"AURICULAR FLUTTER," WITH A REPORT OF THREE CASES.

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In 1911 Jolly and Richie [1] reported a case of Adams-Stokes disease which had been under observation for a period of six years and which showed, in association with the complete heart-block, a very rapidly-beating auricle, the rate of the latter varying from time to time, but usually from 270 to 300 per minute. This condition they called auricular flutter. In adopting the use of the term they refer to the work of McWilliams [2], who, years ago, observed that the application of a faradic current started the auricle into a rapid flutter. In his account he stated that the contractions originated in the stimulated area and extended rhythmically and co-ordinately throughout the tissue. In 1909 Hertz and Goodhart [3] reported a case with an auricular rate of 234 and a ventricular rate varying from 72 to 120. In this case the irregular and varying ventricular rate depended upon a partial heart-block, although this was not recognized at the time. Lewis [4] has since written a very comprehensive article on the subject, in which he reports eight cases which have come under his own observation and eight other cases which have appeared in the literature from time to time, and which, apparently, are all types of this disorder. Hume's [5] case, no doubt, is the same mechanism.
Auricular flutter when first described was considered a very rare condition. As a matter of fact it is probably comparatively common. The reason for its apparent rarity is that it is impossible to detect these extreme accelerations except with the use of graphic methods, and unless a routine examination by these methods is carried out many of the cases will escape recognition. The condition is an abnormal cardiac mechanism characterized by a rapid, rhythmic, co-ordinate contraction of the auricle, the rate usually being somewhere between 200 and 300 per minute. A notable characteristic is the constancy in the auricular rate, any change due to posture or exercise being almost entirely negligible. Some degree of heart-block is usually present; consequently the ventricular rate is slower and may be regular or irregular.

It is a widely-accepted belief that the normal cardiac impulse is brought about by a stimulus which arises in the sino-auricular node, sometimes called the pacemaker, situated near the mouth of the superior vena cava. This node gives rise to impulses in a healthy adult at the rate of about 72 to the minute, the rate being subject to wide variation as the result of exercise, emotion, fever, and various other causes. The details of the mechanism which controls the rate are by no means clearly understood—whether the ordinary accelerations are due to diminished vagus tone or to direct stimulation through the sympathetic or to some other cause, it is not easy to prove; but, at any rate, it seems clear that all these simple accelerations are under the control of this mechanism, whatever it is, and that all the impulses come from the normal point of origin. These have been called tachycardias of a physiological type, because they are simply the normal phenomenon exaggerated.

In auricular flutter the predominating evidence is that the origin of impulse is ectopic; that is to say, some other
point than the normal pacemaker has taken on the function of stimulus production. It is well known that the ordinary premature auricular beat is the result of some pathological, usually ectopic, impulse formation. If a series of these beats arise in quick succession the condition is recognized clinically as paroxysmal tachycardia. This is usually characterized by a rate of 110 to 200—rarely over 180. If, however, this abnormal focus in the auricle gives rise to impulses at a rate greater than 200, the condition has been called auricular flutter. This may seem an arbitrary division, for etiologically and pathologically it apparently does not differ from the simple paroxysmal type, but clinically it differs sufficiently so that it is considered advisable that it be classed in a separate category. It has been found, also, to have a close relation to auricular fibrillation, and when one considers the pathology and mechanism it is easy to see how readily a condition in which there is one abnormal point taking on the function of stimulus production, might pass into a condition in which many foci have appropriated that function.

Some of the reasons for believing that the stimulus which gives rise to this action is ectopic in nature are: First, the already mentioned close relation between this and the isolated premature auricular beat, simple paroxysmal tachycardia and auricular fibrillation. Secondly, that the auricular complex, as recorded by the electro-cardiograph, differs essentially from those which rise from the normal pacemaker. Thirdly, that the rhythm is not under nerve control, as is the normal rhythm, being practically unaffected by posture, exercise or nerve stimulation.

The rate of the auricular beat varies considerably. In the cases collected by Lewis it has varied from 200 to 330 per minute. As already mentioned, this rate is remarkably constant, although observations made at long intervals of
time show some variation in the rate. Observations made on the same day or on succeeding days usually show but little, if any, change.

