
On the Electromotive Changes Connected with the Beat of the Mammalian Heart, and of the Human Heart in Particular

Augustus D. Waller

Phil. Trans. R. Soc. Lond. B 1889 **180**, 169-194
doi: 10.1098/rstb.1889.0004

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IV. *On the Electromotive Changes connected with the Beat of the Mammalian Heart, and of the Human Heart in particular.*

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Communicated by Professor BURDON SANDERSON, F.R.S.

Received and Read June 21, 1888.

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PART I.

§ 1. *Introduction.*

In our investigation of the action of the excised Mammalian heart,* Dr. REID and I left undetermined certain points relating to its electromotive variations, more especially those which accompany the spontaneous beat of the excised organ.† The nature and direction of deflections were very variable, and indicated no regular origin or mode of progression of the excitatory process. In 62 observations we observed in 17 cases apex negativity alone, in 17 base negativity alone, in 16 apex followed by base negativity, in 12 base followed by apex negativity. We then remarked that the numerous irregularities met with in experiments upon the excised Mammalian heart were presumably due to irregularities and inequalities of tissue in the dying organ, which might have been due to differences of temperature, or to accidental injuries, &c.; but we were unable to verify the supposition by any experimental reproduction

* 'Phil. Trans.,' B., 1887, p. 215.

† *Loc. cit.*, p. 234.

of these irregularities. Most of the observations reported in Part I. had for their object to clear this part of the subject as far as possible to me.

§ 2. *Experimental Modification of the Electrical Variation connected with the Spontaneous Beat.*

The methods followed have been in the main those described in the paper already referred to,* with certain modifications of detail, such as the use of D'ARSONVAL'S chloride of silver electrodes (which proved to be convenient and excellent for the purpose in view), and with this difference, that, in order to examine the as far as possible intact and uninjured organ, the heart was examined *in situ*, the thorax being laid open and its walls fixed to a board immediately after the decapitation of the animal. The heart, having been examined *in situ*, was then excised and re-examined electrically.

Experiment 1.—Kitten's heart. March 31st, 1888.

	Time after death.	A to Hg.	A to H ₂ SO ₄ .
	min.	var.	
Spontaneous beat	5	SN	
Excited beat (exc. of apex)	SN	
Spontaneous beat after injury of base	10	S	
Heart excised—			
Excited beat (exc. of base)	S	
„ (exc. of apex)	NS	
Spontaneous beat	20	SN	
„ after injury of apex	N	

When electrode B was in contact with the auricle, which was beating twice to each ventricular beat, the variation was of the following rhythm:—

$n \quad nSN \quad n \quad nSN \quad n \quad nSN,$

or, expressed graphically,



When electrode B was in contact with the base of the ventricle the variation was of the rhythm SN . . . SN, the electrometer not being influenced by the auricular contractions.

* *Loc. cit.*, p. 235 (heart led-off to electrometer from two points A and B).

With $\left\{ \begin{array}{l} \text{A to H}_2\text{SO}_4 \\ \text{B to Hg} \end{array} \right\}$ variation N signifies A negative to B.
 „ S „ B „ A.
 With $\left\{ \begin{array}{l} \text{A to Hg} \\ \text{B to H}_2\text{SO}_4 \end{array} \right\}$ „ N „ B „ A.
 „ S „ A „ B.

Experiment 2.—Kitten's heart. April 1st, 1888.

	Time after death.	A to Hg.	A to H ₂ SO ₄ .
	min.	var.	
Spontaneous beat	9	SN	
Heart excised—			
Spontaneous beat	sN	
" after injury of apex	N	
" " " and of base	sN	

Experiment 3.—Cat's heart. April 21st, 1888. Death by decapitation. Five minutes after death the apex of the heart, consisting of the left ventricle, was pulseless and in firm rigor; the base of the heart, consisting of the right ventricle, was at the same time regularly contracting about 30 per minute; the auricles were also regularly contracting at a rate of 120 per minute; this condition was observed till 20 minutes *post mortem*.

Apex to Hg.	Base to H ₂ SO ₄ .	Variation N.
Liver to Hg.	Base to H ₂ SO ₄ .	„ N.
Neck to Hg.	Base to H ₂ SO ₄ .	„ N.
Neck to Hg.	Auricle to H ₂ SO ₄ .	„ <i>n</i> .
Apex to Hg.	Neck to H ₂ SO ₄ .	„ 0.

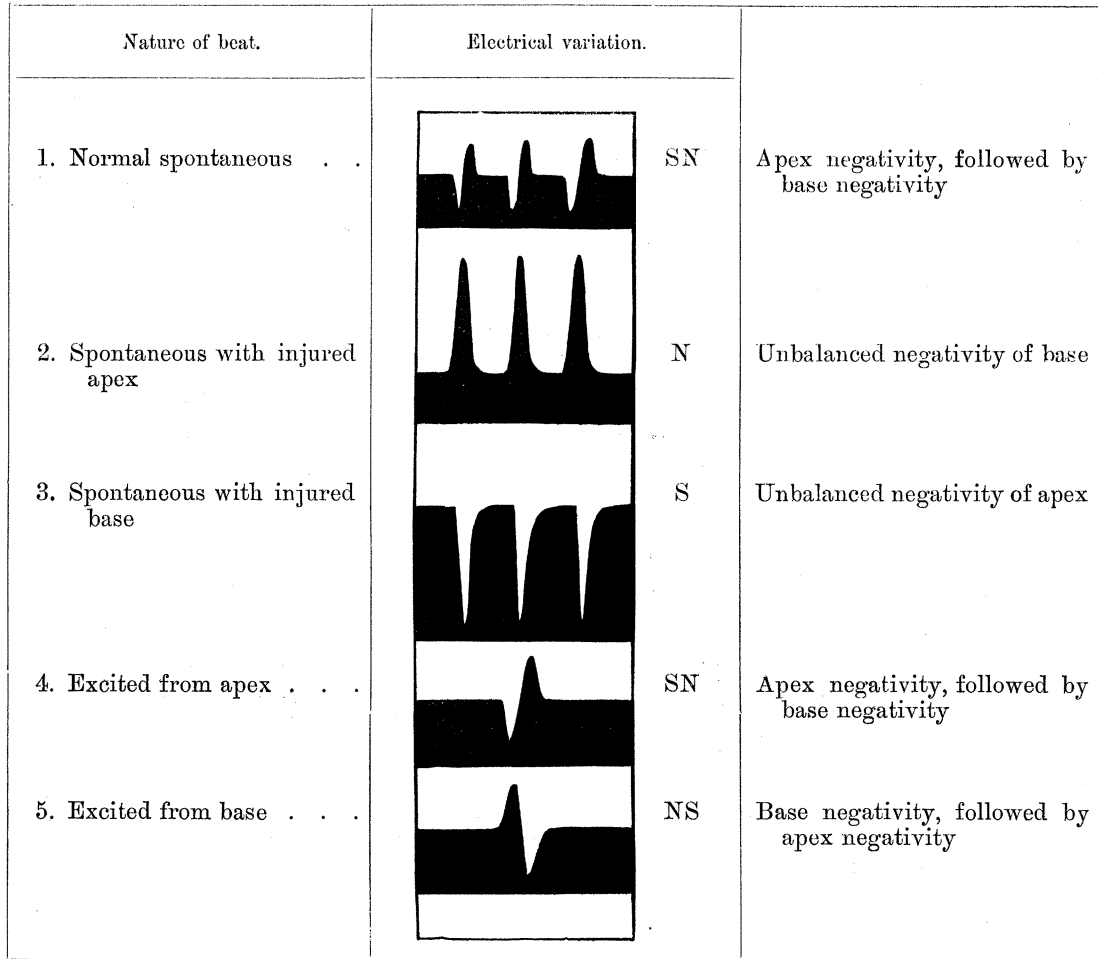
Remarks.—The condition of the ventricles was such that the right ventricle formed a loose pulsating pouch, connected with the upper two-thirds of the firmly contracted left ventricle. The contractions of the right ventricle were regular but small, and visible only at the basal part; the electrometer indicated negativity of the contracting portion. If, with one leading-off electrode applied to an indifferent part, the other leading-off electrode was shifted to a distance from the actually contracting portion, the excursion was quickly lessened and lost; if it was shifted to the auricle, the N variation of a ventricular rhythm gave way to the much more frequent variation *n* of auricular rhythm.

Nothing can be clearer than these effects of injury at base and apex respectively. The diphasic variation SN (*viz.*, apex negativity followed by base negativity) is, in consequence of injury of the apex, converted into the monophasic variation N (unbalanced negativity of base). After a time the diphasic variation SN re-appears, and now it is converted into the monophasic variation S (unbalanced negativity of apex) in consequence of injury of the apex. These facts, illustrated in fig. 1, are precisely similar to those observed by BURDON SANDERSON and PAGE* upon the ventricle of the Frog and Tortoise, the only difference being in the nature of the normal variation antecedent to injury.

* 'Journal of Physiology,' vol. 2, p. 418; vol. 4, p. 335.

