

PAROXYSMAL AURICULAR FIBRILLATION *

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The abnormalities of the cardiac mechanism which are responsible for various forms of cardiac arrhythmia and disturbances in cardiac rate are now well understood, and have been especially well demonstrated by means of the string galvanometer. The analysis of disturbances of the cardiac mechanism, although of ever increasing diagnostic and therapeutic importance, is a field for original investigation which is fast losing its fertility. On the other hand, large problems lie before us in determining the essential causes of these cardiac disturbances. The progress of the treatment of cardiac disorders will be accelerated by the discovery of the now unknown changes, either within the heart or outside of it, which are responsible for these abnormalities of the cardiac mechanism. This is especially true of the large group of disorders characterized by disturbances in the generation of stimuli of the heart beat. These disturbances may predominate either in the auricles or in the ventricles, and may result in the occurrence of premature ectopic beats (extrasystoles), tachycardia of ectopic origin, the so-called auricular flutter or auricular fibrillation. These disturbances may occur in hearts already the site of anatomical lesions, but they may also occur in hearts which give no signs of such lesions.

It is with the hope that some light may be thrown on this question of the essential cause of disturbed excitability or stimulus formation that the following case of paroxysmal auricular fibrillation is reported.

CASE REPORT

History.—I. G., Hospital No. 601, a jeweler, aged 50, born in Russia, was admitted to the hospital Feb. 23, 1912, complaining of cardiac palpitation and weakness. He knows of no cases of cardiac disease or rheumatism in his family, and the family history is otherwise negative. The patient had been a generally healthy man, but always "high strung" and rather nervous. He thought that he had neither scarlet fever nor diphtheria in childhood, but since the age of twelve he had frequent attacks of tonsillitis, and at the age of 15 typhoid fever. When 18 years old he had an apparently rather mild attack of gonorrhea, but there never had been any other venereal affection. His wife is living and well, and they have several healthy children. He had considerable distress after eating for many years, and had always taken large amounts of food, especially bread, and much tea. He used both alcohol and tobacco moderately and had

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never done laborious work. He never had rheumatism, but there was pain without swelling or redness in the right shoulder, beginning about eleven years ago and lasting fifteen months. He continued his occupation as a jeweler with very little interruption up to a few weeks before admission to the hospital.

The present illness began twelve years ago, in 1900, when the patient felt undue fatigue after exertion, and on the following day he had his first attack of cardiac palpitation and weakness, much like that for which he came to the hospital. He was in bed for three days at this time. He then had no such symptoms for about eighteen months, when the second attack occurred, and since then he has had similar attacks, usually lasting only a few hours, every four to six months. Occasionally they have lasted as long as three days. The patient thinks that the gastric disturbance, noticed especially after eating, had something to do with causing the attacks of palpitation. About eight weeks before admission the patient had the first of the series of attacks which brought him to the hospital. The first attack began Dec. 22, 1911, in the usual way, with a boring feeling in the epigastrium, a thumping feeling now and then in the heart, and finally a sudden onset of cardiac palpitation. There was also a feeling of pressure about the chest, and choking, headache and weakness. This attack lasted twenty hours and stopped suddenly. During the next three weeks the patient had two or three attacks of lesser severity, and then, Jan. 13, 1912, an attack began which lasted three and a half days. This was followed by four days when the patient felt quite well, at the end of which time the symptoms returned and continued for seven days. Then he was free from symptoms for ten days, when another attack, lasting a few days, occurred. Finally, after the patient had been feeling well for nearly two weeks, the final attack before admission began, and this time in an unusual way, a feeling of palpitation coming and going for a day or so, and then settling down on February 22, the day before admission, to what seemed to the patient a severe attack. Besides the symptoms that have been mentioned he had moderate pain in the left arm and in the back of the neck. He was able to walk into the hospital, but with some difficulty.

Physical Examination.—Physical examination showed the patient to be a rather poorly nourished, small man, who looked at least ten years younger than his age. He was lying quietly and apparently comfortably in bed, but appeared somewhat nervous. He was alert and highly intelligent. The eyes showed normal pupillary reactions to light and accommodation. The fundi were apparently normal except for slight tortuousness to the arteries. The tonsils were not enlarged. There was slight venous pulsation in the neck. The thyroid was not palpable. The lungs yielded normal physical signs throughout. There were no abnormal pulsations seen over the thorax, while there was a general heave in the region of the left nipple. The precordium was not prominent. The apex beat was in the fifth space, 10 cm. to the left of the midsternal line. The outline of cardiac dullness extended 4.0 cm. to the right of the midsternal line in the fourth space, and 11.5 cm. to the left of the fifth space. The heart sounds were clear throughout, no murmurs being heard. The second pulmonic was slightly accentuated, and had a liquid quality. The radial pulse was distinctly irregular, and had a rate of about 75 per minute. Many of the pulsations were well sustained, but they were irregular in both force and rhythm. The actual heart-rate as counted by auscultation over the heart was 126 per minute. The arteries were distinctly palpable in the arm, at the wrist and especially at the elbow. The temporal arteries were also indefinitely palpable. The abdomen was normal. Neither liver nor spleen was palpable. There was no edema of the extremities. Blood, hemoglobin 95 per cent. (Sahli) corrected reading = 119 per cent. Wassermann reaction was negative. The urine showed neither albumin nor casts. Its specific gravity was 1.030. The amount was 650 c.c. in twenty-four hours. Electrocardiograms showed that auricular fibrillation was present (Fig. 1.)