The ventricular rate is usually slower, due to some degree of heart-block. The most frequent condition is a 2:1 ratio, in which the ventricle responds to each alternate auricular contraction. However, there may be any variation, and instead of 2:1, the response may be at the ratio of 4:1. In either instance the ventricular rate is regular. If, however, as it is now known frequently occurs, the responses are mixed so that a 2:1, 3:1, 4:1, 5:1, &c., may be present, the pulse becomes entirely irregular and may be with difficulty distinguished from auricular fibrillation. The ratio of response may vary with exercise, so that a patient who lying down would have a regular pulse at the ratio of 4:1 might when standing have a regular pulse with a 2:1 response, or a patient with a regular pulse with a ratio of 2:1 while active might have an irregular pulse because of mixed responses when at rest. In most of these cases the original grade of heart-block is increased by digitalis or strophanthus, which suggests that there is probably some impairment of muscle bundle conduction, for it is believed that these drugs have little, if any, influence on the conduction in the normal heart. Following the use of these drugs the regular 2:1 response may be converted into a condition of mixed responses of 2:1, 3:1, 4:1, 5:1, &c. An interesting fact pointed out by Lewis is that in the majority of instances these cases will pass from a 2:1 to a 4:1 ratio rather than from a 2:1 to a 3:1, and if the ventricle is irregular it is likely to consist of mixed 2:1 and 4:1 periods, though, as Case 2 of those I am reporting shows, this is not always true. Isolated 3:1 or 5:1 periods may occur, but they are comparatively rare, and when a 3:1 ratio occurs successively the periods are usually short.
The Polygraphic Tracings.—The first case of Jolly and Richie in which there was complete heart-block and in which the jugular tracings were taken by a Knoll-Hering polygraph showed distinctly the auricular waves. On the other hand, in many cases the jugular tracing is of no value in the diagnosis and may even be of such form as to be entirely misleading. In fact the venous pulse in a 2:1 ratio is likely to be of the ventricular type. Occasionally, when the ratio is 4:1, one may get an auricular wave toward the end of the ventricular pause, and if, as sometimes happens, there is an unusually prolonged pause there may be a series of auricular waves toward the end of it. Sometimes, as in two of my cases, the auricular waves are so prominent as to seriously complicate the c waves, and in the attempt to identify the latter one may get a clue to the condition. The radial tracings are often of much more value in the analysis than the jugular, and a close examination of the arterial pulse curve alone may enable a positive diagnosis to be made. The points to be borne in mind as given in detail by Lewis are: First, that alternation is commonly present; second, that the strength of the beats is substantially influenced by the preceding pauses. This is true, however, only when the pauses are too short for the ventricular contraction to be of maximal efficiency; third, that in the heart-block of flutter there is considerable variation in the As-Vs interval which modifies the expected pauses—a long pause is followed by a shortened conduction interval, and a short pause by a lengthened conduction interval; fourth, that the weak beats are likely to be preceded by a relatively long presphygmic interval. All these factors must be considered and contribute to the difficulties of the analysis. Usually when the ratio of ventricular response is irregular there is a definite grouping of beats, so that groups of three or four beats of irregular lengths may be repeated over and over
again; but in some instances the ratio of response is so mixed that the condition is distinguished from fibrillation with difficulty.

_Treatment._—The general management of the case depends upon the symptoms. If there is evidence of cardiac failure, of course rest in bed is of paramount importance. The latter is just as beneficial in this condition as in any other in which the heart muscle is overtaxed. This was shown in Case II, who had no medication for four days after admission to the hospital, but whose symptoms improved progressively from the time of admission though the pulse remained unchanged. Ordinary sedatives should be used, as indicated in the individual case.

The particularly beneficial drugs, however, are digitalis and strophanthus. In several of the reported cases, during the administration of digitalis the flutter has passed into fibrillation, and when the fibrillation has disappeared the normal rhythm has become re-established. Lewis explains this by saying that fibrillation seems to submerge the abnormal fast rhythm which may not recur when the fibrillation passes off. As has already been stated, digitalis and strophanthus usually slow the heart by increasing the grade of heart-block, so that these drugs may act beneficially in either of these two ways, namely, by causing fibrillation, which may subside leaving the rhythm normal, or by slowing the pulse through increased heart-block.