The early monophasic variation which Dr. REID and I had so frequently under observation with the Mammalian heart immediately after excision was probably of this nature; it is, indeed, "the expression of local predominance of a change taking place throughout the whole ventricle,"* but our opinion concerning it, to the effect that the single variation is proof of a practically single and simultaneous change

Fig. 1.



(Apex of heart to mercury of electrometer. Base of heart to sulphuric acid of electrometer.)

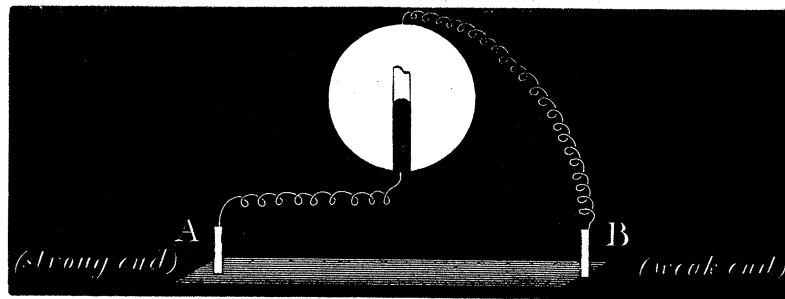
taking place throughout the ventricle, is no longer justified. The diphasic variation can be, as above described, rendered monophasic by injury on the Mammalian, as on the Batrachian, heart; bearing in mind the extreme susceptibility of the Mammalian heart, we must regard as highly probable that a monophasic variation is a consequence of injury, and that the normal variation is diphasic.

Confirmatory experiment.—That a monophasic variation is no evidence of simul-

* *Loc. cit.*, p. 241.

taneity of action throughout a contractile mass, but that it may depend upon unbalanced, and therefore predominant, negativity of a less injured and literally "stronger" portion of tissue, is very clearly shown with a strip cut from a fresh ventricle. It is not difficult in this case to combine mechanical with electrical exploration. Two levers resting upon the ventricle-strip record the passage of the wave of contraction, and the strip is at the same time led off from each end to the electrometer. A stimulus is applied to one end, the contraction passing along the strip is recorded by the two levers, and the electrical variation is watched in the electrometer. Usually the strip will be found not to contract equally strongly in its whole length, but one end gives a stronger contraction than the other. With this inequality of contraction a concordant inequality of electrical action is observed; the variation is not diphasic, but monophasic, indicating a predominant negativity of the more strongly contracting part, while by the asynchronism of the two levers we obtain ocular evidence of the passage of a wave of contraction. Sometimes the effect is the same whether the contraction be started from the stronger or from the weaker end; only in the first case the single phase is an intensified first phase, in the second case it is an intensified second phase. Sometimes it happens that the inequality of tissue is of such a degree that by excitation started at the "strong" end of the strip a monophasic variation is obtained, while by excitation at the "weak" end a diphasic variation is obtained, consisting of a minor first phase (negativity of the weak end), followed by a major second phase (negativity of the strong end).

Fig. 2.



Excitation of A gives Variation S (predominant 1st phase).
 „ B „ „ S (predominant 2nd phase);
 or nS (minor 1st phase; major 2nd phase).

The phenomena observed on the excised isolated auricle are of a similar character. Adopting the disposition above described, we may witness the passage of a wave of mechanical contraction attended by a monophasic variation, due to injury of its ventricular portion and consequent unbalanced negativity of the appendicular end.

What is the order of occurrence of the two phases?

Examination of the uninjured heart *in situ* shows that in the majority of cases it

is (1) negativity of apex followed by (2) negativity of base. Contrary to the case of the Frog, in which the normal variation has its initial phase at the base and its terminal phase at the apex of the ventricle, the normal variation of the Mammalian ventricle exhibits an initial phase at the apex and a terminal phase at the base. In our previously quoted paper on the excised Mammalian organ, Dr. REID and I stated (p. 230) that "in the spontaneous beat of the excised organ the contraction of the apex generally appears to precede that of the base." Out of 25 observations,* in 17 the mechanical effect of contraction manifested itself at the apex first, in 2 at the base first, in 6 there was no appreciable difference. Taken by themselves, these observations went to show that the contraction of the Mammalian heart normally commences at the apex. But the electrical observations by which we sought to confirm this testimony obtained by the mechanical method failed entirely and obliged us to state† "that, as regards the electromotive changes with visible spontaneous beats, our results show no uniformity; we can find in them no evidence either for or against the results we obtained by the graphic method." Observation of the electrical variation of the heart beating *in situ* shows it to be, *in the majority of cases examined* (11 out of 17), composed of (1) negativity of apex, (2) negativity of base; having regard to the fact that the organ is unstable and dying, we may expect to meet with exceptions to the rule, which, although by no means invariable, has been frequently enough verified to allow us to say of the Mammalian ventricle "apex first" with nearly as much certainty as we say of the Frog's heart "base first." As will be shown in Part II., the electrical phenomena of the Human heart afford strong confirmation of this view.

I must admit, however, that these observations on the exposed organ *in situ* have been the most troublesome and unsatisfactory in respect of their irregularities; six exceptions as compared with eleven "regular" results is a considerable proportion, and I have, therefore, sought by further observations to realise the effect of modifying circumstances, and the possible sources of irregularities. I will deal with these points *seriatim*.

A source of fallacy.—A possible source of an error of observation arises from the application of electrode B in close proximity to the auriculo-ventricular groove or in actual contact with the auricle. Under these circumstances, the auricular contraction may influence the electrical reading, which must not therefore be attributed to the ventricle alone; an electrometer reading in reality due to auricular followed by ventricular negativity might be taken to represent basal followed by apical negativity of the ventricle alone. If the auricles should be beating with a more rapid rhythm than the ventricles (as commonly occurs in the moribund Mammalian heart), there will obviously be no danger of confusing auricular with basal negativity; but, if auricles and ventricles should be beating in regular sequence, it is necessary to be on guard

* *Loc. cit.*, p. 249, Table H.

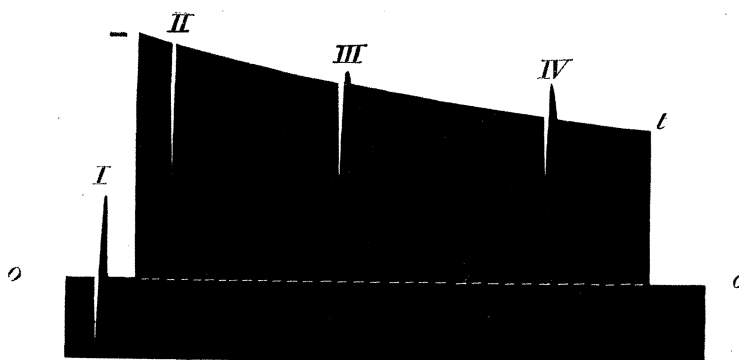
† *Loc. cit.*, p. 234.

against this possibility, which will be particularly misleading if the true basal phase should happen to be weak or absent, but which may also confuse the reading of a normal ventricular variation. An illustration of this point is given in the note to Experiment 1.

The origin of the excitatory process can be experimentally determined.—Apart from the fact that true stimuli are capable of starting the excitatory process from any part of the ventricle, I have found that it is possible by local alteration of temperature to determine the origin of a series of contractions. An excised ventricle which has become quiescent can be made to resume rhythmic contractions by raising its temperature. If this be done in such a way that the apex is more warmed than the base, the diphasic variation at each contraction has directions denoting origin at apex; if in such a way that the base is more warmed than the apex, the directions of the diphasic variation indicate origin at base.

Spontaneous modification of the ventricular variation.—It commonly happens that an original monophasic variation gradually gives place to a diphasic variation; this change may be attributed to either of two causes: (1) to the subsidence of injury at the injured lead-off, or (2) to the development of injury at the normal lead-off.

Fig. 3.



It appears to me probable that both these causes may play a part in producing the effect in question; as regards the first cause, we have in the Mammalian, as in the Batrachian heart, a rapid decline of the negativity manifested immediately after injury; the negativity is doubtless an expression of chemical activity at and near the injured zone, or, in other words, of a state of continued excitation; immediately after injury the degree of the alteration is such as to leave no margin for the manifestation of the excitatory effect (negativity) at the part, and an unbalanced negativity of any other normal part is witnessed in the shape of a monophasic variation when the organ contracts. The alteration is at first at a maximum, and gradually subsides until it leaves a margin of susceptibility within which excitatory effects (negativity) can be evidenced, and now we witness a diphasic variation composed of the preponderant negativity of a normal part and of the recovering negativity of the injured part. It

is convenient to allude to these two phases as the *major* phase and the *minor* phase respectively.

In the diagram, fig. 3, $o \dots o$ denotes the iso-electric state of two led-off points, A and B; the ordinate $o-$ denotes the maximum negativity of B manifested immediately after injury; the line $-t$ denotes the gradually declining negativity of B.

I. represents a normal diphasic variation.

1st phase, A negative. 2nd phase, B negative.

II. represents a monophasic variation after injury of B; unbalanced negativity of A.

III. and IV. represent diphasic variations re-appearing as the negativity of B declines.

1st phase, A negative (major phase).