Subsequent Course.—Digitalis was begun three days after admission, and in three days distinct slowing of the ventricles had occurred and the arrhythmia was much less marked. March 1, at 10:30 a. m., the patient was bled from an arm vein and 40 c. c. of blood was obtained. At 11 a. m. he noticed a sudden change in the feeling of the heart beat, and declared that his attack was over. The examination revealed that the patient's heart was beating regularly, at first at a rate of 108, and in a few hours at about 90. The outline of relative cardiac dullness extended 4 cm. to the right in the fourth space, and 10 cm. to the left in the fifth space. The heart sounds were clear and of good quality all over the precordium. There were no murmurs, but the second aortic sound was accentuated. The patient felt immediately distinctly more comfortable, and seemed to be entirely well except for slight weakness. Electrocardiograms showed that the normal sequential beat had returned (Fig. 2).

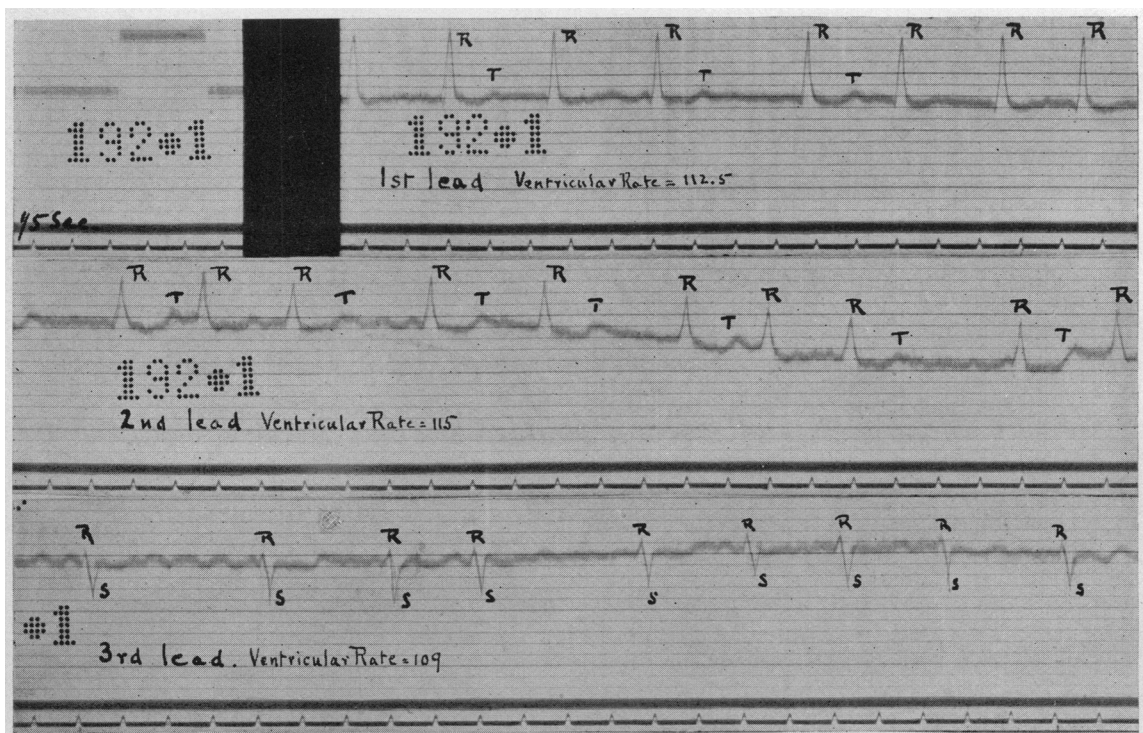


Fig. 1.—Curves 192. February 25. First, second and third leads, showing auricular fibrillation. Ventricular rate averages 112 beats per minute. The time marking is always in 1/5 seconds.

During the night of March 6, after the heart had been beating normally for about sixty hours, the patient felt an occasional jumping feeling in the epigastrium, which he said usually occurred before attacks, and which led him on this occasion to predict the return of cardiac palpitation. During the night it was noticed that cardiac arrhythmia existed for a few minutes on several occasions.

The heart continued to beat regularly, however, until March 10, when the patient awoke at 1 a. m. with the sensation of weakness and felt that his heart was beating irregularly. When examined at 9:30 a. m. the typical arrhythmia of auricular fibrillation was present, the diagnosis of which was confirmed by

electrocardiograms. This arrhythmia continued until the afternoon of March 14, nearly five days. On this day the patient was restless, worried and especially uncomfortable in the morning, but in the afternoon the heart suddenly, practically instantly, became regular. The patient immediately felt much better, and except for muscular soreness and weakness was without symptoms. The cardiac examination gave results similar to those obtained during his first period of cardiac regularity. His condition remained excellent, and on March 22, after the heart had been regular for eight days, the patient was allowed to sit up out of bed for half an hour. March 23, a few minutes before 7 a. m., the pulse became suddenly irregular again. When seen a little later the patient was comfortable, and the heart, although completely irregular, was not beating very rapidly, and nearly all beats produced palpable radial pulsations. The heart sounds were clear. The arrhythmia was still present on March 25, when the patient complained of feeling especially badly, with dizziness, weakness and a

