FIBRILLATION OF THE AURICLES: ITS EFFECTS UPON THE CIRCULATION.*

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PLATES 43 AND 44.

A little more than two years ago, while working at a peculiar form of heart irregularity exhibited by human subjects, which had been described by Mackenzie[1] as "nodal rhythm," I was able to bring forward considerable evidence[2] to show that the irregularity in question results from fibrillation of the auricles. The evidence for that conclusion was based upon a comparison of curves taken by several methods from the human subject and the experimental heart. The similarity of electrocardiograms taken from patients, and from dogs in which auricular fibrillation had been induced, was noticed almost simultaneously by Rothberger and Winterberg.[3] A fuller analysis of these electrocardiograms supported the hypothesis; and the contention was finally proved by the observation that the oscillations characterizing the clinical electric curves arise from the auricular portion of the heart, and especially by the observation that a similar irregularity occurs in the horse and that this irregularity may be shown by inspection of the heart in that animal to result from auricular fibrillation.[4] Briefly, it has been shown that the commonest form of irregular heart action in man is due to this disturbance of the auricular mechanism. The irregularity is of

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[1] Mackenzie, J., *Quart. Jour. Med.*, 1907–8, i, 39.

[2] Lewis, T., *Heart*, 1910, i, 306; *Brit. Med. Jour.*, 1909, ii, 1528.

[3] Rothberger, C. O., and Winterberg, H., *Wien. klin. Wchnschr.*, 1909, xxii, 839; *Arch. f. d. ges. Physiol.*, 1910, cxxxi, 387.

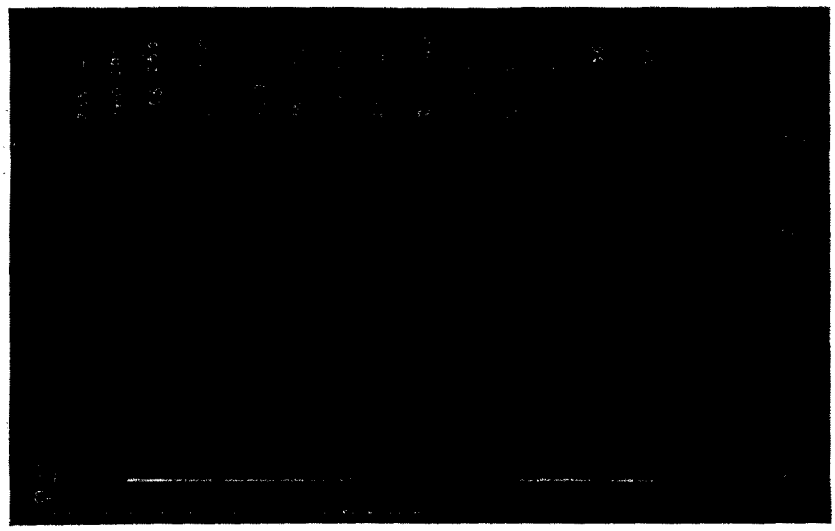
[4] Lewis, T., *Verhandl. d. deutsch. path. Gesellsch.*, 1910, xiv, 112; *Heart*, 1912, iii, 161.

this form in 70 per cent. of all cases of heart failure, as I have been able to demonstrate by electrocardiographic curves taken from over a hundred patients. The conclusion is now generally accepted.

The disturbances of the circulation are so profound in the human subject, when this curious disorder of the heart's action begins, that further study of them seemed desirable. With this object in view, I have undertaken the experiments described in the present communication.

PRELIMINARY OBSERVATIONS UPON METHOD.

The observations have been carried out upon a number of cats and dogs; the former were anesthetized with urethane and ether, the latter with morphia, paraldehyde, and ether. Fibrillation of the auricles was induced by faradic stimulation of the right auricle. In order that the heart should be surrounded by conditions as natural as possible, the stimulation was performed with the chest wall restored, wherever the circumstances of the experiment permitted such restoration. A small window was made in the chest wall over the right auricle, and small flexible electrodes of the fish hook type were fastened into



TEXT-FIG. 1. Cat 30. A kymographic curve, showing the effects on arterial and venous blood pressures of faradization of the right auricle. The actual readings of the venous manometer are written on the curve. A subtraction of 174 mm. must be made to arrive at the actual venous pressure in mm. of magnesium sulphate (specific gravity, 1,046).

TABLE I.

Arterial Pressure.
The Effect of Auricular Fibrillation in Cats. The Figures Represent the Actual Rises and Falls.

Cat.	Onset.	After first few seconds.	After second few seconds.	Offset.	Remarks.
28	−24	+10	+10	0	
	−36	+16	0	+20[5]	
29	0	0	0	0	
	−10	0	+ 4	+ 6	
30	+20	− 8	0	−12	Rate during fibrillation 102 only.
	−25	+ 8	+ 8	+16	
	−24	+10	0	+ 8	
32	0	0	0	0	
	−20	+18	0	+ 8	
31	− 2	0	0	+ 2	Heart exposed.
	−12	+ 6		+ 6	
	−16	+12		+ 4	
41	+12	0	0	−12	Rate during fibrillation 128 only.
	−44	0	0	+38	
	−30	+15	+15	0	
	−32	0	0	+30	
42	−12	+18	0	+ 6	Heart exposed.
	− 2	+14	+ 4	− 4	
	+10	0	0	−10	Rate during fibrillation 193 only.
43	−48	+28	+12	+ 8	Heart exposed.
	−36	0	0	+20	
44	−18	+ 8	0	+10	Heart exposed.
	−30	+20	+ 2	+ 8	
45	−28	+14	+10	+10	Heart exposed.
	−34	+20		+18	
46	+10	0	0	−10	Rate rise of 55 beats per minute. Heart exposed.
	−30	0	0	+30	
	−40	0	0	+40	
47	− 6	0	0	+ 6	Heart exposed.
	−42	+40		0	
	−12	+ 8		+ 6	
48	−16	0	0	+10	Heart exposed.
49	−10	0	+ 4	+10	
50	−14	0	0	+10	Heart exposed.
51	−18	+10	0	+ 6	Heart exposed.
	−20	0	0	+20	
52	−40	+10	+ 2	+24	Heart exposed.
53	−28	+ 8	+ 2	+14	Heart exposed.

[5] The pressure fell 36 mm. at the onset, rose again 16 mm., ran horizontally, and rose 20 mm. at the offset. Thus it returned to its original level.

the auricular substance. The electrode wires were rubber coated and passed between the layers of the chest wall. Any air which had entered the pleura was withdrawn, the window in the chest wall being closed.

CHANGES IN MEAN ARTERIAL PRESSURE.