2nd phase, B negative (minor phase).

The facts of experiment are in complete agreement with this theoretical representation. With a normally beating heart *in situ*, led off from the apex to the mercury of the electrometer, from the base to the sulphuric acid, the level of the mercury in the capillary showed that apex and base were iso-electric in the intervals between the beats, each of which was accompanied by the double variation SN, signifying:—

1st phase, apex negative.

2nd phase, base negative.

The base was now injured by crushing with forceps; on re-applying the electrode to the injured base, the mercury in the capillary came to rest in the diastolic period much nearer the end of the capillary (*i.e.*, North in the field of the microscope), indicating negativity of the base; each beat was now accompanied by a single variation S, indicating negativity of the apex. Ten minutes later the mercury had subsided South (indicating declining negativity of the base); each beat was now accompanied by a variation Sn, signifying:—

1st phase, apex negative (major phase).

2nd phase, base negative (minor phase).

Ten minutes later the variation was still diphasic; but the 1st phase had diminished, while the 2nd phase had increased.

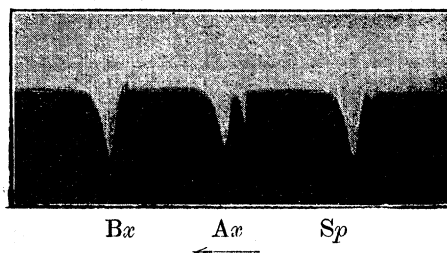
The changes accompanying the subsidence of injury negativity do not always follow the above regular form; in some cases the heart dies so rapidly that spontaneous beats giving the double phase do not re-appear, though mechanical excitation may still be capable of producing a contraction marked by a double phase; in other cases the double phase re-appears at first in an intermittent manner, most beats being still

marked by a monophasic and only a few by a diphasic variation, which, as time goes on, becomes gradually more frequent until it is established as the regular accompaniment to every beat.

Effects of double injury.—As stated above, the conversion of a diphasic into a monophasic variation can be effected at will by injury, and I have just said that the monophasic variation thus effected may gradually give place to a diphasic variation; it remains to add that this may sometimes be done at once by a second injury. Thus, for instance, in a heart giving a spontaneous variation SN (apex negative, base negative), this was replaced by a variation N (unbalanced negativity of base) after injury of apex, and this again was at once replaced by a variation SN after a subsequent injury of the base (*vide* Experiment 2). Apparently, the balance between the two led-off parts can be at least partially restored when, after one part has been injured, the second is similarly injured.

A triphasic ventricular variation.—The ventricular variation sometimes appears to be triphasic, and one might at first sight interpret it as being due to auricular negativity followed by the ordinary diphasic variation. This, doubtless, does frequently give a variation of such a character, but in certain cases a treble variation is undoubtedly caused by the ventricle alone. A variation *n*SN (apex to H_2SO_4 ; base to Hg) or *s*NS (apex to Hg; base to H_2SO_4) cannot be auriculo-ventricular, but must be an irregularity such that negativity of the apex manifests itself twice, once at the beginning and once at the end of the contraction of the ventricles. The most probable interpretation appears to me to be that we have to do here with a case of injured base; apex negativity manifests itself first and is not overcome by subsequent base negativity, which is only sufficient to more or less interrupt the predominant apex negativity; the latter thus appears to be twice manifested. The following photographic observation is one among several others which can, it seems to me, be thus explained.

Photo. 1.



Experiment 4.—*Kitten's heart*, excised 15 min. *post mortem*, April 13, 1888.

Apex to Hg. Base to H_2SO_4 .

Sp = a spontaneous variation *snS*.

Ax = a variation caused by mechanical excitation of the apex, *snS*.

Bx = a variation caused by mechanical excitation of the base, *nS*.

Another instance of the same kind, but in which negativity is twice marked at the base, is given in the note to Experiment 5.

It is possible that in some cases differences of temperature are accountable for irregular variations of this character. If, for instance, the base should happen to be warmer than the apex, the negativity of the latter will outlast that of the former, and, if an excitatory process should begin at the apex, it will possibly be twice manifested, once before the beginning and once after the termination of basal negativity. *Vice versa*, if the apex should happen to be the warmer, an excitatory process commencing at the base might be twice manifested at each ventricular contraction. Obviously, these suppositions require to be submitted to the test of experiment, and I intend to do so as soon as time will allow. At present, however, as will be seen, I am led to pursue the phenomena in another direction, and I have mentioned the supposition now only for the sake of completeness.

§ 3. *Observations on Animals with One or Both leading-off Electrodes applied to the Body at a Distance from the Heart.*

The observations to be described in this section lead up to those which will be discussed in Part II. They are the steps by which I gradually learned on animals what parts of the body are equivalent to leads-off from base and from apex of the ventricles. Instead of exposing the heart and leading off from it by both electrodes, I led off by only one electrode from the exposed heart and by the other from various distant parts of the body; finally, I led off by both electrodes from various distant points on the intact animal.

Experiment 5.—*Cat.* April 12, 1888. Death by decapitation. Five min. *post mortem*, heart exposed. Electrode A from apex to Hg. Electrode B from stump of neck to H_2SO_4 .

Spontaneous variations, SN alternating with SNS. Electrode B shifted from neck to base of heart.

Spontaneous variations, SN alternating with SNS. Thus it appears that the lead-off from the neck was equivalent to a lead-off from the base of the heart.

NOTE.—The alternation, SN and SNS, noticed in this case was observed to coincide with a well-marked bigeminal character of the contractions of the heart, which was beating slowly. The contraction of the base of the heart, upon which electrode B was applied, was evidently stronger at each SNS variation than at each SN variation. (S indicates base negative, N indicates apex negative.) Thus in this case the excitatory process originated at the base, and at every other beat when the base contracted more strongly, the negativity was twice manifested at the base. I have several times noticed this form of electrical disturbance.

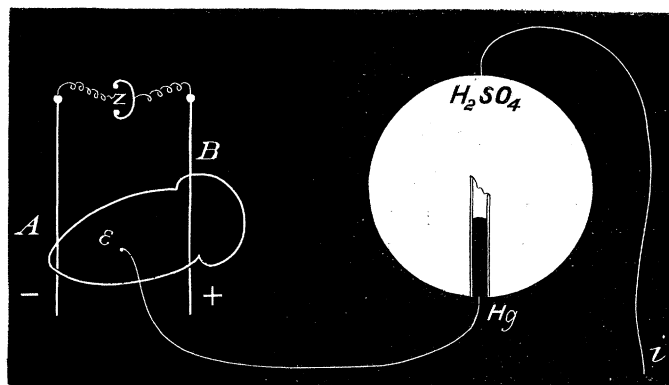
Experiment 6.—*Cat.* April 17, 1888. Killed by decapitation; heart exposed.

Apex to	Hg.	and Base to	H_2SO_4	Variation	N.
Apex to	„	„ Mouth to	„	„	N.
Base to	„	„ Mouth to	„	„	NS.
Apex to	„	„ Mouth to	„	„	N.
Base to	„	„ Mouth to	„	„	NS.
Mid ventricle to	„	„ Mouth to	„	„	NS.
Apex to	„	„ Base to	„	„	N.
Mid ventricle to	„	„ Base to	„	„	NS.
Apex to	„	„ Base to	„	„	N.
Apex to	„	„ Mid ventricle to	„	„	N.

Here again it appears very plainly that a lead-off from the mouth is equivalent to a lead-off from the base of the heart.

Complementary experiment.—In order to obtain some idea of the distribution of potential in surrounding parts when different portions of the heart are at different potentials, I took observations with the capillary electrometer, leading off from various parts of the body, and leading in the current of a Daniell cell by pins transfixing the heart at base and apex (*vide* fig. 4). The observations were taken on animals the day after death, left exactly in the position in which their hearts had been examined on the previous day.

Fig. 4.



Experiment 7.—*Cat.* April 17, 1888. Twenty-four hours after death. Heart transfixed at apex and base by pins constituting the electrodes of a Daniell cell. Direction of current as in diagram above. ϵ and i are the leading-off electrodes to mercury and sulphuric acid. The Daniell circuit is made and broken by a spring key, and the consequent variations of the electrometer observed when ϵ and i are applied to various parts of the body. The results are as follows :—

with	{	ϵ near A	the variation was S,	that is,	-	}
		i near B			+	
with	{	ϵ near A	,,	,,	S,	-
		i in mouth			,	+
with	{	ϵ to leg	,,	,,	S,	-
		i in mouth			,	+
with	{	ϵ near B	,,	,,	N,	+
		i in mouth			,	-
with	{	ϵ near B	,,	,,	N,	+
		i at leg			,	-

These variations were precisely such as might have been anticipated from theoretical considerations; they were reversed with reversal of the Daniell current; they are confirmatory of the supposition above made that a lead-off from the mouth is equivalent to a lead-off from the base, and that a lead-off from the lower extremity is equivalent to a lead-off from the apex. Experiments of a similar character with single induction shocks gave precisely similar results.

Experiment 8.—Kitten. April 21st, 1888. Death by chloroform.

