

## Department of Clinical Reports

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### SHORT PAROXYSMS OF IMPURE AURICULAR FLUTTER PROBABLY INDUCED BY NORMAL SINUS BEATS

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THE occurrence of auricular flutter in patients with complete A-V heart block is not common, but almost thirty such cases, including the first case of flutter ever described, that of Ritchie,<sup>1</sup> have been found in the literature and are discussed in papers by Routier, Mamon, and Le Mant,<sup>2</sup> and by Jourdonais and Mosenthal.<sup>3</sup> Ten more examples of the condition must be added if similar cases reported by Canessa,<sup>4</sup> DiGregorio and Crawford,<sup>5</sup> Israel,<sup>6</sup> Ludwig and Bener,<sup>7</sup> Singer and Winterberg,<sup>8</sup> and the cases of impure flutter and A-V block reported by Solomjany and Boukspan<sup>9</sup> and Lian and Welti<sup>10</sup> are included. We believe that the patient who is the subject of this report is of unusual interest because her electrocardiograms on different occasions showed auricular fibrillation and auricular flutter with complete A-V dissociation, and, on one occasion, very short paroxysms of auricular flutter, apparently initiated by normal sinus impulses, could be observed.

#### REPORT OF CASE

The patient, a housewife of 52 years, entered the hospital Sept. 16, 1938, complaining chiefly of attacks of dizziness and fainting spells which had been associated with bradycardia. She felt well until 1934, when, with the beginning of the menopause, she noticed dizziness for the first time. Her first fainting spell, however, occurred early in 1938, and thereafter the attacks had become frequent and severe. She had been unable to do her housework during the preceding summer.

The past history was of no importance, aside from the fact that she had had a thyroidectomy at the age of 22 years because of "toxic goiter." At this time she noticed weight loss, nervousness, and slight exophthalmos.

Physical examination revealed a well-developed, well-nourished woman who was not acutely ill; the examination was negative except for the features noted below.

The thyroid gland was palpable, and there was a nodule the size of a walnut slightly to the right of the isthmus. The heart was slightly enlarged, with the left border of cardiac dullness 10 cm. to the left of the midsternal line in the fifth intercostal space. On admission, the ventricular rate was approximately 40 per minute, and no irregularities were present. A soft systolic murmur, transmitted to the axilla, was heard at the apex, but no diastolic murmurs were audible. The blood pressure measured 170/70 in both arms.

The urine was normal, and the blood Kahn test was negative. The orthodiagram showed a 38 per cent increase in the cardiac area, and an increase in the

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Received for publication July 8, 1939.

transverse diameter of 18 per cent over the predicted normal (Hodges-Eyester formula). The basal metabolic rate was minus 1 per cent.

During the first few days of her hospital stay she had several minor fainting spells, and electrocardiograms taken during this period (Fig. 1) showed 2:1 A-V heart block and right bundle branch block. She received 0.025 Gm. of ephedrine sulphate and 0.05 Gm. of sodium amytal four times daily, with occasional injections of adrenalin, during this period, but this treatment was not effective, and, on Sept. 20, she became stuporous with a heart rate between 20 and 30 per minute. At this time a number of electrocardiograms were taken, and it will be seen (Fig. 2*A* and *B*) that complete A-V dissociation was present, and that within a period of a few minutes transitions from auricular fibrillation (Fig. 2*A*) to flutter (Fig. 2*B*) and then to slow sinus rhythm, with short paroxysms of what



Fig. 1.—Standard electrocardiograms, taken soon after patient's admission to the hospital, showing 2:1 A-V heart block and right bundle branch block.

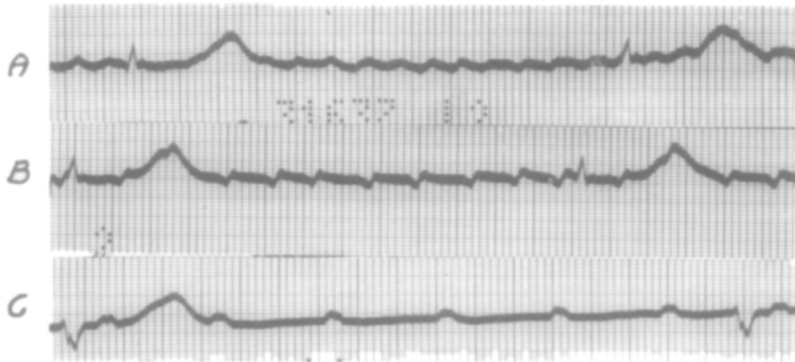


Fig. 2.—*A*, *B*, and *C* are strips of standard Lead II, and show complete A-V dissociation. *A* shows auricular fibrillation, and *B*, auricular flutter. In *C*, normal sinus activity and a slow idioventricular rhythm were present most of the time. Occasional responses to the auricle, not shown in the figure, occurred at this time.

appears to be auricular flutter, occurred (Fig. 3). During the first part of the continuous strip in Fig. 3, a paroxysm consisting of fifteen circus waves is seen, and much shorter runs of the peculiar rhythm are shown in Fig. 3*B* to *E*. In every instance the onset of the circus rhythm was linked by the same interval to a P wave of normal appearance. This unusual auricular rhythm was present for a few minutes only, and a curve taken shortly thereafter (Fig. 2*C*) showed a slow idioventricular rhythm with occasional responses to the auricle. Later, 2:1 and 3:2 heart block appeared.

