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On the Action of the Excised Mammalian Heart

Augustus D. Waller and E. Waymouth Reid

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X. *On the Action of the Excised Mammalian Heart.*

By Augustus D. Waller, *M.D., and* E. Waymouth Reid, *B.A., M.B.*

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16.8.87

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§ I. *Introduction.*

Our attention was directed to the action of the excised Mammalian heart in the course of experiments relating to the electromotive action of various tissues, by our observation of the fact that the electromotive variations indicative of action outlast visible contractions; secondly, that the contractions of the excised organ are of an extraordinarily prolonged character.

We consequently pursued our observations (1) by the galvanometer, (2) by the aid of the graphic method, (3) by the capillary electrometer (LIPPMANN). By the galvanometer and by the electrometer we observed the electrical changes consequent upon spontaneous or provoked contraction, and in the absence of visible contraction. By the graphic method we determined the duration of spontaneous and of excited beats, and, with regard to the latter, the length of the latent period at various times after excision. Incidentally we also observed the time during which the rhythmic beat persisted after excision and the character of that beat under varying conditions of temperature. By both methods we were able in favourable cases to follow the progress of the wave of contraction, both spontaneous and excited. Our observations were made on the hearts of Cats, Dogs, Rabbits, Guinea-pigs, Rats, and Sheep.

§ II. *Tie during which spontaneous contractions continue after excision of the heart.—Mode of decline.*

The statements as regards this point which are most generally quoted are based upon the observations of CZERMAK and PIOTROWSKY, who found for the heart's beat of

Rabbits a minimum persistence of 3 minutes, a maximum persistence of 36 minutes; the mean of 60 observations was 11 minutes 46 seconds.* In the few observations which we devoted to this point we obtained durations considerably longer than the maximum given above.

We obtained, for instance, from three Rabbits' hearts the record of spontaneous beats during 72 minutes, 71 minutes, and 42 minutes respectively, and beats in response to excitation for as long a time as 1½ hours post mortem (Nov. 28). On Cats we recorded spontaneous beats for periods of 25½ minutes (Nov. 30), 23 minutes (Dec. 2). On a Dog we observed them for 2 hours after excision. These periods were longer than we anticipated, but they were exceeded by a figure given by ROUSSEAU in 1808, viz., 29 hours post mortem,—contractions on a guillotined Woman,† also by figures given by VULPIAN, 93½ hours (R. Auricle of Dog), 46½ hours (Auricles of Rat).† BROWN-SÉQUARD also gives some very high figures, 53 hours (Dog), 34 hours (Rabbit), 31 hours (Guinea-pig).‡

It must be observed, however, that in most cases the heart remained *in situ*, that generally the movements observed were of the R. Aur. or of the Vena Cava, and that, as far as can be gathered from the description, the observations were generally of the fibrillation of minute portions placed under the microscope. Our observations, except in the case of the Dog's heart above mentioned, were of complete ventricular contractions giving characteristic records. The heart was removed immediately after decapitation and placed upon an adjustable platform with a light lever resting upon it, and our results are comparable with those of CZERMAK and PIOTROWSKY,* with this addition that we recorded the movements.

Of fibrillation such as that spoken of by the French physiologists we took no account; we noticed it indeed, but did not follow it to its end.

We are not acquainted with any such published records of the movements of the excised Mammalian heart, we therefore submit some examples illustrative of the usual mode of the decline and of its ordinary modifications at various times post mortem. (*Vide* Tracings 1, 2, 3, 4. Tables A, B.)

1. Generally speaking, the decline is fairly regular as to force and frequency of contraction; force of contraction declines, however, more rapidly in the first moments after excision than at later periods (Tracing 1); frequency of contraction diminishes throughout the observation regularly at first (Tables A and B); at a later period the contractions are at long and irregular intervals (Table D).

2. The supervention of a bigeminal character is frequent as a regular irregularity. Sometimes the beat is bigeminal from the instant of excision to the end of observation, sometimes it is uniform at first and gradually becomes bigeminal. The bigeminal

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* 'Wien, Akad. Sitzber.,' vol. 25, 1857, p. 431.