In Case II a grain and a half of digipuratum was given four times a day for eight days. At the end of that time, as indicated by the tracing taken on March 31, there was some increase in the grade of heart-block causing a slowing of the pulse with some characteristic irregularity. What the dominant mechanism was during the next month is a matter of conjecture, but presumably flutter was persisting all this time, and on April 29 a tracing showed what appeared to
be a 4:1 block. The amount of digitalis the patient had been having at that time is not definite. The patient was put on digitalis subsequent to this, and a tracing taken on May 15 showed that the auricle had gone into fibrillation. The date on which this occurred is not known. Whether the fibrillation will be permanent or whether the normal rhythm will become re-established, as frequently happens, cannot at present be stated.*

I shall report three cases, two of them showing typical polygraphic tracings which, when analysed, leave no doubt that the mechanism present is that of auricular flutter. The third is not so typical, but the evidence that it is a case of flutter is quite convincing. I have not been able to get electrocardiograms, but have had to depend entirely upon the polygraph.

Case 1.—E. C., aged 48. Was admitted to the Rhode Island Hospital, November 20, 1912, and came under my care January 1, 1913. Patient is a native of Rhode Island, and a jeweller by occupation. His family history is unimportant. He gives no history of any previous illness which might have any bearing on his present condition. He drinks considerable alcohol habitually and occasionally to excess. A year before admission he began to get short of breath on exertion. About five weeks ago his legs began to swell, and shortly before admission the swelling had extended to his hands, face and genitals. He had to get up at night to void urine occasionally. He was at work until within a week of his admission to the hospital. On admission there was slight swelling of all of the extremities and of the genitals, with some œdema of the abdominal wall, but no definite signs of fluid in the abdomen. There were moist râles in the bases of both lungs behind. Heart apex was in the fifth space, 8 cm. from the mid-line, and the organ was not definitely enlarged. Sounds were regular and clear,

* The auricle was still in fibrillation, August 9, 1913.
the aortic second slightly accentuated. The pulse was regular, of good volume and tension. Examination of the urine showed a trace of albumen with a moderate number of hyaline and granular casts. The quantity was rather scanty. The functional test with phthalein showed 20 per cent. efficiency. Patient had a good deal of dyspnoea, which came on periodically, some days being much less marked than others. It was relieved with morphine. His condition remained the same for a number of weeks, except that the oedema of the extremities was increasing and the dyspnoea was perhaps more marked. His pulse was noted frequently and was always regular. At the time of the regular visit, on the morning of February 10, it was noted to be rapid and irregular. A tracing of the arterial pulse was taken at that time, portions of which are shown in figs. 1 to 4 inclusive. These tracings show considerable stretches of an absolutely regular pulse at the rate of 143 per minute. Interspersed here and there in the tracing are irregularities of greater or less length. The evidence afforded by analysis is that the auricle is beating at the rate of 286 per minute and that the regular strips are a 2 : 1 response of the ventricle, while the irregularities are markedly irregular, the ratio of 2 : 1, 3 : 1, 4 : 1, 5 : 1 all being present.

While the dyspnoea did not seem any more distressing with the onset of this irregularity, the oedema did increase more rapidly after this rhythm was established and was relieved only by the use of Southey's tubes. In a tracing taken February 20 (see fig. 5) the irregularity was quite different—at this time a very definite grouping being quite persistent. For example, a group of five beats of varying lengths is repeated over and over again, the entire length of the group being \( \frac{19.8}{5} \) seconds. The estimated ratio in these groups is 3 : 1, 5 : 1, 3 : 1, 4 : 1, 5 : 1, which would give an auricular rate on this day of about 308. Occasionally
CASE I.

Figs. 1, 2, 3 and 4 are all a part of the same arterial tracing taken on the day the irregularity was first noticed. The short runs where the pulse is regular is a 2:1 block. The irregularities show ratios of 2:1, 3:1, 4:1, and 5:1. At one place in fig. 4 are four consecutive beats at the ratio of 3:1. The calculated auricular rate is 284.

Fig. 5 was taken ten days after the onset. A group of five arterial beats of varying lengths recurs with great regularity. The estimated ratios here are 3:1, 5:1, 3:1, 4:1, and 5:1, giving an auricular rate of about 308. This, however, cannot be absolutely proven from the tracing. The jugular pulse shows chiefly ventricular waves.

Fig. 6 was taken just after the normal rhythm was re-established. It shows some sinus arrhythmia due to Cheyne-Stokes respiration.
there is a break in this group. This analysis of fig. 5 would seem most probably correct, though the evidence is not absolute.

Patient was given tincture of digitalis mxv t.i.d. from February 20 to March 15. The irregularity continued until March 10, when it was noted at the morning visit that the pulse was regular. Tracing taken at that time showed the rhythm to be normal except for its modification from the Cheyne-Stokes breathing (see fig. 6). The patient is still in the hospital without any very material change in his condition. At times his dyspnœa is marked and at times the Cheyne-Stokes breathing is marked, but there are other times when both of these are entirely absent. The œdema is still quite considerable, and although it cleared up materially with the return to the normal pulse rhythm, it is still necessary to remove some of it by Southey tube drainage.

