Atrial Flutter and the Circus Movement Hypothesis

THOMAS LEWIS properly designated atrial flutter as the one disorder of the heart to which Mines' circus experiment would seem especially applicable;¹ he unhappily ignored his own admonition, namely to settle the question of that relationship first, by proceeding early to occupy himself with the much more complex atrial fibrillation. In part for that reason, the question remains controversial. In a recent attempt² to accept at last his advice and commence anew, that is, to consider carefully the "possible relation of circus movement only to flutter,"¹ it was noted that first one must define, or at least describe, atrial flutter.

A "fluttering" in patients' chests long had been recognized as a subjective palpitation prior to the experiments of MacWilliam of which he wrote, "The application of the current sets the auricles into a rapid flutter."³ This is the first objective, if nonspecific, use of the word in medicine. Jolly and Ritchie⁴ next applied the word to a specific electrocardiographic pattern, best described by Lewis: "At each cycle in leads II and III the curve ascends sharply to a blunt summit and returns more gradually. . . . The gentle down-sweep is often noted. In lead I the complexes are usually diminutive. . . . Another and important feature to which I have drawn attention is that the complexes are contiguous: the string is moving constantly and rests for no measurable period on a base line; as soon as one complex is complete the next starts, and this action is continued even throughout ventricular systole. . . . Each complex is a duplicate of the last in form, though this is disguised when ventricular complexes fall with them; the length of the complexes is wonderfully uniform. . . . Another striking feature is that the forms of the auricular complexes in curves taken from different patients usually present a curious and often remarkable resemblance. . . ."⁵ With time and additional electrocardiographic leads, this beautifully precise description has become blurred by a widely prevalent misconception under which many erroneously seem to demand continuous atrial activity in any and every lead even though Lewis, Drury and Iliescu demonstrated an isoelectric line in a lead from manubrium to spine in 1921, in their classic vectorial analysis of flutter, and despite the regular appearance of only "diminutive" complexes in lead I.

Restricting attention to those studies of flutter which meet the criteria of Lewis' description, one may summarize their findings, documented elsewhere,⁶ ⁷ as follows: Data from spontaneous flutter in man and experimental (Rosenblueth-García Ramos) flutter in dogs are in excellent agreement. Left atrial excitation occurs during the prominently inverted wave of lead II, as detected by electrodes in the esophagus or applied to either epicardium or endocardium of the atrium in both man and dog. Left atrial excitation progresses superiorly. Activation then proceeds.
anteriorty and downward over the right atrium (epicardial, endocardial, and precordial exploration in man and dog). Excitation finally returns to its "origin," demonstrably occupying a majority of the atrial cycle in man and all of it in the more easily studied dog. The cycle length increases with lengthening of the presumptive pathway upon enlargement of the central obstacle in the dog or with gross right atrial dilatation in man. In the dog, three independent groups of workers successfully stopped flutter abruptly by section of the presumptive pathway. In man, the validity of vectorial analysis as a representation of the course of depolarization is supported by evidence which includes a close agreement with the results of semidirect and direct atrial exploration described above. In brief, the progression of the momentary atrial vector is counterclockwise in sagittal, frontal, and horizontal planes when these are viewed from the left, front, and above, respectively. At times in the dog and rarely in man, the wave of excitation proceeds in the opposite direction; in such instances, the appearance of leads II, III, and aVF is inverted from the usual form. An appropriate pathway of specialized conduction tissue has been demonstrated anatomically in man, physiologically in animals; at present, however, any relation of such a path to that of the entrapped circuit is purely conjectural.

In contrast, comparable methods applied to aconitine tachysystole in dogs and to spontaneous paroxysmal atrial tachycardia in man show that in each case atrial activation proceeds rapidly and briefly from a point of origin without demonstrable return; much or most of the atrial cycle is electrically silent, with activation confined to the relatively narrow P wave of lead II.

Such considerations, presented in more detail and documented elsewhere,2-6,7 suggest that an entrapped circuit wave (circus movement) is the mechanism responsible for atrial flutter when the diagnosis of this arrhythmia is based upon the classical description of Lewis.

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References
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