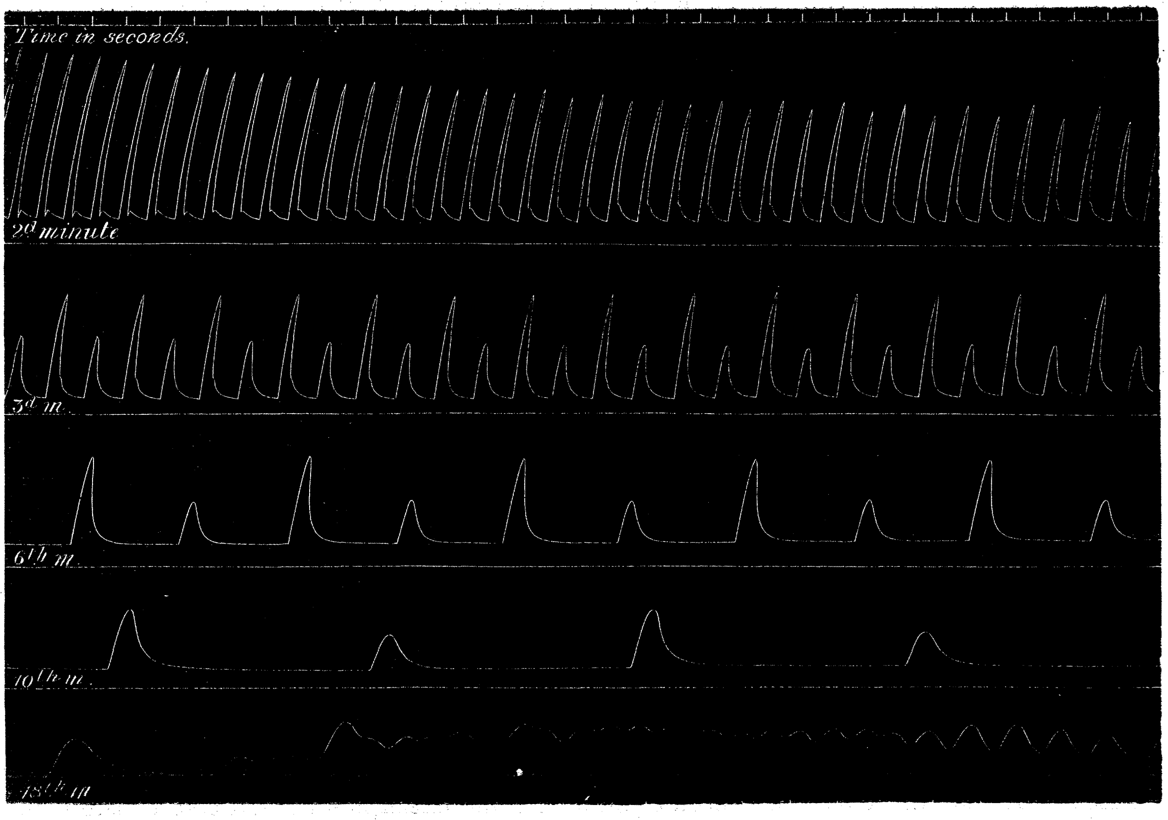
† 'Comptes Rendus,' 1858, p. 3.

‡ 'Journal de la Physiologie,' vol. 1, 1858, p. 357.

character is more pronounced at the base than at the apex, and may be entirely absent from the auricle when it is present in the ventricle. (Tracings 1, 2, 3.)

3. A third feature is sometimes very marked in such cases where the decline in force and frequency is not regular. A pause of unusual length is followed by a beat of unusual height (Table C, Tracing 4). This sign of restorative action during rest occurred with very different frequencies. It is noteworthy, however, that in bigeminal tracings stronger contraction is not preceded by a longer pause, nor the weaker contraction by a shorter pause.

Tracing 1.



Cat's heart. Record begins 1 minute, ends 18 minutes, post mortem. Dicrotism supervened at end of 1st minute, and continued so until 18th minute, when delirium cordis supervened. Reduced.