The patient had many syncopal attacks during the next few days, and, in spite of frequent injections of adrenalin, the ventricular rate varied from 17 to 45 per minute. On Sept. 24, she was given desiccated thyroid gland, in addition to other drugs. Within a few days the ventricular rate gradually increased and her attacks disappeared, and an electrocardiogram taken on October 4 (Fig. 4) showed right bundle branch block with normal sinus rhythm. On October 8, all drug therapy was discontinued, but nevertheless the patient felt well and had no further fainting spells for a period of eight days, at the end of which time she was discharged from the hospital. After her return home she got along satisfactorily for almost three weeks, when, following psychic trauma, the spells of faintness recurred, and she was readmitted to the hospital Nov. 9. While at home she had received no thyroid medication, and was given none until Nov. 26.

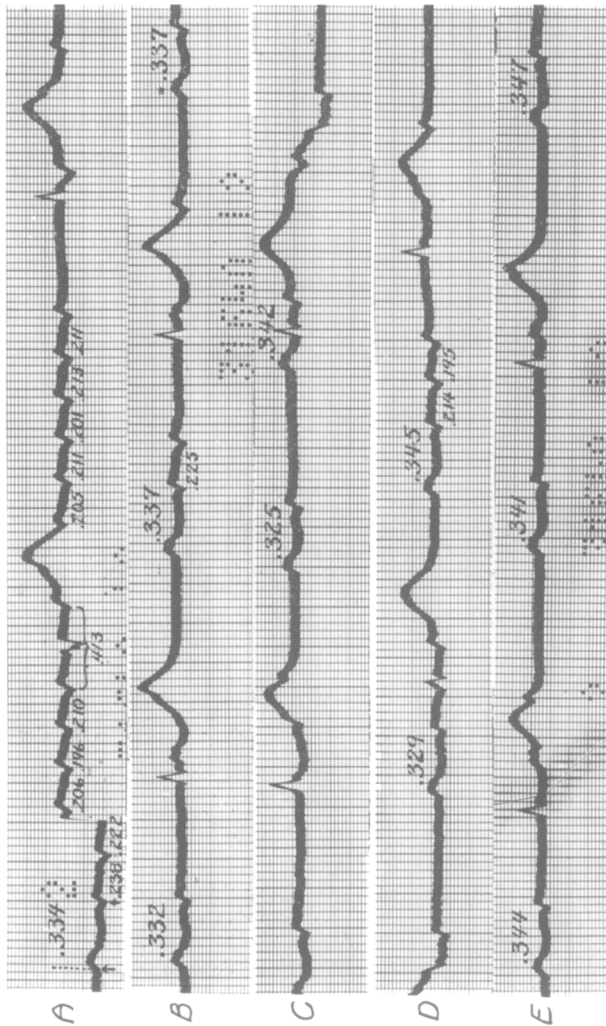


Fig. 3.—Continuous strips of standard Lead II are shown in A through E. The figures marked in ink above the curves represent the time (in seconds) from the beginning of a P wave to the downward peak of the circus wave that follows it. The figures below the tracings give the time between successive circus movements.

During her second hospital stay her heart rate averaged from 40 to 45 per minute before thyroid was given, and 55 per minute thereafter, and no episodes of complete A-V block or abnormal auricular rhythm occurred. The electrocardiograms showed right bundle branch block and 2:1 A-V block, and were similar in all respects to the curve shown in Fig. 1.

## COMMENT

Some of the clinical findings are of particular interest. Without question this patient had organic heart disease, probably of hypertensive-arteriosclerotic origin, but the short periods of complete A-V dissociation and auricular arrhythmia which were observed during her first hospital stay were not precipitated by infection or other obvious cause. Ephedrine and adrenalin were not as effective as usual in establishing idioventricular rhythm and preventing marked bradycardia. Only after she began to receive thyroid extract did her condition improve. It is difficult to be sure that the improvement during thyroid treatment was due to the drug, however, for she was asymptomatic for at least four weeks after the medication was stopped. Willius<sup>11</sup> has pointed out several times that patients with A-V heart block and Adams-Stokes attacks may be greatly benefited by thyroid therapy even when thyroid gland function is apparently normal, and it is possible that thyroid was effective in this case.