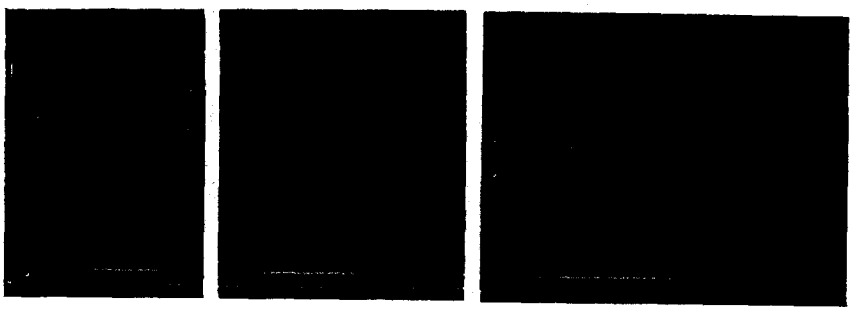
The following observations are based upon manometric readings in five dogs and thirty-seven cats.

TABLE II.

Arterial Pressure.

The Effect of Auricular Fibrillation in Dogs.

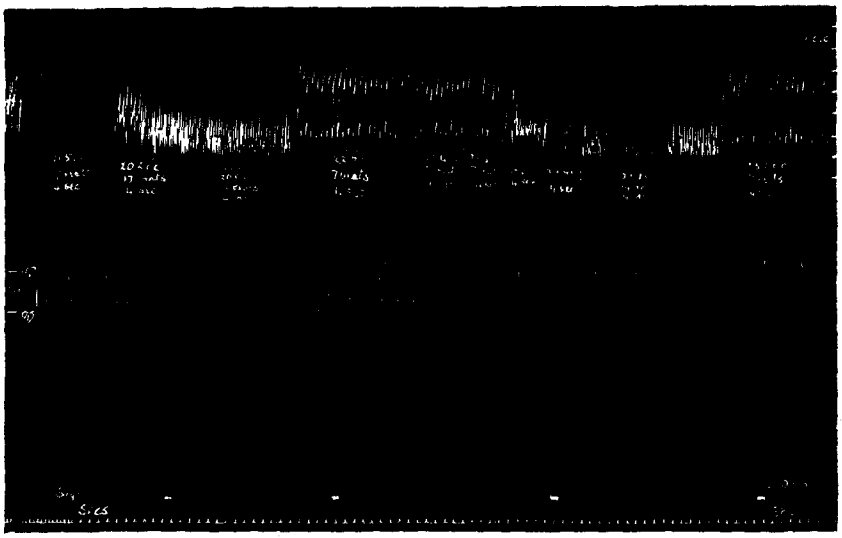
Dog.	Onset.	After first few seconds.	After second few seconds.	Offset.
CP	+ 9	0	0	−10
CL	−30	0	+8	+20
	−40	+12	0	+12
CK	−44	+34		0
	−82	+36	0	+30
CO	+ 9	0	0	− 9
	−15	+21	−6	0
CN	−30	+16	0	+12
	−28	+22	0	0



a *b* *c*

TEXT-FIG. 2, *a*, *b*, and *c*. Three portions of a tracing showing arterial pressure and heart volume curves taken from a series of observations. The first curve (figure 2, *a*) shows a rise of mean arterial pressure at the onset of fibrillation, an increase of cardiac output and a decrease of cardiac volume. The second curve (figure 2, *b*), in which the blood pressure has fallen, shows a deep fall of arterial pressure, a fall of cardiac output, and a slight and progressive increase of cardiac volume. In the third curve (figure 2, *c*), in which the arterial pressure has fallen still lower, a fall of blood pressure occurs at the onset of fibrillation and it is accompanied by a decrease of cardiac output, and by a considerable and progressive increase of cardiac volume. With the rise of pressure in figure 2, *a*, the heart action is far more regular than with the falls of blood pressure in figure 2, *b* and *c*.

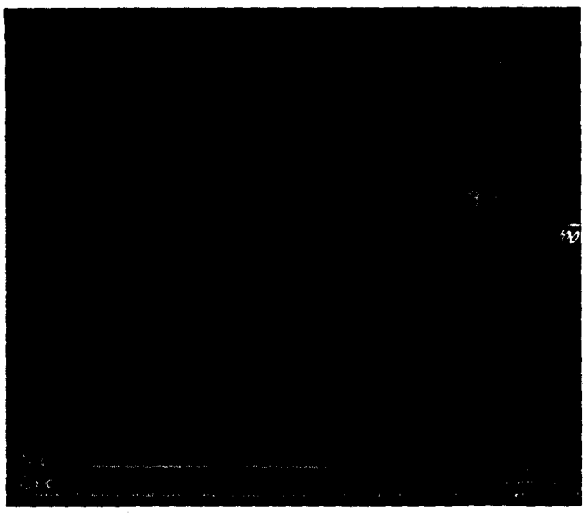
The effect of auricular fibrillation, and its accompanying phenomena, is very varied in experiment. At its onset, the mean arterial pressure may remain unchanged; it may rise at once (text-figure 2, *a*); but much more commonly there is an immediate fall (text-figure 1). The greatest rise has been noted in a cat, and amounted to twenty millimeters of mercury; the greatest fall occurred in a dog, and amounted to eighty-two millimeters of mercury. The fall which is customary in both cats and dogs is usually twenty to forty millimeters of mercury. When an immediate



TEXT-FIG. 3. Cat 42. Curves of arterial pressure and heart volume, showing the effects of auricular fibrillation. The output and number of heart beats per four seconds are marked from time to time along the upper or cardiometer curve. The output varies in the same direction as the blood pressure. In the cardiometer curve, systole is indicated by a downstroke. Note the diminution of heart volume during the period of fibrillation.

rise occurs, it may be succeeded by a fall of less degree within a few seconds; when an immediate fall occurs, it is usually succeeded by a rise of less degree within a few seconds. Thus the circulation tends to accommodate itself to the altered conditions and this is especially so when there is no initial fall of

pressure. In most experiments where there is a fall of pressure,—and these are in the majority,—a quick return towards the previously recorded pressure is observed, as I have stated; the original



a

b

TEXT-FIG. 4, *a* and *b*. Figure 4, *a* (cat 33), gives arterial pressure and intestinal volume curve, showing the effects of faradization of the right auricle. Figure 4, *b* (dog CP), gives curves of arterial pressure and intestinal volume, showing the effects of faradization of the auricle. During the period of fibrillation the mean blood pressure is raised and the intestinal volume is increased.

pressure may be regained or surpassed (text-figure 3); commonly the rise becomes slower and a distinct shoulder is present in the curve (text-figures 1 and 3), which then runs more or less horizontally or rises gradually. The commonest type of arterial curve consequently consists of an abrupt fall, a preliminary and steep rise of about a third or half the extent of the former, and finally a horizontal or a gradually rising curve, the latter spread over a few seconds or a minute. Eventually the pressure lies within five to fifteen millimeters of the original pressure, and when, or shortly after stimulation of the auricle ceases, the pressure returns to normal. This return is abrupt, and often goes beyond the normal limit (text-figure 4, *a*), returning again within a few seconds. If the fibrillation is maintained for five minutes or more, the arterial pressure has usually risen to within five or fifteen millimeters of the original level, though it may remain lower.