Summary.—A man, aged 48, suffering from progressive chronic nephritis with paroxysmal dyspnœa and considerable œdema, suddenly developed an irregular heart action after nearly three months in the hospital. This lasted for a month, when the rhythm suddenly became normal. There were no noticeable symptoms with the onset or end of the attack, but the œdema was considerably more marked while the condition persisted. Cheyne-Stokes breathing was present at the time of the return to the normal rhythm. Digitalis in moderate doses of the tincture was being administered during most of the time of the irregularity. The tracings indicate that the irregularity was due to a very rapidly-beating auricle associated with irregular ventricular responses.

Case 2.—W. K., aged 54. Was admitted to my service at the Rhode Island Hospital, March 17, 1913. Patient was a native of Rhode Island and a jeweller by occupation, and came in complaining of shortness of breath and swelling
The physician who attended him before admission said that for a month his dyspnœa had been very marked, and that it was with difficulty that he could go up even a slight incline. The trouble had been coming on gradually for a year, but in November, 1912, he had an attack of bronchitis with a persistent and troublesome cough which aggravated the condition so that there was increased shortness of breath, some swelling of the legs, and considerable dizziness upon exertion. He had to get up at night five or six times to void urine and was troubled a good deal with constipation. His general health, he stated, had always been good, although he had had hay fever in the summer for a number of years. On admission there was a good deal of dyspnœa with orthopnœa, some signs of a slight amount of fluid in the right chest and râles with a distinct pleural friction. The heart was slightly enlarged and action very rapid but regular. There were no murmurs. His blood-pressure while in the hospital varied from 160 to 190. There was marked œdema of both legs with some dermatitis. The amount of urine averaged from 900 to 1,200 c.c. with a specific gravity from 1'009 to 1'012, showing a slight trace of albumen, but no casts.

The heart action was so rapid as to attract attention at once and a polygraph tracing was made. It showed a rate of 138 per minute with marked alternation of the pulse. Patient, at first, received no medication. Two days later another tracing was taken showing a pulse-rate of 136 and still with marked alternation. The venous tracing on this day showed in places auricular waves at the rate of 272 per minute (see fig. 7). On March 22, four days after admission, a third tracing was taken showing the pulse-rate of 137. Patient had been in bed during the four days he had been in the hospital, but was at this time allowed to get up and was given some exercises at the side of the bed and a
(Case II.)

Fig. 7.—Two days after admission. The pulse is for the most part regular at a ratio of 2:1. At two points it suggests a 1:2 and 3:1 ratio. The auricular rate is 272. Alternation of the pulse is well marked.

Fig. 8.—After eight days of digitalis. Showing mixed responses of 2:1 and 3:1 ratio, with short runs of 2:1. Patient left the hospital and was not observed for a month.

Fig. 9.—Tracing made April 29, one month later. The pulse was regular, 82 to the minute, unaffected by posture and exercise.

Figs. 10 and 11.—Tracings made May 15 showing fibrillation.
tracing taken immediately afterward. The rate was absolutely unchanged. On March 23 the patient was given digipuratum (12 gr.) four times a day. This was continued until March 31. Pulse was observed repeatedly and was always found to be regular with the rate practically unchanged. The first irregularity was noted on March 31, after eight days' treatment with digitalis. A tracing made on that day shows a very interesting condition (see fig. 8). There are short runs of beats of the same length as had been present during all the time when he was under observation, but between these runs are periods of irregularity. Considering the condition while the pulse was regular to have been a 2:1 heart-block, as evidenced by the venous tracing, analysis of the irregular curve shows a heart-block of a 2:1 ratio in the regular strips, and mixed 2:1 and 3:1 ratios in the irregular strips. The irregularity is usually made up of groups of three beats, one of which is at the ratio of 2:1, the following two beats at the ratio of 3:1. In other words, when the pulse is regular there are four ventricular beats for every eight auricular beats, while in the irregular periods there are three ventricular beats for every eight auricular beats. Patient left the hospital on March 31, the day on which this tracing was made. He was not seen again until the afternoon of April 29. He had gone to work and had been able to work for a week. His old symptoms, however, returned, so that he had to give up. The oedema on this date was again quite marked. He was sitting up in a chair and had spent several nights sitting up, not being able to sleep. He was having slight attacks of paroxysmal dyspnœa. Cheyne-Stokes breathing of moderate type was present. Patient was able to move about the house without much evidence of shortness of breath. A tracing was taken which showed a regular pulse. Rate 82 to the minute with slight alternation (see fig. 9).
tracing was continued at intervals for about an hour, but at no time was there any irregularity whatever. The rate was absolutely constant. The patient twice got up from the chair, walked about the room, and a tracing taken immediately after showed no change in the rate. The jugular tracing was not very satisfactory, and while there were some extra small waves, there was nothing characteristic enough to make it absolutely sure whether the flutter is still persisting or not. The absolutely persistent rate unaffected by exercise suggests very strongly that the condition was still present and that there was a 4:1 block. Urinalysis; sp.gr. 1.020, trace of albumen with a number of small hyaline casts. Patient was again seen on May 15, and a tracing showed that the auricle had passed into fibrillation (see figs. 10 and 11).