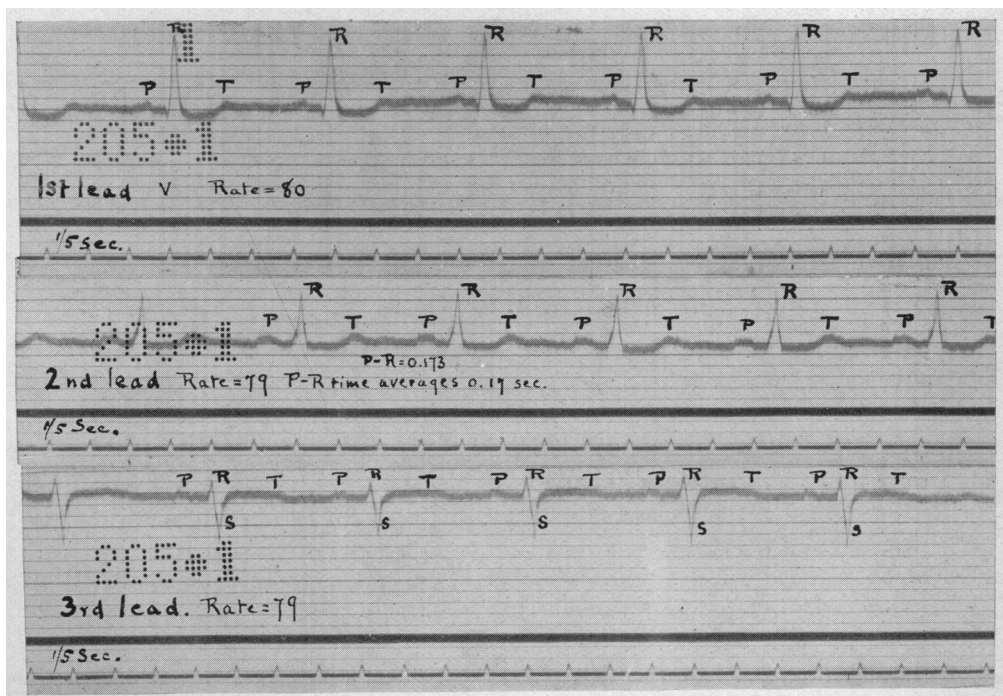


Fig. 2.—Curves 205. March 4. First, second and third leads obtained during the first period of cardiac regularity. The records show that the normal sequential beat is present. P-R time, second lead, average 0.17 second. Rate averages seventy-nine beats per minute.

feeling of helplessness. On March 26, at about midnight, the patient awoke to find that his pulse was regular and rapid, and when examined the next morning the heart was beating perfectly regularly as before. The outline of relative cardiac dullness extended 2 cm. to the right and 11.5 cm. to the left in the fourth space, and the heart sounds were as before. The cardiac rate was 80. Electrocardiograms again showed that the auricles were beating normally, and were once more properly coordinated with the ventricles. The patient seemed in excellent condition without symptoms, and the heart maintained a rate of about 72 for

several days. During the night of April 1, however, the patient had pain in the epigastrium and a feeling of suffocation, and on April 2, at 6 a. m., his pulse became suddenly irregular. When examined at 10 a. m., the heart was beating very irregularly, at a rate of about 140 per minute. Many beats failed to reach the wrist, and the radial pulse-rate was only about 100 per minute. The outline of relative cardiac dullness extended 3.5 cm. to the left in the third space, and 3.0 cm. in the fourth space, and 10.0 cm. to the left in the fourth space. The electrocardiograms were those of auricular fibrillation. From April 2 the cardiac arrhythmia continued as long as the patient was under observation. The heart rate gradually slowed and became more nearly regular, but electrocardiograms showed that auricular fibrillation was still present when he was discharged on May 23, feeling quite comfortable and well. He had been walking about the ward for several days without symptoms. The arrhythmia continued for several weeks after the patient left the hospital. It was subsequently learned that the patient died about two months later with symptoms suggesting acute dilatation.

DISCUSSION

During the time the patient was in the hospital he had three periods of nine, nine and six days, when the heart was beating regularly, between which auricular fibrillation occurred for five and four days. It was also present during the first six days and during the last fifty-one days in the hospital.

The change from one form of cardiac mechanism to another was usually sudden, but premonitory symptoms were sometimes noted by the patient before the onset of the arrhythmia. These were, as have been described, a feeling of gastric distress and a thumping feeling in the cardiac region which was probably caused by the occurrence of premature ventricular contractions (extrasystoles). The occurrence of such beats was recorded by electrocardiograms several times during periods of fibrillation. The actual onset of auricular fibrillation occurred each time after the patient had been asleep, once apparently during sleep. On the first occasion the patient awoke at 1:00 a. m. with a sensation of weakness and felt that his heart was irregular, and the second and third occasions the arrhythmia began suddenly at 7:00 a. m. and 6:00 a. m., respectively. Muscular exertion and psychic excitement, unless it was in dreams, played, therefore, no rôle in initiating auricular fibrillation. Gastric disturbance also did not seem to be a factor at such times. Thus, there was no evidence supporting the patient's statement concerning the influence of gastric distress on the cardiac rhythm, which may be a factor in the production of complete arrhythmia, as v. Müller¹ has pointed out.