1. Mid ventricle to Hg.	Mouth to H_2SO_4 .	Variation nS .
2. Mid ventricle to Hg.	Leg to H_2SO_4 .	,, nS .
3. Leg to Hg.	Mouth to H_2SO_4 .	,, sN .
4. Auricle to Hg.	Leg to H_2SO_4 .	,, s and ss or snS .

This experiment presents several points of interest. The third observation is the first which I have made upon an animal leading off the heart from two remote points, viz., leg and mouth. The variations with each contraction of the heart were small, but unmistakable. (It should be remarked that the chest was open and that circulation had ceased, the animal having been dead about ten minutes.) The variation was such as to indicate—

1st phase. Negativity at leg.
2nd phase. Negativity at mouth.

As will presently be shown, we have reason to admit the leg as indicator of the apex potential, the mouth as indicator of the base potential. Thus we have in this case—

1st phase. Negativity of apex.
2nd phase. Negativity of base.

In the fourth observation the auricles were contracting twice to each ventricular contraction; when the auricle contracted the variation was s , indicating negativity of

auricle ; when the auricle and ventricle contracted in sequence the variation was sS or snS ,* indicating

1. Negativity of auricle, viz., action of auricle.
2. Negativity of leg, „ „ of ventricle apex.
3. Negativity of auricle, „ „ of ventricle base.

Observations 1 and 2 were defective, inasmuch as the leading-off electrode from mid ventricle was shifted between the two observations.

Complementary experiment.—The animal was left *in statu quo*, and two hours later differences of potential were artificially established by pins inserted into the heart, through which were passed make or break induction shocks, or the direct current of a Daniell cell (*vide* fig. 4). The results were as follows:—

With induced currents. (Make current from apex to base ; break current from base to apex.)

		Variations.	
Mouth to Hg.	Leg to H_2SO_4	At make	S.
		„ break	N.
Leg to Hg.	Mouth to H_2SO_4	„ make	N.
		„ break	S.
Leg to Hg.	Heart near base to H_2SO_4	„ make	N.
		„ break	S.
Leg to Hg.	Heart near apex to H_2SO_4	„ make	S.
		„ break	N.

With the constant current. (Current from apex to base through heart.)

		Variation at closure.	
Leg to Hg.	Mouth to H_2SO_4		N.
Leg to Hg.	Heart near base to H_2SO_4		N.
Leg to Hg.	Heart near apex to H_2SO_4		S.
Mouth to Hg.	Heart near base to H_2SO_4		N.
Mouth to Hg.	Heart near apex to H_2SO_4		S.

On reversal of the current the variations were reversed.

The whole series of these variations was exactly as might have been anticipated. They were uniformly such as to indicate potential of the same sign in the vicinity of either pole as compared with the potential at more distant parts. It is worth again calling attention to the fact that potential at leg agreed with sign of pole at apex, while potential at mouth agreed with sign of pole at base, when the body was led off

* It is often very difficult by inspection to distinguish between such variations ; in the above case it was impossible to say whether the variation consisted of two movements in the same direction (sS), or of two movements in the same direction separated by a movement in the opposite direction (snS) ; the latter is probably the correct reading.

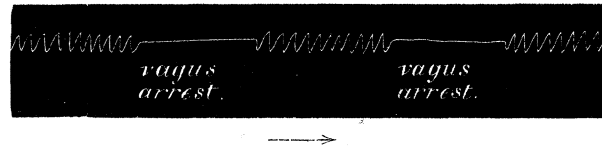
to the electrometer from leg and mouth. And I may add that I also experimentally verified the fact that, while potential at mouth agreed with sign of pole at base, potential at rectum agreed with sign of pole at apex.

In the preceding experiments the electrical variations of the heart were observed by leading off from remote points with the thorax opened, and with the heart therefore lying in contact with the tissues by its posterior surface only. The next step was to determine whether the variations can be observed on the intact animal, with the heart in contact with its normal surroundings.

Experiment 9.—Cat. April 23rd, 1888. Death by chloroform. Led off to the electrometer by electrodes in the mouth and in the vagina. Variations observed synchronous with the heart's beat, but too rapid to allow their character to be determined. Apparently each variation was double, but it was impossible to tell which was the first and which was the second phase, the rhythm being—

..... *snsnsns*

Both vagi exposed, isolated, and divided. Excitation by induced currents of either vagus abolished the variations, the right vagus being in this respect more efficacious than the left. After each period of arrest, the first movement of the mercury in the electrometer was closely watched; it was southwards (with mouth to H_2SO_4 and vagina to Hg); graphically expressed, the effects were—



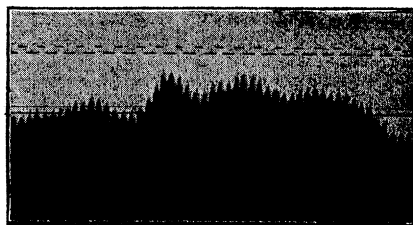
Similar results were obtained with the H_2SO_4 electrode transferred to the eyeball and with the subsequent transfixion of the heart by a pin connected with the Hg of the electrometer. This was between ten and fifteen minutes *post mortem*. The heart was now exposed by opening the thorax, and vagus effects were repeatedly obtained up to about half an hour *post mortem*, excitation of the right vagus being uniformly the more effectual. Towards the end of the experiment the following point was noted: excitation of the right vagus arrested the movements of the auricles and of the ventricles; excitation of the left vagus arrested the movements of the ventricles, and not those of the auricles, which continued to pulsate.* In both cases the move-

* This was one among a considerable variety of effects which vagus stimulation may produce upon the contractions of the Mammalian heart *post mortem*. I have seen vagus stimulation under these circumstances entirely without effect upon any of the four chambers, or followed by complete arrest of the whole organ, or by arrest limited either to the auricles or to the ventricles. I have also seen a delirium cordis entirely uninfluenced by, or entirely suspended during, vagus stimulation.

ments in the electrometer were arrested, showing that they were due to ventricular, and not to auricular, contraction.

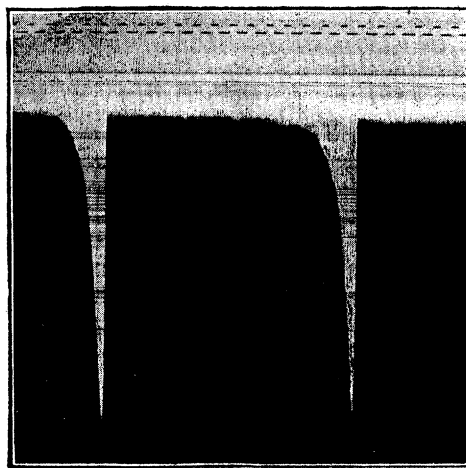
Experiment 9 is, in two respects, a typical one: it is representative of many others which I have made, in which the heart is led off from the mouth or from an eye, and from the rectum or from an inferior extremity; electrical variations are thus unfailingly demonstrated of a character which is illustrated in photo. 2.

Photo. 2.



Electrical variations of Cat's heart, with body intact, and led off from mouth to Hg, from rectum to H_2SO_4 .

Photo. 3.



Cat's heart, exposed immediately after death by chloroform, and injured at the apex by crushing. Led off from auricle to Hg, and from apex of ventricle to H_2SO_4 . Variations sSsssS (read from right to left; s = negativity of auricle, S = negativity of base of ventricles).

It also shows that the variation observed with peripheral leading-off points is exclusively ventricular. I have since repeatedly observed (with special facility when the auricles and ventricles have happened to beat at different rates) that, for the demonstration of any electrical change accompanying auricular contraction, it is necessary that one of the electrodes should be in actual contact with the auricles, and that, as soon as it is shifted to a distance, the auricular variation is lost. Photo. 3, taken with one electrode in contact with the auricle while the other was

applied to the apex of the ventricle, shows the comparative effects of auricular and ventricular events; it will not be surprising that, if both electrodes be applied to the periphery, only the latter event should be manifested.

With respect to the distribution of cardiac electrical potentials ascertained by the determination of "favourable" and of "unfavourable" leading-off points of the body, this, although it properly belongs to Part I., will be more conveniently considered in conjunction with the study of cardiac potentials on Man.

PART II.

Electrical Variations of the Heart on Man.

I now pass to the more important series of observations, to which those described in Part I. were the experimental preface.

It should first be recalled that, of the various points established in this preface, four in particular have a special bearing upon the due interpretation of the observations about to be described.

1. *The normal variation of the Mammalian ventricles is diphasic.*
2. *The variation can be observed on the intact animal by leading off from points of the body remote from the heart.*
3. *Under these circumstances the auricular contraction gives no electrical indication.*
4. *A lead-off from the mouth is equivalent to a lead-off from the base of the ventricles; a lead-off from the rectum or from a posterior extremity is equivalent to a lead-off from the apex.*

An investigation made last year upon my own person gave the following results* :—

Leading off from the surface of the body by the several limbs and from the mouth, I found that some combinations were favourable, while others were unfavourable,† to the demonstration of the cardiac variation. The favourable combinations were the following :—

Front of chest and back of chest.
Left hand and right hand.
Right hand and right foot.
Right hand and left foot.
Mouth and left hand.
Mouth and right foot.
Mouth and left foot.