Summary.—A man, aged 54, with gradually increasing dyspnoea and oedema for a year comes under observation with chronic nephritis and an enlarged heart in stage of decompensation and a pulse of 136 to 138 uninfluenced by posture or exercise. Auricular waves are present in the venous tracing at the rate of 272 per minute, indicating a 2:1 block. With rest in bed improvement was steady, but the pulse was not slowed until after eight days of digitalis, when it became irregular, the latter being due to an increased grade of heart-block. Six weeks later the auricle was found to be in fibrillation.

Case 3.—H. F. H., aged 70. Patient is a retired business man, who was first seen seven years ago. He had been a man prominent in affairs, but had a nervous breakdown at about the age of 60 and had given up business. About the same time (ten years ago) he began to have attacks of palpitation of the heart. These attacks would last from one or two hours to perhaps twelve. He was first seen in one of these June 4, 1906. The attack had come on late in the afternoon after nine holes of golf. When seen he was
somewhat apprehensive, the pulse was very irregular and the rate 100. There was no shortness of breath, but a sense of pressure in the præcordium and a slight tendency to cough. He went to bed and slept well. The following morning the pulse was 84 and regular. The physical examination showed nothing which would seem to have any definite bearing on the attacks. There was slight emphysema of the lungs. Heart was not definitely enlarged. Blood-pressure 100. Arteries hardly so thickened as usual at that age. Urine showed a few small hyaline casts, but otherwise practically normal. The second attack in which he was seen on September 1, 1906, came on apparently as the result of some disagreeable news. Heart was irregular as before, and the rate 100. His statement at that time was that these attacks were occurring every month or two, but he was seen in them only occasionally. The third attack on which notes were made was on November 30, 1908. This came on while in the bath-room in the morning. The explanation given by the patient was that he had had some indigestion the night before, with considerable gas in the stomach and intestines. The pulse was irregular as before, but in a couple of hours was regular and 70.

Patient was seen from time to time, but no note was again made until July 25, 1911, on which date he was seen just following an attack of palpitation which occurred the night before, and which disappeared before morning. He said then that he was having attacks about once a week. They would come on suddenly and leave suddenly. In the meantime, however, as far as his heart was concerned, he was not troubled, and was able to climb hills and stairs easily. Patient continued to have these attacks of palpitation at frequent intervals. Early in 1912 he was given $\frac{3}{4}$ gr. powdered digitalis three times a day for a week at a time, and would then omit it for a week. He continued that
Fig. 13 is a continuation of fig. 12. This tracing was started during an attack on October 8, 1912. A preliminary arterial record was being made before attempting the venous curve, and it was at that time that the normal rhythm became re-established. At the time of the transition from the abnormal to the normal rhythm there was a standstill of the heart for 5-2 seconds. Just before the transition is a period of definite spacing from which the auricular rate might be about 330.