Auricular fibrillation changed to the normal sequential beat three times while the patient was in the hospital. The first change occurred at 11:00 a. m., half an hour after 40 c.c. of blood had been withdrawn from an arm-vein, the second during the afternoon following an uncomfortable and restless forenoon, while the third change occurred at midnight,

1. Müller, v.: The Nervous Affections of the Heart, *THE ARCHIVES INT. MED.*, 1908, i, 1.

apparently during sleep. The changes were always sudden, and as the conditions under which they occurred were so different, no conclusions as to the possible cause of the changes can be drawn. The blood-letting was thought to have been a possible factor, but it was repeated on April 22, after fibrillation had been present three weeks, without any change in the cardiac mechanism. The patient stated that the return to the regular rhythm usually occurred when he was quiet.

The administration of digitalis may have been a factor in causing the disappearance of auricular fibrillation on two occasions. The drug was begun in the form of digipuratum tablets, 0.1 gm., three days after admission, five tablets the first day, decreasing one tablet each day. On February 29 the patient was nauseated after having had 1.5 gm. of digitalis, and the drug was discontinued at 3:30 p. m. The fibrillation ceased at 11:00 a. m. the next morning.

The drug was begun again in the same manner on March 11, two days after the onset of fibrillation, and continued for four days, when it was stopped after the sequential beat had returned. Small doses of the tincture, 0.2 c.c., three times a day, were given during this period of normal rhythm with the idea that it might assist in maintaining the regular beat, but on March 23 fibrillation again set in, and digipuratum was begun as before. This was continued for two days, and was discontinued after ten doses, as the patient complained of feeling dizzy and weak. Thirty-six hours after drug was discontinued, the normal beat again returned. The drug was withheld after the next onset of fibrillation had been present fifteen days, because it was felt that its administration probably had no effect in stopping the auricular fibrillation, and that the sequential beat would return spontaneously. As this did not occur, digipuratum was again administered. At this time and again later, it was given until nausea occurred, but produced no apparent effect on the auricular fibrillation. Digitalis was given primarily not with the idea of influencing the auricular activity, but because the rapid, irregular and insufficient ventricular activity present at the time of admission demanded it. In fact, as Mackenzie² and others have pointed out, digitalis may be apparently a factor determining the onset of auricular fibrillation. In our case fibrillation seemed prone to cease a day or two after the drug had been pushed to its physiological limit and discontinued. On one occasion fibrillation came on while small doses of the tincture were being taken.

The ventricular activity was favorably influenced by digitalis, and the rapid, irregular heart beat was reduced to a rate of about 80, and became almost regular, while fibrillation continued. During the periods of regular cardiac activity the electrocardiograms showed that there was no prolongation of the time of conduction from auricles to ventricles, the

2. Mackenzie: *Digitalis, Heart*, 1910-11, ii, 295.

P-R time in the second lead records averaging 0.17 seconds. This case does not lend support, therefore, to the suggestion put forward by Lewis³ "that the slowing of the heart, when auricular fibrillation is present and digitalis is given, is due to an increase of a previously existing defect in the conduction to the ventricle of those impulses which are built up rapidly and irregularly in the auricle."

BLOOD-PRESSURE

The relation of changes in blood-pressure to changes in the cardiac mechanism was studied. The difficulty of determining the blood-pressure in cases of auricular fibrillation is evident, as the ventricular contractions, varying in force, produce variations in the blood-pressure from beat to beat. On account of the fact that exact estimations were impossible, the blood-pressure was not recorded at first, during auricular fibrillation, but later when the heart was beating more nearly regularly, the pressure was determined by the auscultatory method, at which the strongest beats produced sounds in the brachial artery and also at which practically all beats produced sounds. The average of these two readings was considered as the systolic blood-pressure. In a similar way two diastolic readings were taken and averaged. The blood-pressure readings are seen in the accompanying chart (Fig. 3). Although the blood-pressure was constantly high, marked changes took place, the systolic pressure varying from 125 to 215 mm. Hg. The diastolic pressure was more constant, but varied from 85 to 120 mm. Hg. In the second period of regular rhythm, from March 14 to 23, the blood-pressure rose gradually from 125 mm. Hg to 175, when fibrillation again set in. The blood-pressure did not fall with the onset of fibrillation, but twenty-four hours later it had fallen 25 mm. Hg, and remained relatively low until about thirty-six hours after the heart had become regular once more. Then the systolic pressure rose to 185 mm. Hg, remained high for three days, and then fell to 130 the day before fibrillation again set in. The blood-pressure was taken between 10:00 a. m. and 11:00 a. m. each day, and no observations were obtained immediately before and after a change in cardiac rhythm, so the day-to-day readings give no accurate indications as to changes in blood-pressure as a factor in causing changes in the cardiac mechanism. On the hypothesis that the high blood-pressure might be a factor in maintaining fibrillation, however, the patient inhaled 0.2 c.c. of amyl nitrite about three hours after the onset of fibrillation on March 23, at which time the systolic blood-pressure was 180 mm. Hg. He became nervous and dizzy, but except for the initial rapidity the heart beat did