Tables I and II give selected figures of the arterial pressure changes in the eighteen cats and five dogs.

CHANGES IN VENOUS PRESSURE.

The venous pressures have been taken from the femoral vein by means of a manometer containing magnesium sulphate in solution (specific gravity, 1,046). The tip of the venous cannula was passed into the femoral vein so that its mouth lay in the iliac vein; and the readings were taken by noting the position of the meniscus in relation to a millimeter scale, and were recorded in writing on the curve at intervals of a few seconds. All experiments in which no movements of the meniscus with respiration were discerned have been discarded.

The changes of venous pressure when the auricle passes into fibrillation are slight. They have been determined in a number of observations upon five cats and two dogs, and the observations permit certain general statements to be made. When the arterial pressure is maintained at the original level, the venous pressure also remains unchanged. A rise of venous pressure is the customary event, for there is usually a fall in arterial tension; and the latter being usually twenty to forty millimeters of mercury, the former generally amounts to five to ten millimeters of magnesium sulphate solution. The arterial and venous pressures move in opposite directions, and this relation is generally maintained not only during the preliminary, but also in the later stage of an observation. Thus,

if the arterial fall is deep and the recovery prompt, the venous pressure after first rising falls again; and when at the offset of fibrillation the arterial pressure reaches its original level, or when it is restored during the fibrillation period, the venous pressure also falls to normal. While these general rules hold good, there are natural exceptions from time to time, for the fluctuations of venous pressure as a result of the change in cardiac mechanism are usually small, and the venous pressure shows variations which are independent of the nature of the heart beat (tables III, IV, and V).

TABLE III.

CAT 47.

Arterial pressure in mm. of mercury.	Venous pressure in mm. of magnesium sulphate.	
82	72–75	
80	72–75	
		Auricles fibrillated.
52	82–92	
52	82–87	
74	77	
80	72	
80	72	
		Normal rhythm restored.
80	72	
78	72	
78	72	
78	70–72	
78	70–72	
		Auricles fibrillated.
60	82	
56	87	
70	92	
78	77	
76	75	
76	75	
75	75	
76	72	
76	77	
		Normal rhythm restored.
78	74	
78	60–72	
78	60–72	
79	60–72	

TABLE IV.

CAT 30.

Arterial pressure in mm. of mercury.	Venous pressure in mm. of magnesium sulphate.	
132	91	
	81–90	
	81–90	
120	80–86	
		Auricles fibrillated.
98	103–104	
	91–96	
	88–96	
104	88–96	
		Normal rhythm restored.
111	71–81	
104	74–81	
		Auricles fibrillated.
81	94–101	
	86–92	
92	82–91	
		Normal rhythm restored.
96	70–76	
98	71–79	
		Auricles fibrillated.
78	88–98	
	86–94	
	85–94	
	88–96	
	86–95	
	90–96	
94	83–92	
		Normal rhythm restored.
111	71–79	
116	71–79	
120	71–78	
		Auricles fibrillated.
84	94–101	
99	86–91	
	84–91	
	91–98	
	91–98	
102	91–96	
	81–91	
		Normal rhythm restored.
120	70–78	
	70–76	
		Auricles fibrillated.
132	61–69	

TABLE IV (*Continued*).

CAT 30.

Arterial pressure in mm. of mercury.	Venous pressure in mm. of magnesium sulphate.	
90	86–91	
104	81–86	
	81–86	
	86–91	
108	86–91	Normal rhythm restored.
125	66–71	
	61–66	
	61–66	
123	59–63	Auricles fibrillated.
84	76–81	
	79–83	
90	81–86	Normal rhythm restored.
118	61–66	
	59–63	
	59–61	
118	59–61	

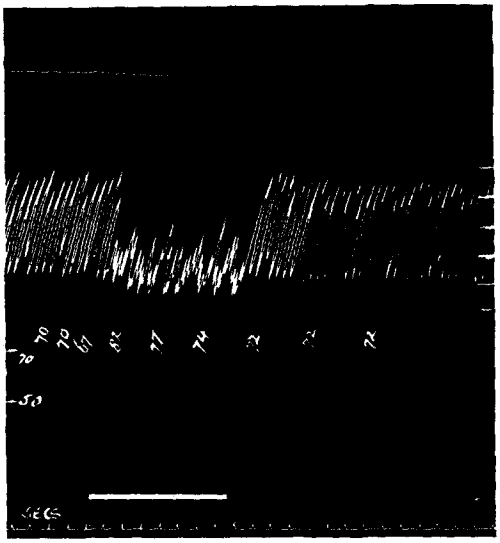
TABLE V.

CAT 29.

Arterial pressure in mm. of mercury.	Venous pressure in mm. of magnesium sulphate.	
116	67	
	69	Auricles fibrillated.
118	70	
	75	
114	77	Normal rhythm restored.
110	80	
100	40	Auricles fibrillated.
104	45	
	47	
102	50	Normal rhythm restored.
96	50	

TABLE V (*Continued*).

Arterial pressure in mm of mercury.	Venous pressure in mm. of magnesium sulphate.	
128	43	
	42	
124	39	
		Auricles fibrillated.
118	45	
118	45	
116	45	
		Normal rhythm restored.
118	37	
114	37	
78	33	
78	33	
		Auricles fibrillated.
	CAT 29.	
68	40	
68	32	
68	32	
		Normal rhythm restored.
78	33	
76	33	



TEXT-FIG. 5. Cat 47. A record of arterial and venous pressures and of heart and intestinal volumes, showing the effects of auricular fibrillation. At the time this curve was taken the intestinal record was not working well and the dip in the curve, indicating diminution of intestinal volume, is only just seen.

That the changes in the arterial and venous systems are the direct outcome of altered cardiac mechanism is indicated by the general manner in which the pressures behave. The peripheral circulation is affected passively. This is also shown by records of intestinal volume. Examples of venous readings are given in text-figures 1, 2, *c*, and 5.

CHANGES IN THE INTESTINAL CIRCULATION.

The volume changes in the small intestine were measured in the usual manner by enclosing a loop of the gut in an air-tight box connected to a recorder. The activity of the vasomotor system was checked at the termination of each series of observations by the injection of a suitable dose of adrenalin solution, so as to demonstrate the customary fall of volume with the rise of blood pressure. Observations have been made upon six cats and two dogs from this point of view; they have given uniform results.

The volume curves at the onset of fibrillation have always run parallel with the arterial pressure changes; a fall of arterial pressure is accompanied by a fall of volume (text-figure 4, *a*), and conversely a rise of blood pressure, be it primary or secondary, is accompanied by a rise of intestinal volume (text-figure 4, *b*). The changes in volume at the offset of fibrillation correspond in the same fashion with the changes of arterial pressure.

As might be anticipated, alterations such as occur in the arterial and venous pressures are consequently explained by the altered cardiac mechanism and by this alone. Observations which have been made upon the output of the heart are confirmatory of this conclusion.

CHANGES IN THE VOLUME OF THE HEART.