Figs. 14, 15 and 16 are all parts of a tracing during an attack on October 15. They show the transition from the abnormal to the normal in both the arterial and venous curves. They show in many places that what would seem to be the c waves do not coincide with the corresponding upstroke in the arterial curve. The jugular curve also shows many accessory waves besides the c and v waves which are at the rate of about 330 a minute.
quite faithfully throughout the year 1912. He was much better, and was quite free from attacks from February until September. Between September 20 and November 8, 1912, he had five attacks, and has had none since.* In four of these attacks I saw him, and in two of them I was able to get a polygraph tracing, in each of which the transition from the abnormal to the normal rhythm is shown. The tracings were examined only casually, and the condition considered one of paroxysmal fibrillation. Because the tracings had rather more than usual interest as showing the transition from the abnormal to the normal rhythm, they were sent to Dr. Lewis, who made a careful analysis, and ventured the statement that he believed the condition a case of flutter with extremely complex responses, giving as his very convincing reasons: First, the variable relation between the arterial upstroke and what was apparently the corresponding c upstrokes; second, the numerous waves in the jugular tracing in addition to the c and v waves; third, a tendency to spacing, which is very definite just before the long pause in one of the radial curves, and, in addition, the frequent recurrence of beats of the same length; lastly, that the rate of waves in the venous curves in certain stretches is about 330 per minute, too slow for fibrillation, and just the rate calculated for flutter.

Summary.—A retired business man, aged 70, with neurotic tendencies, under observation for seven years, has had frequent attacks of palpitation for ten years. These followed a nervous breakdown. The attacks come on at irregular intervals every few weeks, and are characterized by a slight sensation of oppression in the praecordium, apprehension, tendency to cough, and an irregular pulse of

* He had an attack lasting about five hours on August 3, 1913. None occurred in the interval between November 12 and August 3.
about 100 per minute. There is no marked disturbance of the circulation. The onset and end of paroxysms are generally definitely recognized unless the end comes during sleep. Between attacks there is no evidence of any cardiac or renal insufficiency. Patient thinks they usually come on as the result of some emotion or indigestion associated with gas. He has been on small doses of digitalis for about fourteen months. During that time he had five attacks between September 20 and November 8, 1912. He has had no others. Four of these were observed, and in two a polygraph tracing was obtained showing the transition from the abnormal to the normal rhythm. This transition is marked by a very long standstill of the heart, in one instance more than five seconds. Twice at the change patient has mentioned a feeling of faintness and a sensation of things growing black.

The pulse tracing is very irregular, but there is occasional evidence of spacing, and the c waves in the venous curve are complicated by a waves, so that often the former cannot be identified. There are occasional runs of auricular waves at the rate of about 330 per minute.

REFERENCES.


DISCUSSION.

Dr. HIRSCHFELDER (Baltimore, Md.): I am very much interested in the cases which Dr. Fulton has reported, as I have for a number of years been interested in these conditions upon the border line between the normal rhythm and auricular flutter. In the cases which Dr. Fulton has reported he gave us some very interesting examples
of auricular fibrillation block, but we cannot always be certain in the tracings, on account of the spacing, whether we are dealing with a block or with a sudden change in the rate of the auricle itself. One sees that very well exemplified if one changes or alters the high tense of irritability of the auricle in an animal by faradizing, by stimulating it with faradic stimulant, as, for example, by applying it to the top of the auricular appendix. The state of flutter and the state of fibrillation are apparently a quantitative relation as regards irritability rather than qualitatively different. That is, if one makes use of a graded stimuli one finds that with the mildest stimulus to which one can get response one obtains occasionally extra response. If one stimulates with a still greater intensity one obtains a sudden doubling of the rate. I was particularly interested in this question when I first began to investigate the condition in 1906, because at the time the dominant theory was the theory of August Hoffman. As I say, if one stimulates the auricle with faradic stimuli which are far too frequent to give rise to response to the individual stimuli, one obtains a gradation from extra systole to a paroxysm, but not an absolute sudden doubling of the rate, and upon cessation of the stimulus one finds a sudden return of the auricle as well as of the ventricle to a normal rate. [Remarks uncorrected.—Ed.]

Dr. FULTON: I was very glad to hear Dr. Hirschfelder's remarks. There were a good many points which it was impossible for me to bring out in the time allotted. I emphasized particularly the analysis of the radial curve, because I am convinced that many cases of this condition can be diagnosticated without any mistake from the radial curves alone. I admit, as he says, that there are other conditions which might bring about a somewhat similar appearance, but with careful analysis I think there is no question but that the radial alone is of great value in making the diagnosis, and the reason that it is of value is that the cases have been studied with the electrocardiograph and the polygraph, so that the tracings which are so clear with the electrocardiograph may now be identified by the use of the other instrument. The question of extra systole is of course one to be considered. I do not believe that if Dr. Hirschfelder had an opportunity of analysing the cases I have that he would think there is any possibility of these tracings being produced by extra systoles. The condition is closely related to fibrillation, the essential difference being that in flutter the stimulus production is confined to one abnormal point, while in fibrillation it occurs in a number of points in the auricle.