* 'Journal of Physiology,' vol. 8, p. 229.

† I use the terms "favourable" and "unfavourable" for the following reason :—With a moderately sensitive electrometer no variation is seen with an unfavourable combination, and a small variation is seen with a favourable combination; with a very sensitive electrometer a small variation is seen with an unfavourable, and a comparatively large variation with a favourable, combination.

The unfavourable combinations were :—

Left hand and left foot.
 Left hand and right foot.
 Right foot and left foot.
 Mouth and right hand.

At that time I could not see the reason of this difference, and was surprised to find it so. There was, for instance, no apparent reason why a lead-off from mouth and right hand should be ineffectual, while a lead-off from mouth and left hand should be attended with a marked variation. And it was the most common and easily verified case. One electrode kept in the mouth while the other dips into a basin of salt solution, into which first the left hand then the right hand is plunged, yields a ready demonstration of a favourable in contrast with an unfavourable lead-off. Another illustrative contrast is furnished by leading off from hands and feet. If the right hand and either of the two feet be led off, a marked electrical variation is manifested at each pulsation of the heart; if now the left be substituted for the right hand, no variation is apparent, or at most a slight one.

Deferring the further enumeration of cases, I may at once offer the explanation of these apparently anomalous results.

The contraction of the ventricles is not simultaneous throughout the mass, but traverses it as a wave (at the present stage the direction of the wave of contraction is immaterial). Inequalities of potential, at different parts of the mass, are consequently established at the beginning and at the end of each systole. Or, to reverse the order of statement, the inequalities in question are proof of the passage of a wave of excitation. The distribution of these inequalities of potential is represented diagrammatically in fig. 5.

These data being transferred to the entire body, as in fig. 6, we have the dark portion $a, a, a \dots$ as the area in which the potential of A is distributed, and the light portion $b, b, b \dots$ as the area in which the potential of B is distributed.

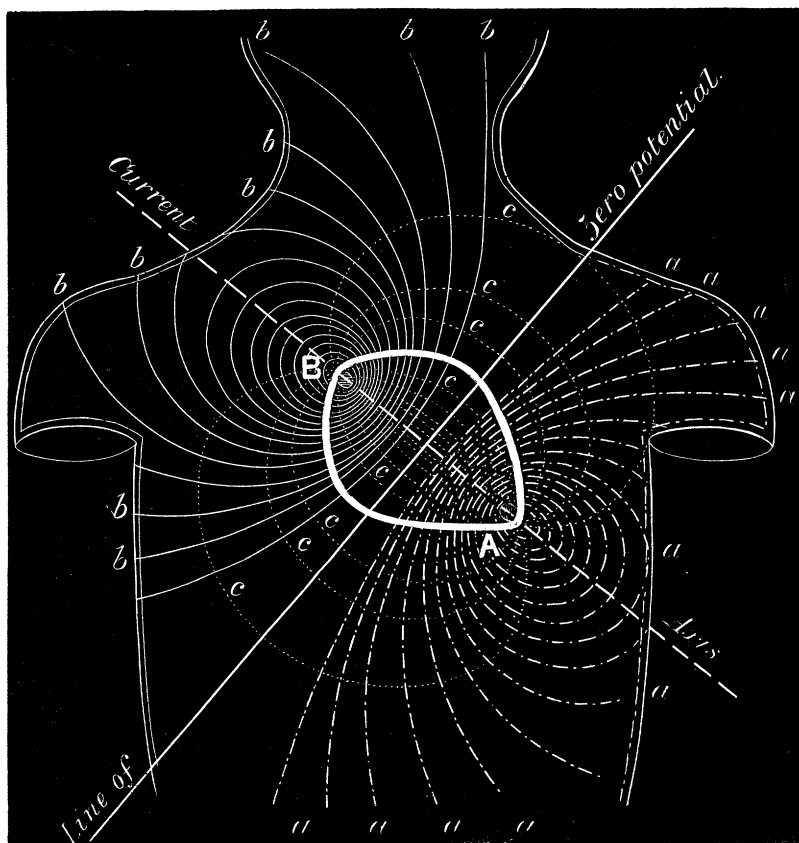
Electrical variations will be manifested when any two points a and b are led off; no electrical variations will occur when any two points a and a , or b and b , on the same equipotential line, are led off; small electrical variations will be obtained when two points a and a , or b and b , on different equipotential lines, are led off.

This is precisely what has been demonstrated in the experiments given above.

Points $a, a, a \dots$ are represented by the left arm, the left leg, the right leg, the front of the body, and by the rectum, &c. Points $b, b, b \dots$ are represented by the mouth, the eye, the right arm, and the back of the chest. And, if the reader will refer to the results given above, he will notice that variations have been observed when two dissimilar points (a and b) have been connected with the electrometer, while variations have been absent or faint when two similar points (a and a , or b and b) have been explored. The difference of result, when the mouth and the right hand

or the mouth and the left hand are joined to the electrometer, is now of obvious significance. Mouth and right hand are similar points b, b , mouth and left hand are dissimilar points b, a . The same remark applies to the contrasting results of exploration of one or other hand with either leg. The right hand and either leg are points b, a ; the left hand and either leg are points a, a .

Fig. 5.



A and B are two points of apex and base respectively.

A straight line between A and B represents the axis of current between A and B if any inequality of potential should arise between the two points.

The dotted lines $c, c, c \dots$ represent lines of current diffusion.

A straight line at right angles to the current axis represents the line of zero potential.

The broken lines $a, a, a \dots$ represent equipotential lines surrounding the point A.

The continuous lines $b, b, b \dots$ represent equipotential lines surrounding the point B.

To these results I may now add those obtained when one of the leading-off electrodes is in the rectum.

Favourable combinations are :—

Rectum and mouth.

Rectum and right hand.

Unfavourable combinations are :—

Rectum and left hand.
Rectum and right foot.
Rectum and left foot.

Thus we see that whereas in combination with a lead-off from the mouth the only unfavourable extremity is the right arm, this is the only favourable one in combination with a lead-off from the rectum; likewise, any one of the three other extremities constitutes a favourable combination with a lead-off from the mouth, and an unfavourable combination with a lead-off from the rectum. The reason is obvious: the mouth is a point *b*, the rectum is a point *a*; the mouth and rectum series of combinations with other points *a* or *b* are consequently the counterparts of each other.

The logical completeness of these experimental facts is further borne out by :—

1. Observations on Quadrupeds.
2. Observations upon two cases of *situs viscerum inversus*.

1. *Observations on Quadrupeds*.—The asymmetry in the distribution of potential in the Human body originating from the heart is not found in Quadrupeds so far as I have examined them. On Cats, for instance (*post mortem*, but of course only during the continuance of cardiac contractions), the following combinations were found to be favourable :—

Mouth and either posterior extremity ;
Eye and either posterior extremity ;
Rectum and either anterior extremity ;
Rectum and eye or mouth ;
Either anterior extremity and either posterior extremity ;

whereas the following were found to be unfavourable combinations :—

Mouth or eye and either anterior extremity ;
Rectum and either posterior extremity ;
The two anterior extremities ;
The two posterior extremities.

These results show that the distribution of potential from the heart occupying an approximately median position behind the sternum is in accordance with fig. 7.

2. Having ascertained the mode of distribution of potential on the normal Human body with the heart tilted to the left, I at once sought for a case of *situs viscerum inversus* with the heart tilted to the right. By the kindness of Dr. CHEADLE and of Dr. LEWIS I obtained the opportunity of examining two such cases (one male and one female), and found the differences from the normal exactly as expected, viz., the favourable combinations :—

Mouth and right hand ;
Left hand and left foot ;
Left hand and right foot ;

and the unfavourable combinations :—

Mouth and left hand ;
Right hand and right foot ;
Right hand and left foot.

The above results are diametrically opposed to those obtained on the normal subject. Those obtained with the combinations indicated below were identical* in the two cases, favourable combinations in both being :—

Mouth and right foot ;
Mouth and left foot ;
Left hand and right hand ;*

while in both we find the unfavourable combination :—

Left foot and right foot.

Detailed comment is needless ; the complete harmony of the facts stated will be clearly recognised in the tabular summary on p. 191 with the assistance of the appropriate diagrams (figs. 6, 7, 8).

In each instance of the entire series a favourable combination is formed when the electrometer is connected with two heteronymous points a and b, while an unfavourable combination is formed when the connection is made with two homonymous points a, a or b, b.

The above observations supply abundant proof that in the contraction of the heart there is a time during which the apex and the base are not iso-electric.

From our knowledge of the diphasic variation of the hearts of Animals, we are further assured that on Man the inequality shall be of a double character ; the part which first becomes negative is also the part which first ceases to be negative, the part which last becomes negative is also the part which last ceases to be negative, so that a diphasic change will occur in consequence of—

1. Unbalanced negativity where action commences.
2. Unbalanced negativity where action lasts longest.

What are the character and direction of the electrical changes observed on Man with the beat of the heart ?