Fig. 4.—Standard electrocardiograms, taken ten days after thyroid medication was started. Normal sinus rhythm and right bundle branch block are present.

Short paroxysms of auricular fibrillation or flutter, in man, have been observed by several workers. Wolfert<sup>12</sup> described the electrocardiograms of two patients showing the latter condition and was able to study the effect of vagus stimulation and different drugs on the duration of the paroxysms. In his patients the attacks of circus rhythm were precipitated by auricular premature beats, whereas in two cases reported by Semerau<sup>13</sup> the attacks were thought to be initiated by normal sinus impulses. It is generally believed that, in man, both fibrillation and flutter are usually precipitated by auricular extrasystoles. Such beats cannot induce circus rhythm unless the auricular muscle is in the so-called "fibrillary state," and, although such a condition is most likely to exist at the time of a premature beat, there is no reason to believe that an impulse arising in the sinus node is incapable of precipitating circus movement.

Several features of the tracings shown in Fig. 3 indicate that short paroxysms of flutter induced by normal auricular beats occurred in

our patient. That the oscillations in question were produced by circus movement is suggested by the saw-tooth form of the waves and by their similarity to those seen in Fig. 2*B*. In both figures there are slight variations in the outline of the oscillations and in the auricular rate, so that impure flutter, with changes in the path of the circus movement, was probably present. All of the short paroxysms that were recorded are shown in Fig. 3, and in all instances they follow P deflections of normal outline. Measurements of the time from the beginning of these P waves to the downward peak of the succeeding circus oscillation were made wherever these intervals could be accurately determined. These intervals are indicated above the tracings in Fig. 3, and it will be seen that the difference between the longest (.347 sec.) and the shortest (.325 sec.) of these is only slightly over 0.02 sec. There can be little doubt, in our opinion, that the short paroxysms of flutter were induced by auricular beats arising in, or near, the sinus node.

Experimental evidence supporting the foregoing is found in observations made by Lewis, Feil, and Stroud.<sup>14</sup> They observed that when rapid, rhythmic, induction shocks, applied to a dog's auricle, were increased in rate until evidences of faulty conduction appeared, and then suddenly stopped, aftereffects, consisting of auricular fibrillation or flutter, regularly occurred. The circus rhythm thus induced might be of extremely short duration or it might last for one-half hour or longer. In one animal (dog KD), after rhythmic stimulation, a single afterbeat, believed to represent a single circus movement, occurred. Normal rhythm then returned, but for several cycles each normal beat was followed by an ectopic response, and these premature beats were separated from the normal beats by the same interval as the cycle preceding the single afterbeat. This led the authors to believe that these single premature beats were also caused by circus movement, precipitated, in this instance, by a natural heartbeat.

It is possible that in our patient the auricular fibrillation and flutter which were present shortly before the curves of Fig. 3 were taken had a depressant effect on muscular conduction analogous to the effect of rapidly recurrent shocks on the dog's auricle, and that the paroxysms described are similar to the aftereffects noted in dogs after such stimulation. Few electrocardiograms have been taken immediately after attacks of circus rhythm in man, so that it is not known whether or not these aftereffects are of frequent occurrence. Other influences tending to depress conduction in this patient's heart were probably present during the time when both auricular fibrillation or flutter and complete A-V dissociation were observed, and it is possible that the short episodes of circus rhythm occurred while the state of depressed conduction was passing off.

The paroxysms of circus movement that have been described were of such short duration that there was no opportunity to try the effects of carotid sinus stimulation or of any other procedures. It is possible

that reflex vagal stimulation might have caused longer paroxysms in our patient, as it did in the first case described by Wolferth. Carotid sinus stimulation in this patient on other occasions, however, was without obvious effect upon A-V conduction or upon the auricular rate. Measurements of the cycle lengths during circus rhythm are shown beneath the tracings in Fig. 3. The only paroxysm long enough to enable one to say much about the mode of termination is the first one, consisting of fifteen circus movements (Fig. 3A). In this instance the paroxysm stopped abruptly, without appreciable change in cycle length. In the paroxysm consisting of three cycles of circus movement, seen in Fig. 3D, there is slight reduction of cycle length with the termination. We do not believe there are enough data here to justify speculation concerning the factors involved in the termination of the paroxysms.

## SUMMARY

Electrocardiograms of a patient with transient auricular fibrillation and flutter and complete A-V heart block are presented. Further tracings, taken immediately after the resumption of normal sinus activity, show short paroxysms of what is believed to be impure flutter induced by normal sinus beats. Some experimental evidence tending to support this view is mentioned.

The authors wish to thank Dr. F. N. Wilson for his assistance in the preparation of this paper.

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