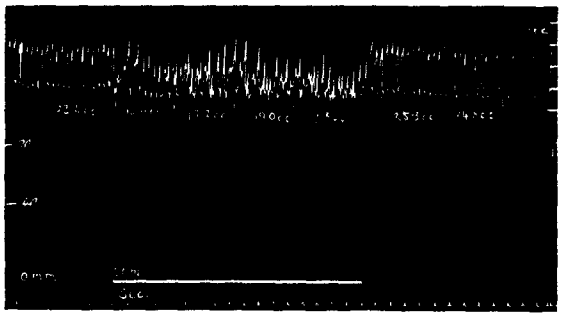
The mean volume changes of the heart at the onset and offset of fibrillation of the auricles have been estimated by opening the chest, cutting away all but a short sleeve of pericardium, enclosing the whole heart in a glass cardiometer and rendering the mouth of the cardiometer air-tight by tying the sleeve of pericardium firmly over it. The records were taken in twelve cats by means of the recorder designed by Starling[6] for the purpose. The recorder may be calibrated and the excursions are uniform for like changes of volume within the necessary limits.

The volume of output has been calculated by adding together the length of all the downstrokes, representing systoles, over a definite period of time. The

[6] Jerusalem, E., and Starling, E. H., *Jour. Physiol.*, 1910, xl, 286.

excursions yielded by changes of volume of 1 cm. are ascertained at the subsequent calibration. The method gives a fairly accurate expression of output in cubic centimeters while the heart beats regularly, and its accuracy is independent of the variations in excursion consequent upon respiration. It is also, I believe, fairly accurate when the ventricle beats irregularly, though under such circumstances the readings tend to be too high. This is due probably in some part to the inefficiency of certain ventricular beats. While the auricles are fibrillating, a large number of ventricular contractions fail to open the aortic valves; yet it is impossible to avoid a certain excursion in the recording lever as a result of change in the shape of the heart when no blood leaves it, even with the closest fitting cardiometer. Such excursions add to the sum of the calculated output. Consequently in some of the experiments the readings during the fibrillation period have been obviously too high, but the comparison of readings over periods of change of blood pressure has given uniform results. The figures obtained have been regarded relatively rather than absolutely, and treated in this fashion they are of value.

When the blood pressure changes in response to onset or offset of fibrillation, or when it changes during the fibrillation, the output also changes. If there is a fall of pressure there is a fall of output; if there is a rise of pressure there is a rise of output. Examples are given in tables VI and IX and text-figures 2, 3, 5, and 6.



TEXT-FIG. 6. Cat 43. Vagi cut. Arterial pressure and heart volume curves, showing the effects of auricular fibrillation. The heart output and arterial pressure vary in the same directions. There is only a slight decrease in the mean size of the heart.

Mean Volume Changes.—When the auricles pass into fibrillation, the heart demonstrates a change in mean volume, of which the direction is practically constant. The volume decreases.[7] This is

[7] I shall refer briefly to certain exceptions to this rule in my concluding remarks.

TABLE VI.

Cat.	Before fibrillation.			After onset.				A few seconds later.			After offset.		
	Blood pressure (mean).	Heart rate.	Output per 4 sec. in c.c.	Blood pressure (mean).	Heart rate.[8]	Output per 4 sec. in c.c.	Volume change in c.c.	Blood pressure (mean).	Heart rate.[8]	Output per 4 sec. in c.c.	Blood pressure (mean).	Heart rate.	Output per 4 sec. in c.c.
42	98	105	21.5	87	255	20.2	−1.4	108	225	26.0	100	105	22.7
	98	105	21.4	87	255	19.0	−1.5	104	210	24.9	104	105	23.2
	Left vagus cut.												
	104	172	20.9	82	270	17.1	−0.3	100	262	21.7	98	150	21.7
43	82	135	24.8	60	165	14.8	−1.3	60	165	15.0	84	135	26.2
	Right vagus cut.												
	68	180	19.8	46	300	15.0	0	47	292	15.0	67	180	23.0
	Left vagus cut.												
	65	172	23.4	46	255	16.9	−0.4	50	270	19.0	64	180	24.8
44	82	169	29.4	63	285	16.0	−1.2	62	277	16.0	82	177	28.0
	73	170	15.3	34	258	6.1	+1.0	64	255	11.8	75	168	17.2
47	82	202	40.7	57	365	30.3	−1.4	76	250	39.1	78	200	39.8
	78	195	38.7	58	360	22.7	−1.4	75	250	37.7	78	200	40.6
49	67	186	25.5	58	300	18.0	−0.4				66	177	25.2[9]
	All cardiac nerves divided.												
	60	163	22.4	49	270	15.2	0	55	285	19.4	63	157	23.4

[8] Heart rates are calculated from cardiometer curves; approximate readings.

[9] Fibrillation lasted only a short while.

indeed obvious from mere inspection of the organ. The decrease in size is seen whether the arterial pressure rises (text-figures 2, *a*, and 3), falls (text-figures 5 and 6), or remains stationary. It is due entirely to change in the size of the ventricles. The auricles, it is true, are included in the cardiometer, but they pass into a position of diastole, or increased mean volume, when fibrillation begins. Examples of the mean volume change will be found in tables VI and IX, and in the figures to which reference has been made.

SUMMARY OF PREVIOUS OBSERVATIONS.

As far as I have recorded my observations, they show that fibrillation produces changes in arterial pressure, which consist of certain falls and certain rises of pressure. The venous pressure moves in the opposite direction from the arterial pressure. If we compare increase and decrease of arterial pressure with increase and decrease of intestinal volume, it may be said that they change in the same direction, as does also the cardiac output. We may conclude, therefore, that the changes in arterial pressure are due solely to the change in the mechanism of the heart beat and we may seek the cause of all the circulatory pressure changes in this organ.[10]

We have to consider the cause of the usual steep fall of arterial pressure at the onset of fibrillation. We have to determine the cause of the partial or complete recovery in certain instances, the cause of the variation in the reaction in different experiments, and the cause of the fluctuation of pressure during fibrillation in a given experiment. We have also to seek the reason for the change of cardiac volume.

It has become more and more apparent, during the course of the experiments, that the explanation of most of these phenomena lies in the acceleration of the heart's rate, though it has not been altogether easy of proof. The effects of auricular fibrillation and of simple tachycardias, provoked by applying regular induction shocks to the right auricle, have been compared.

[10] The changes are not affected by section of the vagi or sympathetics.

DEGREE OF VENTRICULAR ACCELERATION WHEN THE AURICLES FIBRILLATE, AND THE EFFECT OF REGULAR ACCELERATION OF SIMILAR EXTENT.

A number of figures, giving the ventricular rates before and after the onset of fibrillation, will be found in tables VI and IX; these rates were calculated from cardiometer curves. In blood pressure tracings the rate of the ventricle cannot often be estimated during the period of fibrillation, because the irregularity of the ventricle is so gross and because usually so many beats of the ventricle fail to affect the arterial pressure. The curves of heart volume give more accurate readings, and readings which are approximately correct. But at the present time, that the true grade of acceleration may be appreciated, I give the following figures (table VII), taken from electrocardiographic curves in a separate series of experiments. The conditions of the animals in these experiments were similar to those described in the present series.