In answer to this question, I shall select the most favourable cases for examination, *i.e.*, those in which the variation has presented itself in a well-marked and readable form. I have followed two channels of information, each presenting special advantages and disadvantages. Simple inspection of the capillary under a $\frac{1}{4}$ objective is the ready and convenient means of investigating a series of combinations. Photography of the oscillating column of mercury with simultaneous photography of cardiac move-

* The *direction* of the variation with the two hands is opposite in the two cases, as will presently be noticed.

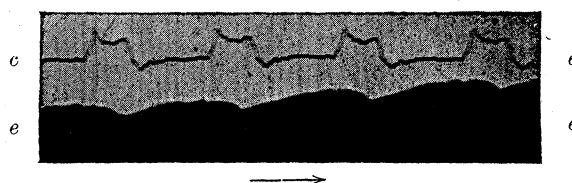
ments is the difficult but indispensable means for the close investigation and determination of special features in the rapid oscillations of the mercurial column.

By simple inspection I ascertained that *each beat of the heart is accompanied by a double movement of the mercury*, but I was unable to completely determine the character of this double movement. The whole movement consists of a comparatively large, prolonged portion, preceded by a small and extremely brief portion. There is no difficulty in determining the direction of movement as regards the second or major phase: the difficulty affects only the first or minor phase, which is so small and rapid as to appear with some instruments as a preliminary tremor, with others less sensitive I have failed to see it; but even with the most sensitive instruments which I have used I have failed to assure myself of its direction. I may, however, state at once that, as regards the second or major phase, I have always found its direction such as to indicate that any point *b* became negative to any point *a*. Simultaneous photographs of the double movement and of the heart's impulse show that the electrical precedes the mechanical event at whatever distance from the heart we choose to explore any two points *a* and *b*; they show, further, that in direction the first minor phase is opposed to the second major phase, being such as to indicate that any point *a* becomes negative to any point *b*.

By what has preceded, it has been defined that points designated *a, a, a* are in the region of apex potential and that points designated *b, b, b* are in the region of base potential.

The diphasic variation is, therefore, composed of a first phase indicative of *negativity of apex*, followed by a second phase indicative of *negativity of base*; this signifies that the excitatory process commences at the apex and lasts longest at the base, or, expressed in terms of mechanical action, that the contraction by which the ventricle discharges its contents commences at the apex and closes at the base.

Photo. 4.



Heart of Man. Led off to Hg from mouth, to H₂SO₄ from left foot, the variation is *nS*. *c, c* = cardiogram; *e, e* = electrometer line.

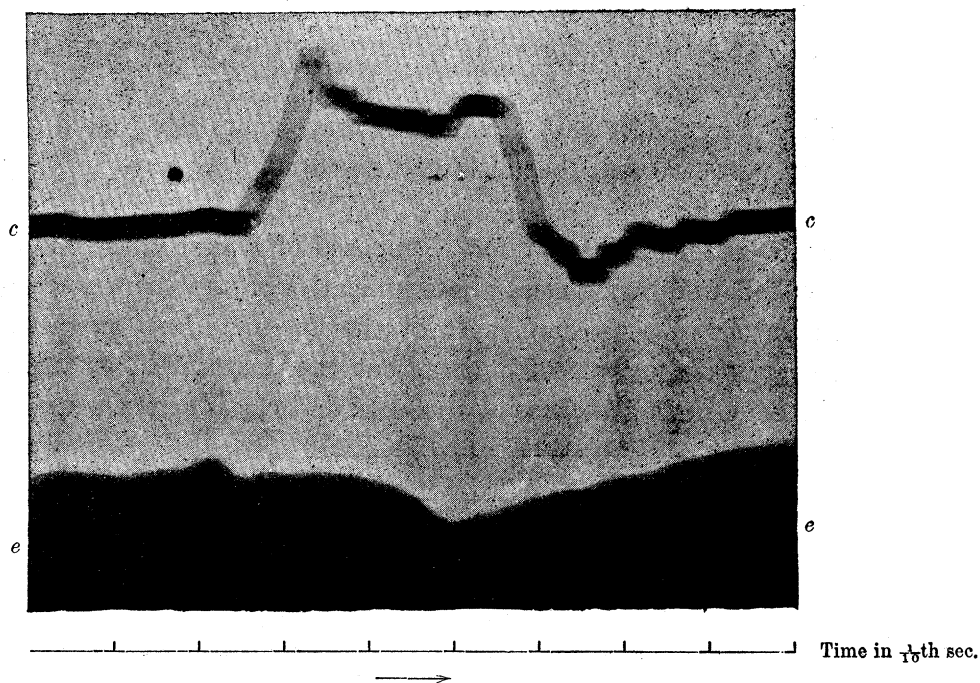
NOTE.—The rate of propagation of the excitatory state in the Human heart may be deduced from the time of culmination of the 1st phase, but it is obvious that an estimate thus derived can under the circumstances be no more than an approximation. The interval between the initial point and the maximum of the first phase is about $\frac{1}{50}$ second; taking the length of the ventricles at 10 cm., this gives for the rate at which the excitatory state travels a value of 5 metres per second, on the suppo-

sition that it passes in a straight line from end to end of the organ. I do not, however, attach much importance to this estimate, having regard to the nature of the data and suppositions involved. The initial point of the second phase, which is presumably the indication of declining negativity of the apex, occurs about $\frac{2}{10}$ second after the initial point of the first phase. The interval between the initial point and the maximum of the second phase is about $\frac{1}{10}$ second.

I regard these time relations as an indication that the contraction, while beginning at the apex, lasts longest at the base.

It has been suggested to me, as possible, that the contraction of the entire heart, commencing at the venous orifices of the auricles, is propagated thence by the auriculo-ventricular curtains and muscoli papillares to the apical vortex, and thence upwards to the base of the ventricles. This is a speculation for or against which I can at present see no positive evidence.

Photo. 5.

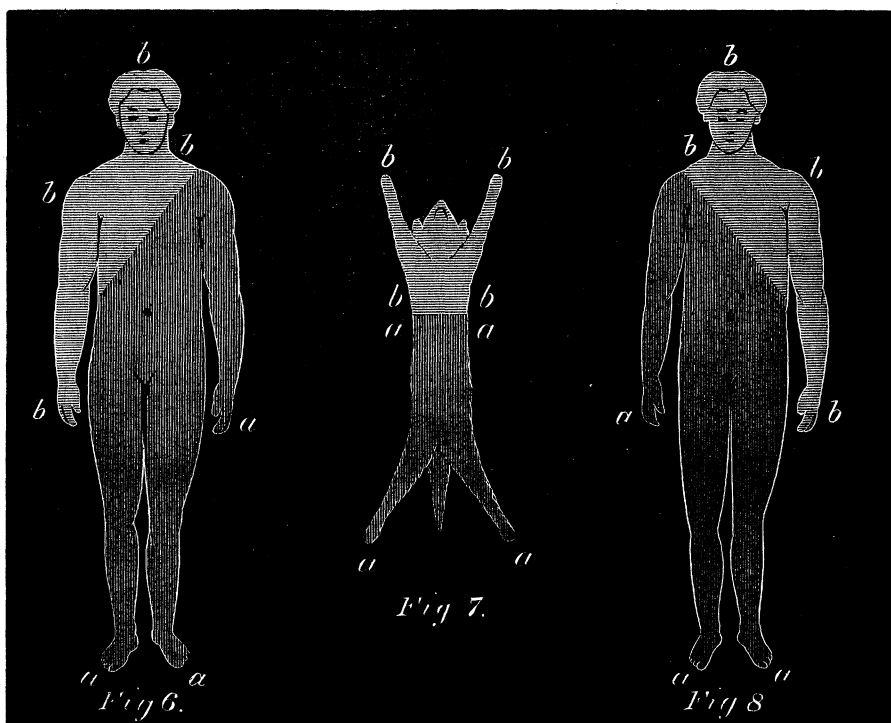


Enlargement ($\times 6$) of a single systole of photo. 4, to show:—

- I. First phase *n* (apex negative). II. Second phase *S* (base negative).
c, c = cardiogram; *e, e* = electrometer line.

			Normal. (Cons. fig. 6.)	Reversed. (Cons. fig. 8.)
1	Mouth to H ₂ SO ₄	Left hand to Hg	(s) N	—
2	" "	Right hand to Hg	—	(s) N
3	" "	Left foot "	(s) N	(s) N
4	" "	Right foot "	(s) N	(s) N
5	Left hand "	Right hand "	(n) S	(s) N
6	" "	Left foot "	—	(s) N
7	" "	Right foot "	—	(s) N
8	Right hand "	Left foot "	(s) N	—
9	" "	Right foot "	(s) N	—
10	Left foot "	" "	0	0

Figs. 6, 7, 8.



The capital letter N, or S, gives the direction of the second phase; that of the first phase, which by inspection could not be determined in each case, is represented by the small letters *n*, *s*, in parentheses.

The direction of the second phase is in every case such as to indicate B negative to A.

On reversal of the connections, as given above, the electrometer movement was in every case opposite to that indicated in the Table.

In one instance only (No. 5) the directions in the normal and reversed subjects are opposite; a reference to the figures 6 and 8 shows that it is the only instance in which two points, *a* and *b*, are simply transposed.

