lewis' figures in order to demonstrate that the measurements of the proposed circus path were incomplete. This is true, but Prinzmetal did not show the companion figure depicting measurements obtained from the same preparation when the auricles were driven at a slow rate from a point close to the inferior vena cava, which in the flutter experiments was used as the reference point for zero time. When both illustrations are compared, it can be seen that in the normally beating auricle two points a few millimeters apart were activated within 0.014 second and 0.016 second after stimulus. In auricular flutter, when the previous point of stimulation was used as reference, the identical point to the right underwent excitation at 0.031 second (the delay in conduction being the result of the well known decrease in conduction velocity at rapid auricular rates), the identical point on the left at 0.137 second, or ten time later than expected. This, of course, Lewis explained as evidence that the impulse was delayed in reaching this area because it traveled over almost the entire circus path before reaching it from the opposite side, the time lag being consistent with the distance traversed. That this region was excited from two different directions, depending on whether flutter or sinus rhythm was present, was demonstrated by the direction of the steep excitation deflection recorded from paired electrodes placed over this area. In this one example of induced flutter and in four similar experiments a circus movement was likely to be present. Supporting experimental evidence for the presence of a large circus path in auricular flutter was recently advanced by Rosenblueth and Garcia Ramos34 who have introduced a new method of maintaining auricular flutter by crushing the muscular bridges between the venae cavae thus creating an artificial obstacle around which the impulse may circulate perpetually. Their method is rapidly becoming a standard procedure for inducing a maintained flutter in animals. It strongly supports Lewis' experiments.
HAS A RAPID ECTOPIC FOCUS (HETEROTOPIC TACHYSYSTOLE) BEEN DEMONSTRATED AS THE CAUSE FOR CLINICAL AURICULAR FIBRILLATION AND AURICULAR FLUTTER?

The opponents of the theory of circus movement have presented impressive experimental evidence which favors the opinion that at least certain types of the auricular disorder may be the result of rapid repetitive impulse formation arising from a localized focus (tachysystole). For the disorders following faradic stimulation this was first demonstrated by Rothberger and Winterberg.\(^1\) Lately, the topical application of various toxic agents notably acetylcholine, veratrine, and aconitine have been employed to induce auricular fibrillation and flutter in dogs. In a large series of experiments, Scherf and his co-workers have demonstrated that the arrhythmia induced by these agents may be blocked by local cooling, vagal stimulation, or stretching, only to reappear gradually when these procedures were discontinued.\(^2\)\(^-\)\(^4\) Flutter induced from a focus in the auricular appendage was abolished by clamping the muscular connections to the main auricular mass. It is of considerable interest that cooling was effective in blocking the aconitine-induced disorder but not the outwardly similar irregularity caused by faradic or rhythmic electrical stimulation or by the application of acetylcholine.\(^5\) Prinzmetal and his co-workers have presented an equally formidable body of evidence demonstrating that under their experimental conditions no gross circus movement was evident, and that destroying the intercaval area had no effect on the disorder, an observation in direct contrast to Rosenbluth but previously reported also by Brams and Katz.\(^6\)

The inference from these experiments, largely based on irregularities induced by the topical application of toxic agents, is, of course, that (1) no circus movement was observed, and (2) that spontaneous auricular fibrillation and flutter are also caused by a local focus. It is impossible to assess whether the jump to this conclusion is justified or whether the auricular dysrhythmia may not be the end result and the visible expression of various mechanisms.

The different behavior to cooling of variously induced auricular fibrillation led Scherf to the statement “that we are not justified in considering fibrillation to be due to a single mechanism.”\(^7\) The visible blocking of such disorders by cooling or clamping cannot be considered incontestable proof against a circus movement since it has been noted in undoubted circus contractions in turtle muscle strips by Garrey. Prinzmetal’s actual measurements of the path of excitation in aconitine-induced flutter in dogs seems to rule out a circus movement effectively in his experiments. His observations on the human heart, however, are confined to measurements obtained from either esophageal leads or precordial points and are less conclusive since the path pursued by the auricular excitation wave could be followed only over a limited distance.

Since re-entry is possible in excitable tissue, the occurrence of many circus movements of very much smaller dimensions arising in damaged tissue and traversing the syncytial cardiac muscle in an eddy-like fashion remains a theoretic possibility, even if a large circular path in Lewis’ sense should prove a rare occurrence. It might be assumed that the focus itself, and the area surrounding it, stimulated by agents and procedures known to alter conduction velocity and refractory period, might consist of damaged fibers in which excitation re-enters continuously. These possibilities are not excluded in Scherf’s and Prinzmetal’s experiments and are at present beyond the scope of experimental verification. Finally, a small repetitive focus, a large circular excitation with tangential offshoots, and small reverberating waves of re-entry may all have a place in the production and maintenance of the clinical syndrome of auricular fibrillation and auricular flutter.

Since the completion of the manuscript an experimental paper by B. B. Brown and G. H. Acheson has appeared in Circulation,\(^8\) comparing certain characteristics of aconitine-induced irregularities with those obtained by rapid electrical stimulation. The two “flutterers” responded differently to various agents and could be observed simultaneously in one auricle. These observations support the concept that several mechanisms may give rise to similar clinical events, as has been outlined.
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