3. Lewis: *Auricular Fibrillation and Its Relation to Clinical Irregularities of the Heart*, Heart, 1909-10, i, 306.

4. Lewis: *Fibrillation of the Auricles; Its Effects Upon the Circulation*, Jour. Exper. Med., 1912, xvi, 395.

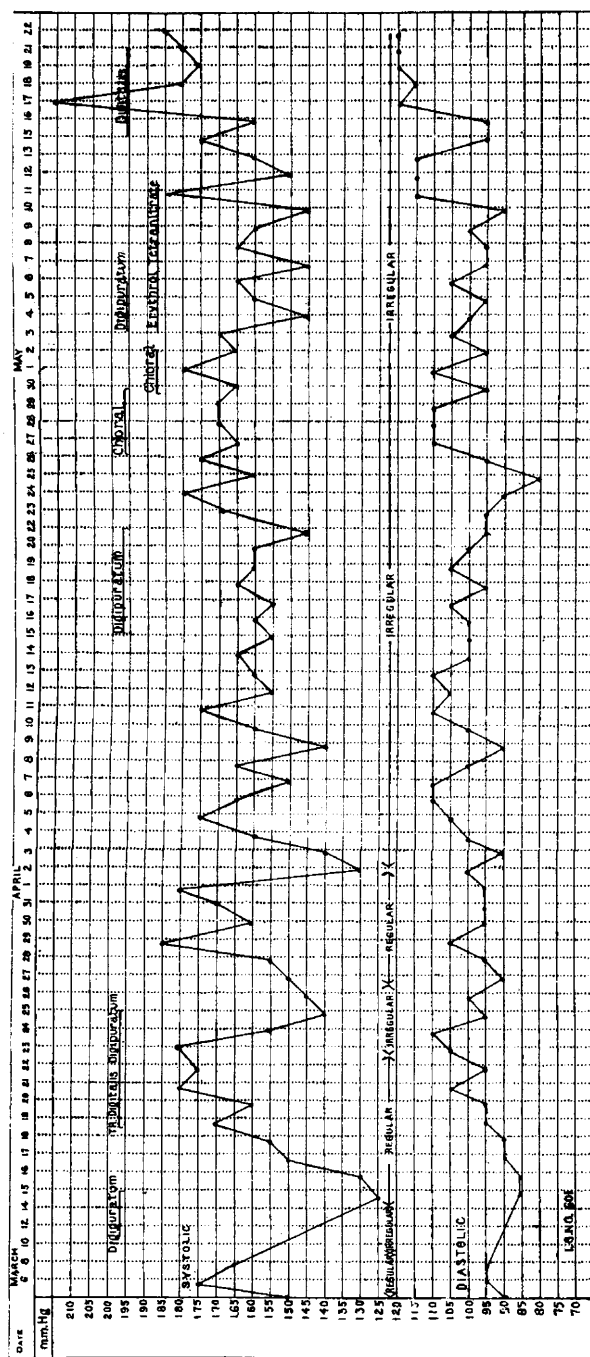


Fig. 3.—The systolic (upper line) and the diastolic (lower line) blood-pressure. The type of cardiac activity and the drug administration are also indicated.

not change, and the blood-pressure was apparently not lowered. On another occasion (April 22), three weeks after the onset of fibrillation, the systolic pressure fell during the operation of blood-letting to 115 mm. Hg without any change in the cardiac mechanism. Erythrol tetranitrate and chloral were also administered during fibrillation, but without affecting appreciably the blood-pressure or the cardiac mechanism. On the other hand, the systolic pressure reached 215 mm. Hg on one occasion during fibrillation without any change in the beat. It may be said, therefore, that no relationship between changes in blood-pressure and changes in the cardiac mechanism can be made out. The blood-pressure varied greatly, both during auricular fibrillation and during the normal rhythm, but the blood-pressure was maintained generally at a high level, regardless of the form of cardiac activity present. These blood-pressure observations are in accord with those made by Lewis⁴ on the experimental animal, in which he found that the onset of fibrillation was usually accompanied by a fall of blood-pressure, but the effect was inconstant. The circulation tended to accommodate itself to altered conditions, and to return to the same blood-pressure as that observed before the onset of fibrillation.

The effect of pressure on the neck over each vagus nerve was recorded by electrocardiograms several times, both during the normal rhythm and during fibrillation (Figs. 4-7). The abnormal susceptibility of the heart to mechanical stimulation of the vagi was striking. The length of time pressure was made over each nerve and the length of the responding pause may be tabulated as follows:

AURICULAR FIBRILLATION (FIGS. 3 AND 4)

	Pressure Over Right Vagus Seconds	Ventricular Stoppage Seconds	Pressure Over Left Vagus Seconds	Ventricular Stoppage Seconds
	3.0	3.3	3.4	4.8
	3.4	4.0	3.1	4.0
	2.8	4.0	3.6	4.4
	3.0	3.1
	3.0	3.4
Average	3.04	3.56	3.37	4.42
Proportion . . .	1	1.17	1	1.22

NORMAL BEAT (FIGS. 5 AND 6)

	Pressure Over Right Vagus Seconds	Ventricular Stoppage Seconds	Pressure Over Left Vagus Seconds	Ventricular Stoppage Seconds
	1.4	3.2	2.0	3.6
	1.9	3.0	2.0	4.0
	1.9	3.0	2.0	1.9
Average	1.73	3.06	2.0	3.17
Proportion . . .	1	1.77	1	1.53