TABLE VII.

	Before onset of fibrillation.	After onset of fibrillation.
Dogs	95	185
	170	230
	157	237
	183	330
	149	255
	102	205
	117	198
	165	240
	160	270
	185	283
Cats	151	304
	192	330

Other figures are given in the sequel.

The acceleration of the ventricle, when the auricles pass into fibrillation, is from 50 to 100 per cent. under normal experimental conditions. The rate of the ventricle during the period of fibrillation varies in dogs and cats approximately between 180 to 330 beats per minute.

In several experiments, a simple manometric comparison was made between the effects of auricular fibrillation and regular ac-

celeration. The mercurial manometer was employed, and after electrodes had been fastened to the right auricle the chest was closed. Examples of the results are seen in table VIII. The

TABLE VIII.

CAT 52.

	Ventricular rate rises from	Blood pressure changes from
Fibrillation	207 to —	107 to 86
Tachycardia	184 to 190	102 to 104
Tachycardia	176 to 285	113 to 109
Tachycardia	176 to 288	113 to 107
Tachycardia	178 to 242	112 to 112
Tachycardia	178 to 236	113 to 115
Tachycardia	177 to 208	111 to 113
Tachycardia	176 to 210	112 to 113
Tachycardia	176 to 236	112 to 113
Tachycardia	175 to 256	112 to 110
Tachycardia	180 to 286	112 to 103
Tachycardia	174 to 256	110 to 110
Tachycardia	176 to 258	110 to 108
Tachycardia	171 to 268	111 to 103
Tachycardia	174 to 276	109 to 93
Tachycardia	172 to 310	107 to 73
Fibrillation	174 to —	109 to 79

CAT 53.

Tachycardia	236 to 395	90 to 51
Tachycardia	228 to 364	89 to 66
Fibrillation	230 to —	88 to 62
Tachycardia	186 to 338	77 to 43
Tachycardia	188 to 305	77 to 47
Tachycardia	200 to 258	80 to 74
Tachycardia	195 to 246	81 to 78
Tachycardia	190 to 236	82 to 81
Tachycardia	183 to 236	82 to 81
Tachycardia	192 to 236	83 to 81
Tachycardia	192 to 204	82 to 82

ventricular rate during the periods of simple tachycardia could be estimated readily from the arterial curves and signal of stimulation. The estimation of rate during the periods of fibrillation was not possible. It will be seen that considerable acceleration of the ventricle (to 242, 256) may occur without change of arterial pressure, or

that such increase of rate may be accompanied by a slight rise or slight fall of arterial pressure. It will also be seen that, as higher rates are reached, small falls of pressure ensue and that eventually, as the rate increases, the fall becomes greater, until when the heart rate rises to 300 or more per minute, falls result which are equivalent to those obtained in fibrillation. These rates are of the degree known to occur when the auricles are fibrillating. The actual figures naturally vary very much from experiment to experiment, and an absolute comparison is by no means easy. It is difficult to obtain comparable falls with simple and with irregular acceleration, for the degree of fall varies with so many factors; for example, with the original height of venous and arterial blood pressure and with the original heart rate. The chief defect of the method now described lies in the impossibility on most occasions of ascertaining the ventricular rate during the period of fibrillation. Other examples, from experiments in which the chest was open, and in which the rates were calculated from cardiometer curves, are given in table IX. These figures confirm the conclusion that the rate is

TABLE IX.

Vagi and Sympathetics Cut.

Cat 50.

	Ventricular rate rises from	Blood pressure falls from	Heart decreases in size by approximately	Output per 4 secs. in c.c. decreased from
Fibrillation	200 to 283[11]	86 to 72	1.3 c.c.	
Fibrillation	192 to 275[11]	84 to 72	1.3 c.c.	
Fibrillation	174 to 230[11]	80 to 66	1.4 c.c.	25.2 to 21.0
Tachycardia	170 to 190	71 to 71	0.25 c.c.	22.6 to 21.2
Tachycardia	172 to 301	73 to 73	0.33 c.c.	23.9 to 22.8
Tachycardia	181 to 204	60 to 59	0.25 c.c.	18.7 to 14.9
Tachycardia	190 to 263	55 to 49	0.5 c.c.	17.8 to 10.8

Cat 51.

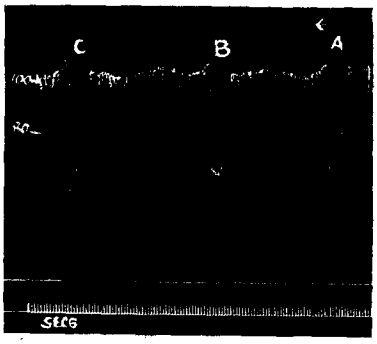
Sympathetics Cut.

Fibrillation	156 to 205[11]	58 to 48	1.4 c.c.	23.0 to 21.2
Tachycardia	155 to 195	58 to 57	1.0 c.c.	
Tachycardia	153 to 185	57 to 56	0.8 c.c.	24.2 to 23.1
Tachycardia	150 to 223	59 to 54	1.4 c.c.	23.3 to 17.5
Tachycardia	150 to 190	60 to 59	1.0 c.c.	
Fibrillation	140 to 216[11]	58 to 47	1.0 c.c.	
Fibrillation	139 to 196[11]	56 to 42	1.0 c.c.	

[11] These figures, taken from counts in the cardiometer curve, are approximate.

a predominant factor in producing the usual fall of blood pressure, though they indicate that acceleration, when the ventricular action is regular, is less profound in its effects than is acceleration when the ventricular regularity is disturbed. The last named experiments were performed for the sake of heart volume curves, rather than for the rate readings.

The question has been approached in a more direct manner in further experiments. In these, animals in which the chest wall was subsequently closed have been utilized. In two experiments the blood pressures were recorded with the mercurial manometer, and the rates were calculated from simultaneous electrocardiograms. While the blood pressure was running steadily, a faradic current or repeated induction shocks at varying rates were passed into the auricle through the chest wall by means of the specially protected electrodes. When a pressure fall of uniform extent was obtained after several faradic stimulations, a fall of similar amount, as far as this was possible, was obtained with regular induction shocks; this was accomplished by varying their rate of incidence. The experiment was then repeated, and at the same time electrocardiograms



TEXT-FIG. 7. Cat 67. Unlike the remainder of the figures, this blood pressure curve reads from right to left. It shows a fall of blood pressure obtained with (*A*) faradization of the auricle, (*B*) regular acceleration of the auricle, and (*C*) faradization of the auricle. The heart rates corresponding with these three curves were 216, 225, and 231 respectively.

were taken from the right fore limb and left hind limb. Such experiments are not easy, because, apart from variations in the general

line of blood pressure, similar curves of blood pressure during fibrillation are not often obtainable; and it is necessary that the comparison should be made in the shortest possible time, so that the two curves lie close together. Text-figure 7 is an example of such an experiment, and selected figures are given in table X. The read-

TABLE X.