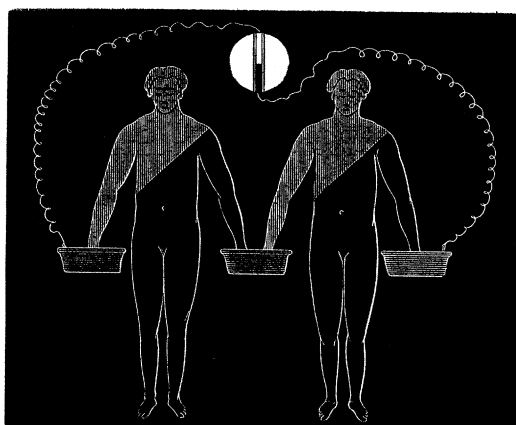
POSTSCRIPT.

(Added February 7, 1889.)

The observations recorded in Part I., § 3, can be carried out on Animals at liberty. I have done so on Dogs and on a Horse, with the result that any two anterior or posterior extremities constitute an "unfavourable lead-off," and that any one anterior in conjunction with any one posterior extremity forms a "favourable" combination. For purposes of demonstration I give the observations the following form:—A large Dog, trained to stand still with his feet in vessels of salt solution, is made to do so with a favourable and an unfavourable pair of extremities in connection with the electrometer, a commutator being interposed so that either pair can be switched on to the electrometer without delay or disturbance. The mercury pulsates distinctly or not at all according as connection is made with a favourable or with an unfavourable combination.

To the observations recorded in Part II. I have added the following:—If two persons are connected with the electrometer as shown in fig. 9, their contracting hearts form battery when they are synchronous, and the normal variations are seen reinforced in degree; when, on the other hand, the two hearts are alternating in action there is interference of their electrical variations; during this interference the movement of the mercury may be quite illegible, or the rhythm of each heart may be separately legible by following their separate pulses, the event depending upon the rates at which the two hearts are beating.

Fig. 9.

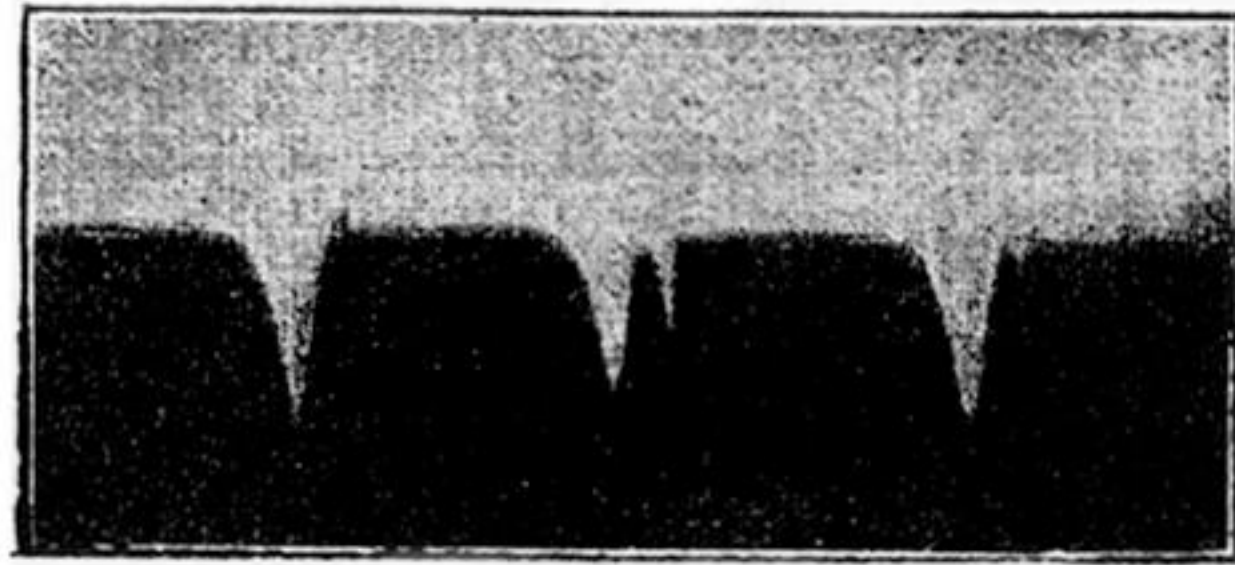


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Photo. 1.



Bx Ax Sp
 ←

Experiment 4.—Kitten's heart, excised 15 min. *post mortem*, April 13, 1888.

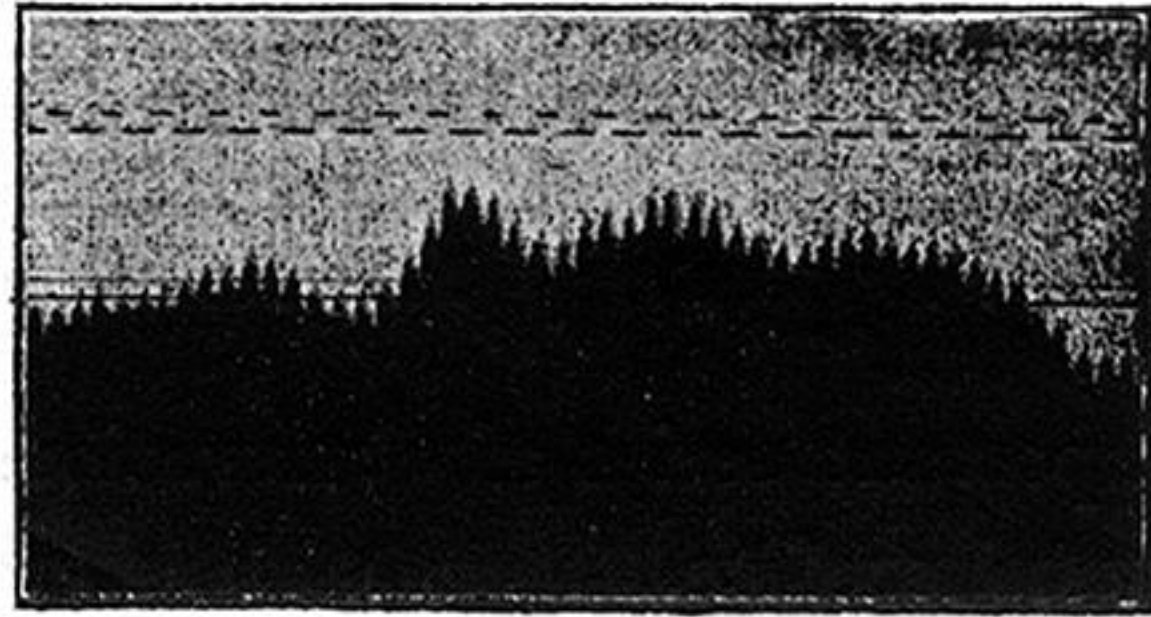
Apex to Hg. . . Base to H_2SO_4 .

Sp = a spontaneous variation *snS*.

Ax = a variation caused by mechanical excitation of the apex, *snS*.

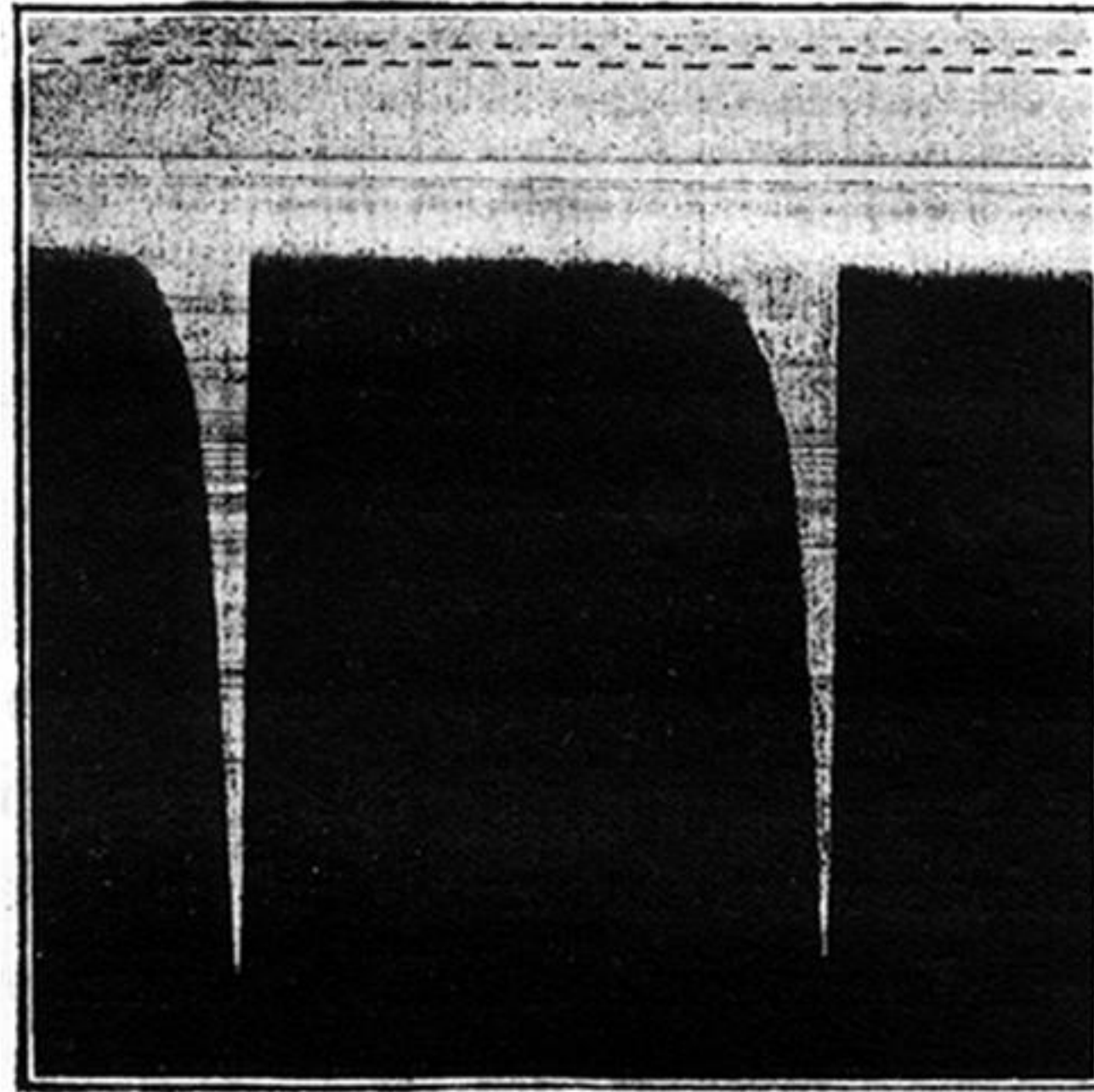
Bx = a variation caused by mechanical excitation of the base, *nS*.