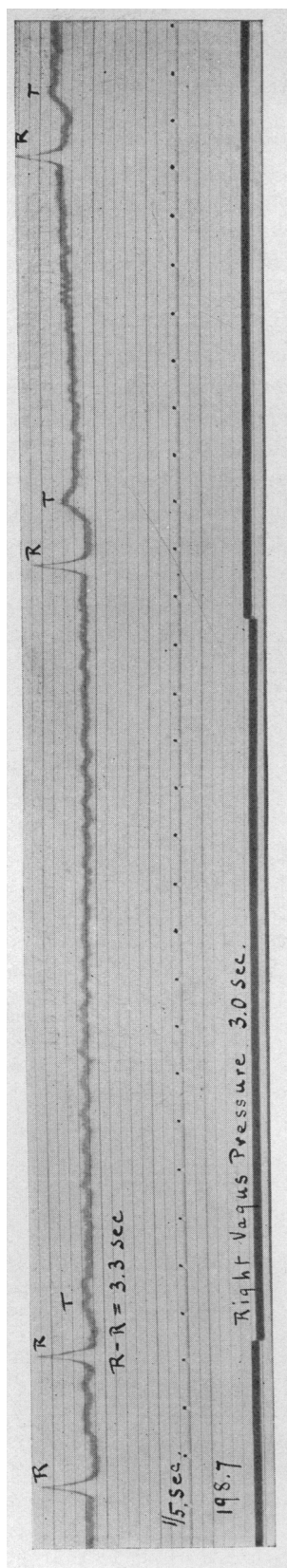


Fig. 4.—Curve 198.7. February 28. The effect of pressure for three seconds over the right vagus during auricular fibrillation.

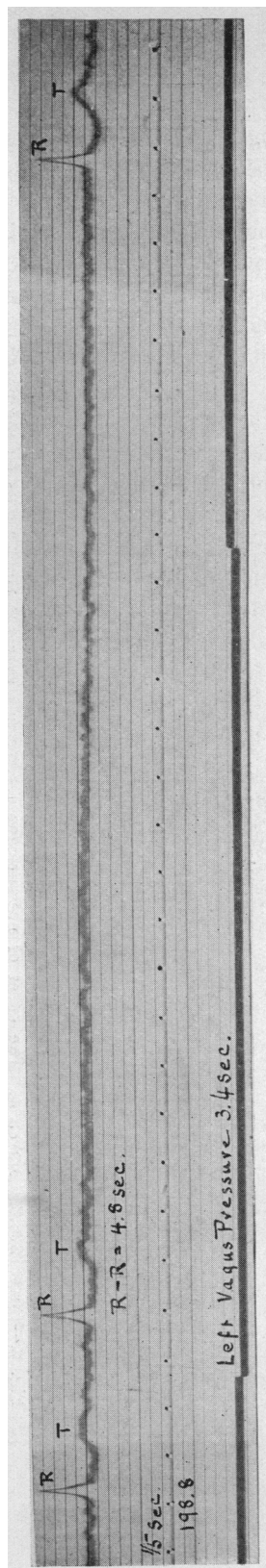


Fig. 5.—Curve 198.8. February 28. The effect of pressure for 3.4 seconds over the left vagus during auricular fibrillation.

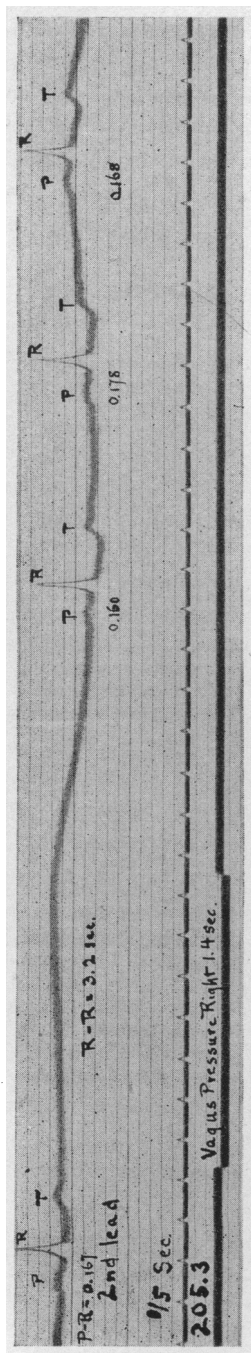


Fig. 6.—Curve 205.3. March 4. The effect of pressure for 1.4 seconds over the right vagus on the normally beating heart.

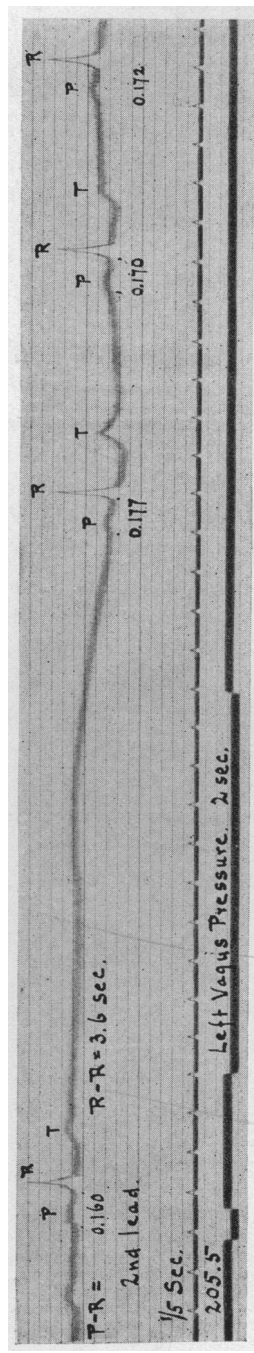


Fig. 7.—Curve 205.5. March 4. The effect of pressure for 2 seconds over the left vagus on the normally beating heart.