		Blood pressure falls from	Heart rate rises from	
I	Fibrillation	108 to 94 = 14	188 to 216	
	Tachycardia	112 to 96 = 16	185 to 249	
II	Fibrillation	120 to 100 = 20	180 to 220	
	Tachycardia	126 to 90 = 36	179 to 261	
	Tachycardia	112 to 87 = 25	180 to 249	
III	Fibrillation	86 to 68 = 18	150 to 207	
	Tachycardia	104 to 85 = 19	154 to 222	
IV	Fibrillation	102 to 68 = 34	145 to 216	(Figure 7, *A*, *B*, and *C*.)
	Tachycardia	100 to 65 = 35	147 to 225	
	Fibrillation	100 to 62 = 38	147 to 231	

ings confirm the previous observations. Regular and irregular accelerations of similar rate produce approximately equal falls of pressure, but the irregular acceleration is almost always a little more profound in its effects. That is to say, if equal falls of pressure are obtained, it is subsequently found that the rate during the period of regular acceleration slightly exceeds that of the period of fibrillation.

When the auricles fibrillate, a number of heart beats fail to affect the arterial curve and are entirely lost. The series of experiments was therefore extended, and arterial curves were taken from five animals with Hürthle's manometer, the two records, arterial and electrocardiographic, lying on the same plate. Repeated observations are required before curves are secured in which systolic and diastolic readings are alike and in which a comparison is valid. Nevertheless, out of a large number of such curves, some may be chosen from which the required data are obtainable; the readings conform to those obtained in other ways. Two observations are chosen for illustrative purposes; in selecting them attention has been paid to the form of the arterial curve during the period of fibrilla-

tion. In figure 1, *a* and *b* (plate 43), there are two falls of blood pressure. The original systolic and diastolic pressures are the same in both curves during the interval of depression; the average diastolic pressure is somewhat lower in the curve which shows fibrillation. The average ventricular rate is 300 for the fibrillation curve, and 306 for the curve which shows regular acceleration. Thus the falls of pressure for similar rates are approximately equal, though again the fibrillation has a somewhat more profound effect. Figure 2, *a* and *b* (plate 43), are from a similar experiment. The original pressures in each curve are almost the same. The initial fall is somewhat greater in the fibrillation curve; eventually the blood pressure rises above that shown at a similar phase of figure 2, *a*, and once more falls below it. The respective rates for figure 2, *a* and *b*, are 315 and 328.

If we continue to consider the cause of the usual fall of arterial pressure, the part played by the average rate of heart beat at such times seems clear. It appears to be the all important factor. There is, however, another conceivable cause of the fall of pressure. It has been recently emphasized by Gesell[12] as a reason for a pressure fall in auricular fibrillation. Gesell produced complete heart-block experimentally; thus he dealt with hearts in which the ventricles beat at uniform rates; he then experimented with the auricles, eliminating their systoles by inducing fibrillation in them. In this way he produced falls of blood pressure of 10 mm. of mercury. His conclusion, from this and other experiments, that the auricles aid in ventricular filling to an extent which may not be neglected, cannot be questioned; but at the same time, the absence of auricular filling cannot be considered a serious factor in the production of the falls of pressure with which we now deal. Such a possibility requires discussion, because in the comparison which has been instituted in the present series of experiments this factor is neglected. In those instances in which regular acceleration is induced and in which it is of a sufficiently high grade to produce large falls of blood pressure, auricular filling does not occur. The auricles are as much out of action as they are when they fibrillate, for each auricular contraction falls with the preceding ventricular systole; this is evident in the electrocardiograms; *P* falls with the preceding *T* (figure 2, *a*). I draw attention especially to this fact, because the comparison between the regular and irregular acceleration is more perfect. The question arises as to what extent the absence of auricular filling influences the result. Gesell produced falls of pressure of only small extent, about 10 mm. of mercury. It must be remembered that in these experiments each ventricular cycle was accompanied by four or five auricular cycles at the time when fibrillation of the auricles was induced. The ventricle fills, under ordinary circumstances, chiefly during the early and late phases of diastole. The information which we require is the relation of

[12] Gesell, R. A., *Am. Jour. Physiol.*, 1911, xxix, 32.

the amount of early filling by static pressure to the late or forcible auricular filling. When the ventricle beats slowly, the filling during mid-diastole is slow, unless, as in Gesell's experiments, there is additional or forced feeding as a result of supernumerary auricular contractions. Ten mm. of mercury, therefore, are probably too much to allow as the amount of the fall which is attributable to the auricular factor. That the auricular factor is an unimportant one is clearly evidenced by the varying effect which fibrillation of the auricles has upon arterial pressure. In place of a fall, the blood pressure may remain unaltered; it may actually rise. If in figure 3, *a* (plate 44), the beats marked by asterisks are compared, the second is seen to succeed an auricular contraction. The two beats are of almost the same values; the diastolic pressures are equal, the systolic pressure of the second beat exceeds that of the first by less than 7 mm. of mercury. The difference is fully accounted for by the difference in lengths of preceding pauses; these are 8.6 and 9.4 thirtieths respectively. The filling which precedes the two contractions seems to have been equally rapid. The absence of auricular filling when the auricles fibrillate seems to be almost if not quite compensated for by increased venous pressure, when there is a fall of arterial pressure.

The degree of acceleration not only accounts, I believe, almost entirely for the usual steep fall, but accounts completely for the variations in the amount of fall from time to time, and also for the fluctuations of pressure which are seen during the course of a single observation. Comparisons are of most value under the last named circumstance. When the ventricle beats its fastest, a number of contractions produce no arterial pulsation. The shortest cycles of figure 3, *a* and *b*, correspond to the steepest falls of pressure and to those portions of the curve at which no arterial pulsations are visible. After a careful examination of the curves in my possession I find that there is no exception to the rule which these figures illustrate. If previous events are taken into consideration, each rise and each fall of pressure are fully accounted for by the lengths of the cycles found to correspond to it. That the curve of rate of fall or of rise should not be absolutely parallel to the curve of lengths of pauses is natural, for the effect of a longer or shorter pause is not confined to the immediately succeeding cycle.[13] This is especially the case where the initial fall and terminal rise are concerned; for over these periods the supply of blood to the heart does

[13] Where there is an abortive contraction, as in figure 3, *b* (marked with asterisks), the beat which follows has a greater effect on arterial pressure than may be accounted for by the previous pause; this evidently results from some of the blood of the previous diastole being carried over for one heart cycle.

not immediately meet the requirements of the particular pauses in question; the supply may be excessive, or it may be scanty.

