A DISCUSSION of the mechanism of auricular flutter or fibrillation requires first a precise definition of what is meant by those terms. They were coined originally to designate more or less characteristic clinical syndromes, but they are now applied loosely to a heterogeneous group of experimental results that may bear only a superficial resemblance to those syndromes. Thus, it is possible to define flutter and fibrillation exclusively on the basis of rate, as was done recently by Prinzmetal and his associates. This definition, however, is undesirable, because it groups together conditions which have quite different features; for example, it does not distinguish fast auricular activity that is stopped by acetylcholine from activity with the same frequency that is not stopped by acetylcholine.

As pointed out by Wiener and Rosenblueth, the structure and properties of auricular muscle are such that they allow for at least two modes of recurrent activity. In the first, impulses start at one or several points, they spread through the muscle and disappear at its boundaries; recurrence cannot take place without reinitiation; we may designate these impulses as beats. In the second, an impulse travels continuously in one direction over a closed circuit and recurs cyclically because the front of the impulse meets always nonrefractory tissue; we may designate this activity as cyclically recurrent or circus movement.

That mammalian auricular muscle is capable of exhibiting automatic beats in the absence of connections with nodal tissue has been shown by Rosenblueth and García Ramos. Two different types of automatic activity were observed, a slow and a fast activity. The differences between the two types refer not only to rate but to other features, in particular to response to the action of acetylcholine. The suggestion that some of the clinical auricular dysrhythmias may be due to ectopic automatic foci has thus a sound experimental basis.

That mammalian auricular muscle may exhibit cyclically recurrent activity has also been demonstrated. The observations of Lewis, Feil and Stroud strongly supported this thesis, and conclusive proof of the development of such activity in appropriate experimental conditions was furnished by Rosenblueth and García Ramos. Multiple recording around an artificial obstacle allowed us to follow the temporal and spatial course of the impulses and made it clear that a single impulse was traveling unidirectionally around the obstacle in one or the other direction. The suggestion that some of the auricular dysrhythmias may be due to a cyclically recurring impulse has thus, in turn, a solid experimental basis.

The problem of the mechanism of auricular flutter and fibrillation is a clinical problem; that is, it can be answered only by studies carried out on patients that exhibit these dysrhythmias. An evaluation of these studies leads to the conclusion that, unless the human auricle differs importantly from that of cats or dogs, there are no data in favor of the view that these perturbed rhythms are due to the discharges of automatic foci. The only two autonomous auricular activities known so far are those described by Rosenblueth and García Ramos. The minimal rate of the fast activity is about 20 per second, much too fast even for auricular fibrillation. Flutter and fibrillation would thus be comparable to the slow activity, but this activity is promptly abolished by the injection of acetylcholine or by vagal stimulation, procedures that do not cancel the dysrhythmias in question.

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Prinzmetal and associates\(^1\) have argued that flutter is due to ectopic discharges because its electrocardiogram is similar to that of hearts treated locally with aconitine, a drug that elicits repetitive discharges. The logic of this argument is faulty. The resemblance is entirely explicable on the basis of a gross correspondence of rate. The cyclically recurrent activity elicited by Rosenblueth and García Ramos also yields electrocardiograms that resemble those of flutter.

There are, on the contrary, many data in support of the thesis that both auricular flutter and fibrillation are due to cyclically recurring activity—i.e., to "circus movements" of impulses. Some of these follow:

a) As pointed out by Wiener and Rosenblueth,\(^2\) in view of the conduction velocity of auricular impulses (0.4 to 0.5 meter per second), of the duration of the refractory period of the muscle (0.1 second) and of the rates of flutter (3.7 to 6.2 per second) and of fibrillation (6.7 to 10 per second), the appropriate perimeters of the obstacles involved, if these activities are cyclically recurrent, should be over 10 and 5.0 to 6.2 cm., respectively; the perimeter of the human inferior cava is about 9.7, and that of the superior cava, about 6.6 cm. The relative constancy of the rates of the dysrhythmias supports the view that these rates are determined not only by the physiologic constants, but also by the anatomic structure of the muscle. The common sequence, fibrillation → flutter → beats, is probably no accident.

b) Acetylcholine accelerates both flutter and fibrillation, much as it accelerates experimental cyclically recurrent activity by shortening the refractory period.

c) The study of the propagation of the flutter wave carried out by Cabrera and Sodi\(^1\) showed a regular successive circular activation on the sagittal plane. These observations provide a direct proof of the cyclically recurrent characteristic of the abnormal activity in the patients they studied.

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The Mechanism of Auricular Flutter and Auricular Fibrillation
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