Thus it is seen that neither during auricular fibrillation nor during the normal beat can any definite difference be made out between the effects of right and left vagus pressure, such as can sometimes be demonstrated in man (Robinson and Draper⁵), and as can be almost constantly shown with faradic stimulation of the nerves in the dog (Cohn,⁶ Robinson⁷). The ventricles were stopped for a longer time proportional to the length of pressure when the heart was beating normally than when auricular fibrillation was present, whether the left or the right nerve was pressed on.

The mechanism responsible for the ventricular pauses which followed vagus pressure during auricular fibrillation is apparently different from that which brings about the stoppage of the normally beating heart. The electrocardiograms indicate that the activity of the fibrillating auricles was unaffected by pressure over the vagi, while they show that the normally beating auricles were brought to rest by this procedure. The manner by which vagus pressure brings about the long cessation in the ventricular activity in cases of auricular fibrillation has been discussed by Robinson and Draper,⁵ and it was concluded that the ventricular pauses are apparently due to the blocking of stimuli from the auricles. There was no evidence as to whether this striking effect depends on an increased susceptibility of the conducting fibers to vagus stimulation or whether it depends on the stimuli from the fibrillating auricles being of such a nature that only slight lowering of the conductivity succeeds in blocking them. The possibility must also be considered of some quantitative change in the auricular activity which is not to be detected in the electrocardiograms, but which makes it incapable of stimulating ventricular contractions.

When the heart was beating normally there is practically no evidence that pressure over either vagus nerve lowered the conductivity, as the P-R time was never prolonged beyond 0.21 second after the cardiac stoppage in any of the records. The marked effect on the rate of the whole heart is in sharp contrast to the effect on conduction. It may be, however, that this effect on rate is responsible for the lack of effect on conduction, for during the cardiac standstill the conductivity of the heart is freed from the influence of functional fatigue, and so has its normal capabilities at the end of the cardiac pause. The harmonizing of the effects of vagus pressure on the heart during auricular fibrillation with that observed when the heart was beating normally is difficult.

5. Robinson and Draper: I. Studies with the Electrocardiograph on the Action of the Vagus Nerve on the Human Heart, *Jour. Exper. Med.*, 1911, xiv, 217; II. Studies with the Electrocardiograph on the Action of the Vagus Nerve on the Human Heart, *Jour. Exper. Med.*, 1912, xv, 14.

6. Cohn: *Jour. Exper. Med.*, 1912, xvi, 732.

7. Robinson: The Influence of the Vagus Nerves on the Faradized Auricles in the Dog's Heart, *Jour. Exper. Med.*, 1913, xvii, 429.

The marked susceptibility of the heart to mechanical stimulation of the vagi may have been another expression of the cardiac abnormality which caused the auricular fibrillation. The hearts of patients with auricular fibrillation are often markedly affected by pressure over the vagi and an abnormal response to this procedure by a normally beating heart may possibly indicate that auricular fibrillation is more likely to occur in it than in a heart less affected by vagus pressure. It is difficult to say whether this abnormal response indicates a so-called heightened "vagus tone" or whether the heart itself is responsible for the marked response. A series of experiments were undertaken with the hope primarily that some new light might be thrown on the question of the relation of vagus activity and auricular fibrillation, but the results of the experiments⁷ do not afford any positive evidence as to the rôle the vagi may play as a causative factor of auricular fibrillation in man. The dog's auricles may be thrown into fibrillation and tachycardia by direct faradization as readily after the vagi are cut as before, but vagus stimulation holds the auricles in their abnormal state of activity after it has been once set up by faradization.

The effect of atropin in our case was observed only during fibrillation, and after 0.9 mg. atropin had been given hypodermically the ventricular rate increased markedly from 84 to 158 beats per minute, recorded by an electrocardiogram taken forty minutes after the administration of atropin. The waves of fibrillation were not so distinct in the records obtained during the rapid ventricular rate as before. The great increase in ventricular rate following atropin indicates that the vagi were constantly strongly active at this time in controlling the ventricular rate. A desire not to disturb the normally beating heart prevented the use of atropin during the periods of regular rhythm.

A series of electrocardiograms were made directly from various points on the chest wall during the normal beat and during fibrillation. The technic described by Lewis⁸ was followed, in the hope of gathering further evidence regarding the significance of the waves of auricular fibrillation in the electrocardiograms. The results were unsatisfactory, as neither the P waves nor the waves of auricular fibrillation were distinct.

When this patient is considered from an anatomical rather than from a physiological viewpoint, it is found that the heart is normal as regards its size, position and the character of the heart sounds. No valvular murmurs were ever heard, and although the second aortic sound was somewhat accentuated, the muscle sounds were of good quality. The outline of cardiac dulness was measured repeatedly, and no changes in

8. Lewis: The Origin of the Electric Oscillations and the Direction of Contraction of the Ventricle in Instances of Complete Irregularity of the Heart (Auricular Fibrillation), *Quart. Jour. Med.*, 1911, v, 11.

size as those observed by Lewis and Schleiter⁹ could be made out, as an accompaniment of the changes in mechanism. A slight increase in the size of the heart between the time of admission and of discharge was noted, however, the total width increasing 1 cm. during this time.