A short pause is equivalent to a fleeting acceleration; a long pause is equivalent to a fleeting retardation. Moreover, a short pause counts more heavily than a relatively long one; the study of regular acceleration teaches that great acceleration produces effects out of proportion to the increase of rate. If over two equal periods the rate is the same, but over one the heart action is regular while over the other long and short pauses are mixed, the effect is more profound over the latter. Whenever the blood pressure rises at the onset of fibrillation, or wherever it rises above the original point during the progress of fibrillation, the action of the ventricle is more regular, both in the incidence of the excursions and the amplitude of the beats, in arterial and cardiometer curves (text-figures 2, *a,* and 3); it is also slower. The rates corresponding to the four rises of pressure tabulated in table I were 102, 128, 193, and 255.[14] Where the ventricle beats fast, the arterial pulse and cardiometer curves are very irregular (text-figures 5 and 6), for many of the contractions of the ventricle fail to raise the aortic valves. The initial falls of pressure in text-figure 3 are marked in the cardiometer curve by very irregular heart action; the upstrokes of diastole are notched by the weak contractions which are abortive. The periods of raised pressure in the same figure correspond to more regular heart action. Contrast with this the gross irregularity of text-figure 6; it is maintained throughout, and the pressure is low throughout. When it is remembered that such gross irregularity signifies rapid action, the reason of the fall and its maintenance is more obvious. The same point is illustrated by the falls of text-figure 2, *b* and *c*. Where the pulse irregularity is great, and beats are dropped, the pressure falls steeply; where the action is more regular the pressure rises; and where beats are dropped, as we have seen, the pauses which precede them are curtailed.

Thus the fluctuations of arterial pressure are explained by a detailed consideration of heart rate. Falls are due to excessive rapidity, rises to less rapid action; a rise above the initial pressure is comparable to the occasional small rises which result from simple acceleration. Briefly, the changes in the peripheral circulation are

[14] In this instance the original rate was 200.

most serious when the acceleration is greatest. That is a conclusion which is fully borne out by clinical experience. When fibrillation is present, the rate of ventricular action is one of the most important indications of the gravity of the condition as a whole.

The initial fall is usually the deepest, as I have stated, though this is not invariably the case; but it is deepest in a sufficient percentage of cases to place its occurrence beyond the possibility of coincidence. The most rapid ventricular action is also found at the onset of the disturbance and accounts for it. The reason why the ventricle beats more rapidly when fibrillation begins is probably that at the onset of fibrillation the tissues which transmit the impulse from auricle to ventricle are usually unfatigued. When after a fall the initial pressure is recovered and fibrillation persists, the recovery may be assigned to lessened conduction power between auricles and ventricles. The recovery is probably due also in a measure to increased venous pressure. The falls which accompany acceleration are the result of curtailment of the diastolic periods; a heightened venous pressure, by promoting the filling, will tend to compensate for it.

The final rise at the termination of the fibrillation needs no detailed explanation; it is due, as is the frequent overriding of the initial pressure, to the transference of the obstructed blood from the venous to the arterial system.

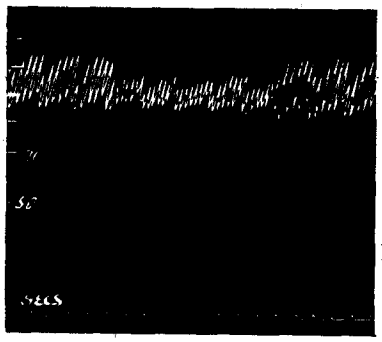
CAUSE OF THE MEAN VOLUME CHANGE.

It has been stated that when the auricles pass into fibrillation, the heart volume decreases. A similar reaction is found when the heart rate shows regular acceleration (table IX and text-figure 8). Up to a certain point the greater the acceleration, the greater is the decrease of volume. It may be, as in table IX, cat 51, that if regular and irregular accelerations are approximately equal in degree, the volume decrease is of approximately equal amounts. On the other hand, and perhaps more commonly, the regular acceleration may be accompanied by a relatively smaller decrease (table IX, cat 50).

The chief factor in producing diminution of heart capacity when the auricles fibrillate is the same as that which produces it when the acceleration is regular. The diastolic intervals are curtailed; it is the diastolic volume which shows the chief reaction; the systolic volume also suffers, though to a less extent. The heart is smaller at all phases of its beat. But the decrease in heart volume is independent of the velocity of flow through it. The flow is usually decreased when there is a fall of pressure, but it may be increased when there is a rise above the initial pressure (text-figure 3). Thus, although the diastolic intervals are decreased, the blood flow into the heart may be even in excess of the initial flow. Under these circumstances the average rate of inflow must have increased. The reason for such an increase is not to be found in an increased venous pressure; it must consequently be due to a lowering of mean diastolic ventricular pressure. There seems, therefore, to be no alteration of the ventricular function which is spoken of as "tone." The decrease of heart volume appears to result

from more purely mechanical causes; shortening of the diastolic periods as a result of acceleration is sufficient alone to account for it. Section of all the cardiac nerves does not influence the reaction.

The decrease of mean heart volume is the almost constant reaction of the normal heart; but in the human subject the onset of auricular fibrillation is often accompanied by obvious increase of volume. I have seen dilatation on a few occasions in the present series of experiments. The actual cause of this unusual reaction has been difficult to ascertain, but it may have been due, as probably is



TEXT-FIG. 8. Cat 50. Arterial pressure and heart volume curves, showing the effects of stimulating the auricle with a succession of regular induction shocks. The blood pressure is lowered and the heart volume and output are decreased.

the reaction of the human heart, to nutritional changes in the ventricular muscle. A weak or failing muscle will not necessarily react in the same manner as does fresh and vigorous tissue.

There is perhaps no quality of heart muscle which it is more difficult to estimate at a given moment than its state of exhaustion, and I write with hesitancy of the relation of dilatation to such a quality at the onset of fibrillation. More observations are required before any views can be exposed with confidence. Where it occurs, the dilatation of the ventricles at the onset of fibrillation is usually seen towards the end of an experiment. It is usually associated with relatively low blood pressure. This will be evident from an examination of table VI; dilatation of the heart or unchanged volume accompany initially low arterial tensions.

On one occasion a series of curves was obtained from an animal, in which a complete transition was seen from decreasing to increasing volume. Three curves from this series are shown in text-figure 2. The blood pressure, which falls steadily throughout the whole series, rises in the first curve when fibrillation begins; it falls in the second and third curves. The volume decreases by several cubic centimeters in the first curve; it shows slight progressive increase in the second, and considerable and progressive increase in the third. The explanation of this series of volume changes seems to be that from first to last there was a steady exhaustion of the muscle, so that while at first, being vigorous, it reacted in the usual fashion, maintaining its tone, toward the end the increase of rate was speedily followed by loss of tone.

SUMMARY AND CONCLUSIONS.

When the auricles fibrillate, the following effects are observed.

1. The arterial blood pressure may rise, fall, or remain stationary. Usually it falls. If it falls, it generally rises again towards or to the initial pressure.

2. The venous pressure changes are the reverse of the arterial.

3. The intestinal volume and the cardiac output changes are in the same direction as those of arterial blood pressure.

4. From these observations it may be concluded that the peripheral circulatory effects are purely passive.

5. The volume of the heart decreases except in instances where there is reason to believe that the circulation is failing.