Photo. 2.



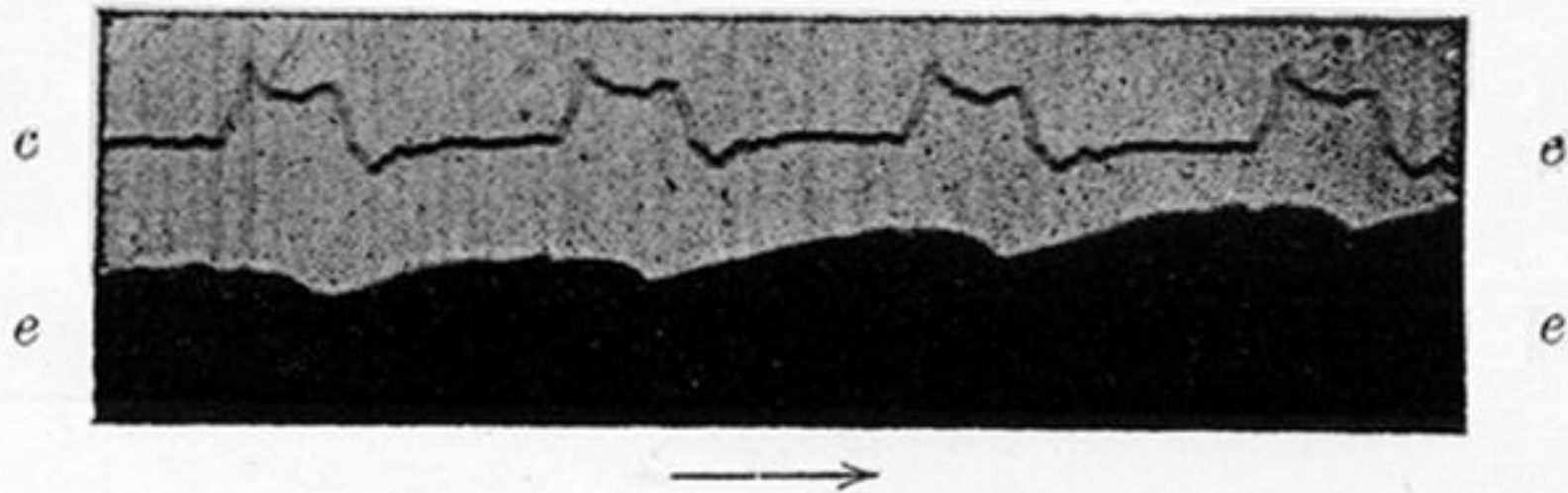
Electrical variations of Cat's heart, with body intact, and led off from mouth to Hg,
from rectum to H_2SO_4 .

Photo. 3.



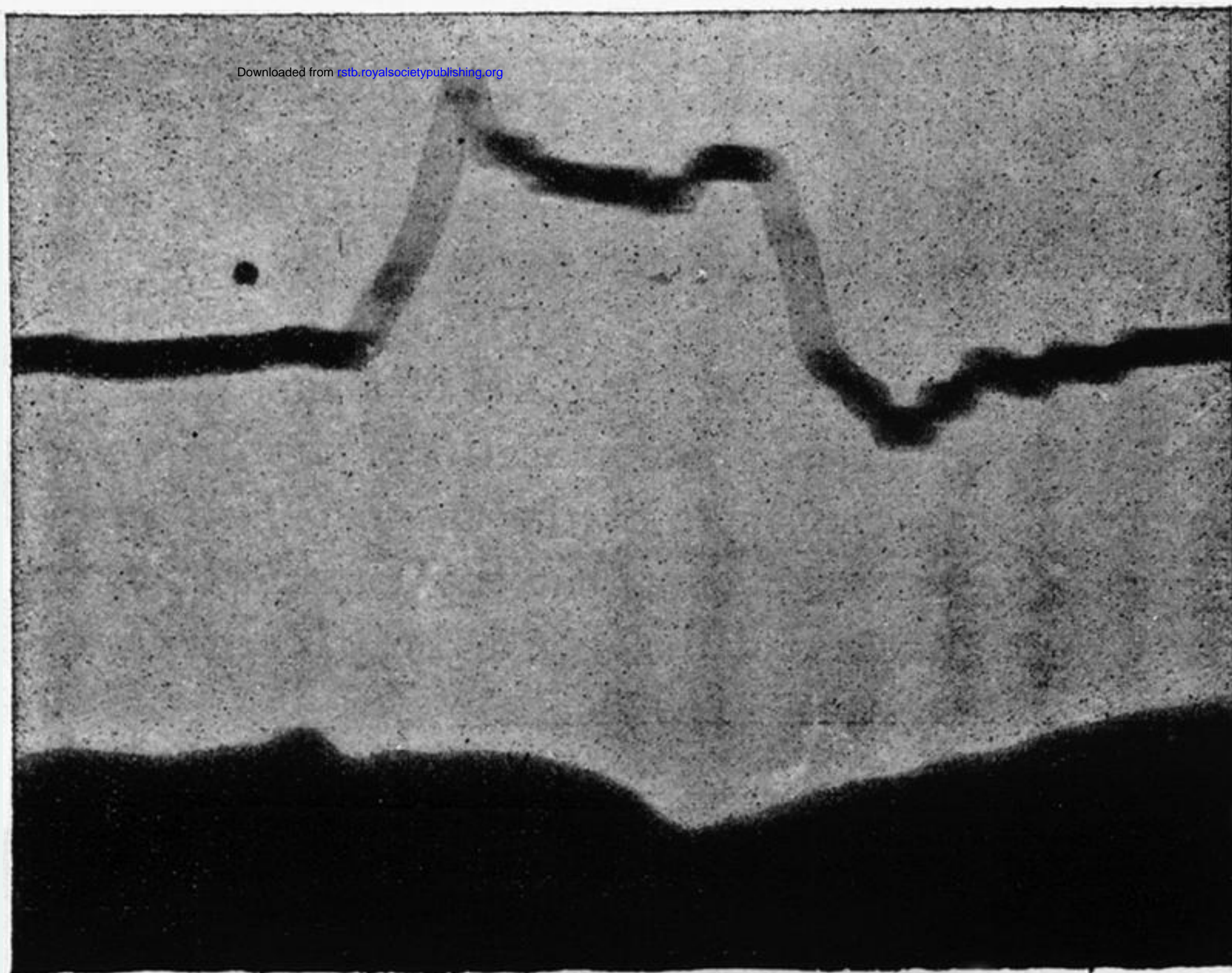
at's heart, exposed immediately after death by chloroform, and injured at the apex by crushing. Led off from auricle to Hg, and from apex of ventricle to H_2SO_4 . Variations *sSsssS* (read from right to left; *s* = negativity of auricle, *S* = negativity of base of ventricles).

Photo. 4.



heart of Man. Led off to Hg from mouth, to H_2SO_4 from left foot, the variation is nS . $c, c =$ cardiogram; $e, e =$ electrometer line.

Photo. 5.



Time in $\frac{1}{10}$ th sec.

→

Enlargement ($\times 6$) of a single systole of photo. 4, to show:—
First phase *n* (apex negative). II. Second phase *S* (base negative).

c, c = cardiogram; *e, e* = electrometer line.