The constantly high blood-pressure and the palpable arteries in the arms and temporal regions led to the diagnosis of arterial sclerosis. The urine gave no evidence of diseased kidneys, as the usual tests were repeatedly negative except for several hyaline casts on two occasions. The functional activity of the kidneys was tested May 7 by the technic of Rowntree and Geraghty,¹⁰ 6 mg. of phenolsulphonephthalein being injected intramuscularly. The first appearance of the drug in the urine was not definitely determined, but was estimated at about ten minutes. During the first hour 36.8 per cent. and during the second hour 21.9 per cent., or a total for two hours of 58.7 per cent. of the drug was excreted. Thus the functional activity of the kidneys was but slightly below the normal limits of excretion (40-60 per cent. the first hour, and 60-85 per cent. in two hours). The sole definite anatomical lesion, therefore, is apparently arterial sclerosis.

Arterial sclerosis is frequently a prominent characteristic of cases of auricular fibrillation. Thus Lea¹¹ found it present in 57.1 per cent. of the non-rheumatic cases in his series. No definite change in the cardiac muscle has been described in these cases with sufficient frequency to allow any conclusions to be drawn as to an anatomical basis for auricular fibrillation, and the post mortem examinations of some of the hearts in which fibrillation had been present reveal hearts which are anatomically sound (Gossage and Hicks¹²). Lewis³ believes that it is changes in the walls of the auricles themselves which probably cause fibrillation, and he has expressed the opinion that anemia of the auricular walls may be a factor in setting up fibrillation. Changes in the blood-supply of the auricular walls may well have been an important factor in causing the attacks of fibrillation in our patient. The evident arterial sclerosis and the high but markedly changing blood-pressure might readily have caused abnormalities in the cardiac blood-supply, which not only rendered the auricles more prone to fibrillation and abnormally susceptible to vagus stimulation, but also furnished the immediate cause of an attack of auricular fibrillation by a temporary diminution of the cardiac circu-

9. Lewis and Schleiter: The Relation of Regular Tachycardias of Auricular Origin to Auricular Fibrillation, *Heart*, 1912, iii, 173.

10. Rowntree and Geraghty: An Experimental and Clinical Study of the Functional Activity of the Kidneys by Means of Phenolsulphonephthalein, *Jour. Pharm. and Exper. Ther.*, 1909-10, i, 579.

11. Lea: Some Points in Relation to the Etiology of Auricular Fibrillation, *Quart. Jour. Med.*, 1911, iv, 423.

12. Gossage and Hicks: On Auricular Fibrillation, *Quart. Jour. Med.*, 1913, vi, 435.

lation. There is very little in support of this hypothesis, but arterial sclerosis and high blood-pressure have been prominent in several cases of transitory auricular fibrillation which have been observed. Thus Hornung¹³ reports three cases in which these abnormalities were prominent features, and arterial sclerosis was also mentioned in two of Fox's¹⁴ six cases. The case reported by Lewis and Schleiter, although only 28 years old, had somewhat thickened arteries, but on the one occasion recorded the blood-pressure was low.

The paroxysms of auricular fibrillation may occur over a period of many years, and our patient had them apparently for twelve years. One of Hornung's cases suffered with attacks of cardiac arrhythmia for five years and another for at least ten, while one of the cases reported by Fox, the case originally observed by Cushing and Edmunds, had attacks for over twenty years. These cases, therefore, do not always deserve the bad prognosis which is usually accorded to auricular fibrillation. They may have anatomically normal hearts and be closely allied to cases of paroxysmal tachycardia, as Lewis and Schleiter point out. The prognosis may not necessarily be more grave than in tachycardia. However, the auricular fibrillation tends to become permanent, as Fox's cases well illustrate, and this is what happened in our case, as fibrillation was present during the last two months that he was in the hospital, and continued until his death about two months after his discharge. There is, therefore, apparently a group of cases which may be termed paroxysmal auricular fibrillation, in which arterial sclerosis may be an underlying cause of the abnormal cardiac activity, and in which attacks of fibrillation may recur over a period of many years in patients with anatomically normal hearts, without great impairment to the general health.

SUMMARY

A case is reported in which transient attacks of auricular fibrillation were observed and recorded by electrocardiograms. These attacks had apparently occurred over a period of twelve years, and auricular fibrillation became permanent while under observation. No definite cause could be discovered as initiating the attacks. No definite relation could be established between changes in blood-pressure, which was always abnormally high and variable, and the changes in the cardiac mechanism. The heart was strikingly susceptible to pressure over each vagus nerve, both during fibrillation and during the normal cardiac activity. This abnormality may be another expression of the changes causing auricular fibrillation. Electrocardiographic records of these vagus effects were obtained.

13. Hornung: *Über atypische Tachykardische Paroxysmen*, Deutsch. Arch. f. klin. Med., 1907, xci, 469.

14. Fox: *The Clinical Significance of Transitory Delirium Cordis*, Am. Jour. Med. Sc., 1910, cxl, 815.

The heart of the patient gave no signs of an anatomical lesion, but arterial sclerosis, a frequent accompaniment of auricular fibrillation, was present. The presence of this lesion and the high, inconstant blood-pressure suggest the possibility that changes in the cardiac blood-supply may have been the underlying causative factor in this case. Other cases showing attacks of auricular fibrillation occurring over a period of years have been reported in which high blood-pressure and arterial sclerosis were prominent features. These cases seem closely allied to paroxysmal tachycardia, and although the auricular fibrillation tends to become permanent, the prognosis for them is not necessarily so grave as that which is given in ordinary cases of auricular fibrillation with outspoken cardiac lesions.

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