All the changes described in the foregoing paragraphs, and also the variations in blood pressure reactions which occur from time to time, are attributable to alterations in the rate of ventricular contraction. Similar, though perhaps less profound changes, are seen when the heart rate accelerates in like degree in response to regular induction shocks.[15]

EXPLANATION OF PLATES.

PLATE 43.

FIG. 1, *a* and *b*. Cat 64. Hürthle manometer curves from the carotid, and electrocardiograms. In *a* the auricle was faradized. In *b* regular induction shocks were employed. The falls of pressure are equal; the respective rates of ventricular action are 300 and 305. The time marker is in thirtieths of a second.

FIG. 2, *a* and *b*. Cat 65. Similar curves from another animal; *a* = stimulation with regular induction shocks at a rate of 328 per minute; *b* = faradization; rate of ventricle 315 per minute. The falls of pressure are almost equal.

PLATE 44.

FIG. 3, *a* and *b*. Cat 67. Two electrocardiograms and two Hürthle curves; *a* shows fluctuations of arterial pressure and the return to the normal rhythm at the very end of the curve. The fluctuations of pressure were due to variations in heart rate. The measurements of the beats are expressed in thirtieths of a second. *b* shows the recovery of arterial pressure towards the end of a period of fibrillation, and its cause,—decrease of heart rate.

[15] This conclusion applies only to induction shocks applied to the auricle; the effect of similar stimulation of the ventricle is far more profound. Stimulation of the auricle seems to result in contractions of the ventricle, which are the most efficacious.

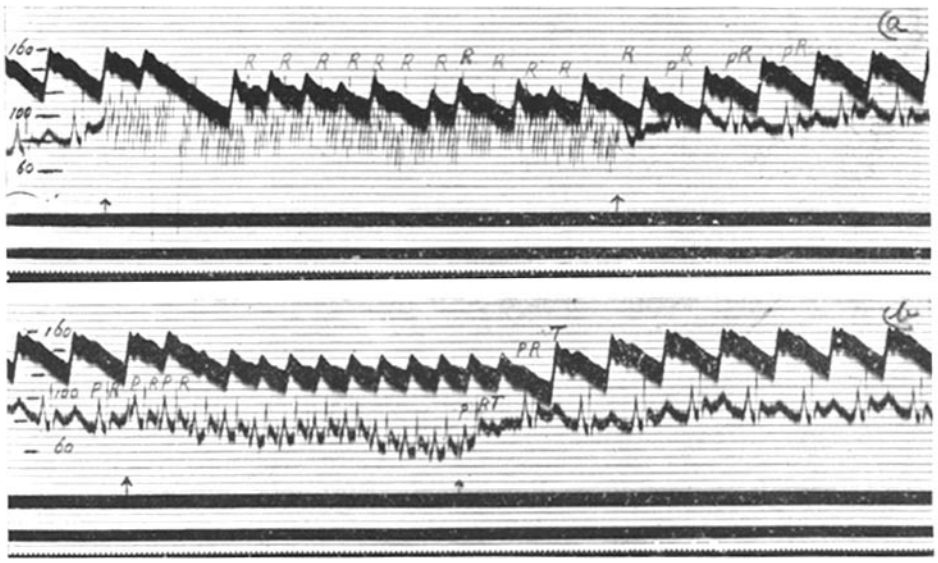


FIG. 1

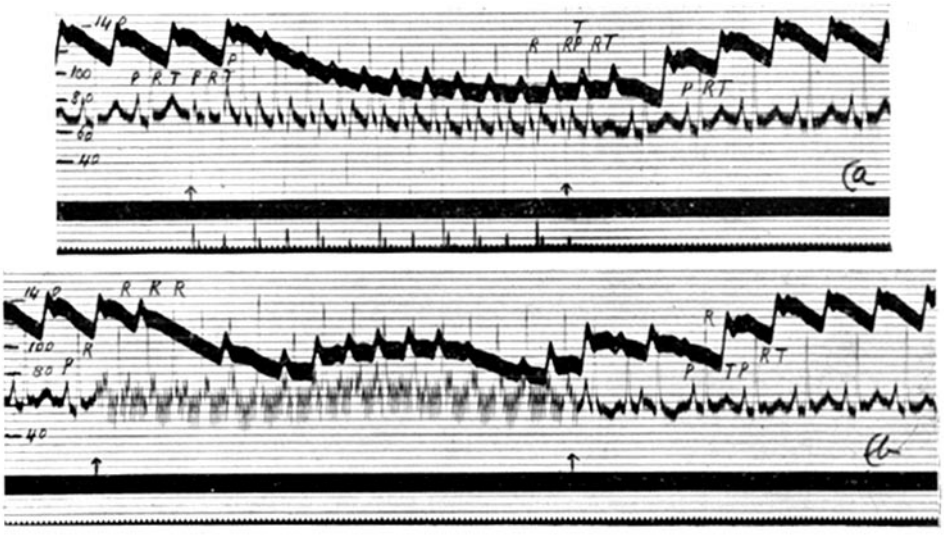


FIG. 2.

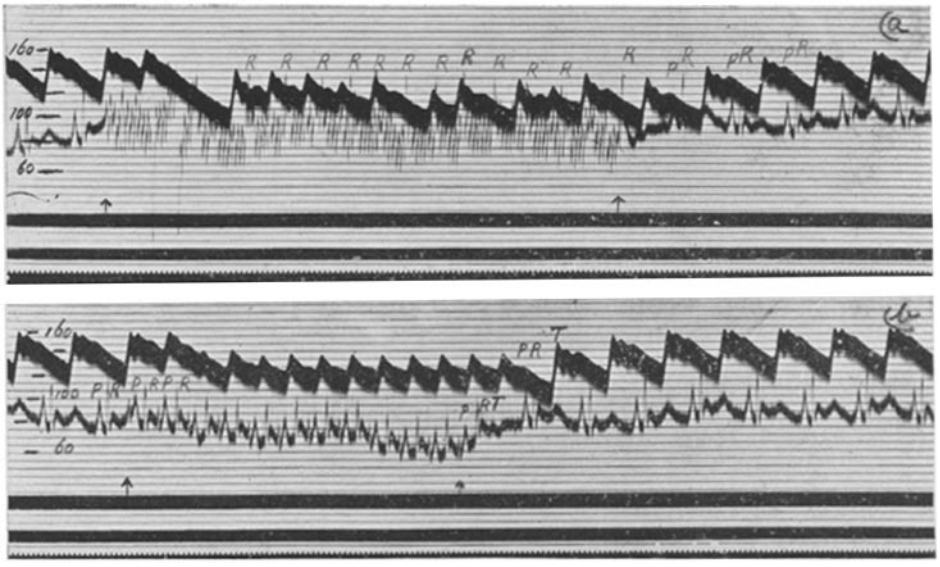


FIG. 3.