Note.—The tracings on smoked paper were taken on a cylinder having three rates of revolution. All tracings read from left to right.

1. Quick rate, 1 mm. of surface = ·004 second. 2. Middle rate, 1 mm. of surface = ·022 second.
3. Slow rate, 1 mm. of surface = ·15 second.

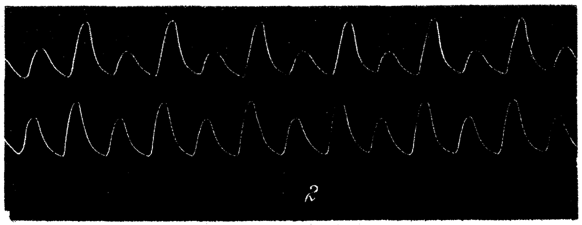
Tracings 1 to 8 inclusive are taken with the slow rate. Tracings 9, 10, 11, 14, and 15 inclusive are taken with the middle rate. Tracings 12 and 13 inclusive are taken with the quick rate.

4. Beats in groups are sometimes seen as an irregularity (Tracing 4).

5. Extreme irregularity in the form of irregular rapid fibrillation (delirium cordis) occasionally occurred (Tracing 1).

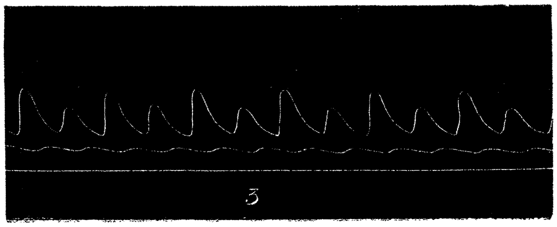
6. In the first few minutes after excision the sequence of auriculo-ventricular

Tracing 2.



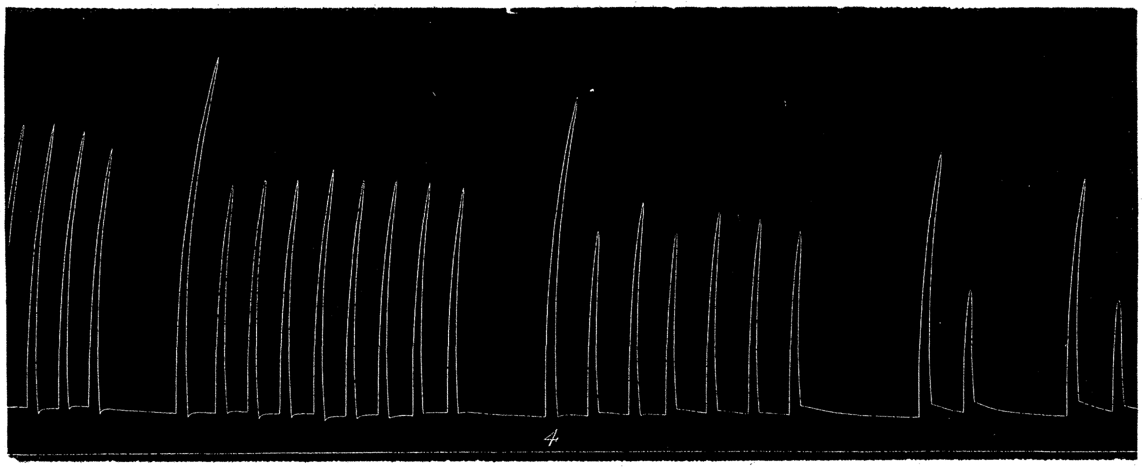
Tracing of Cat's hearts, 5 minutes post mortem, showing bigeminal character of beat, taken by a double cardiograph consisting of two levers resting upon the ventricle near the base and near the apex; the bigeminal character is more marked near the base than near the apex.

Tracing 3.



Rabbit's heart; double cardiograph, consisting of two levers resting on auricle and ventricle; the bigeminal character is evident in the ventricle, while it is absent in the auricle. (We have never seen it in the auricle.)

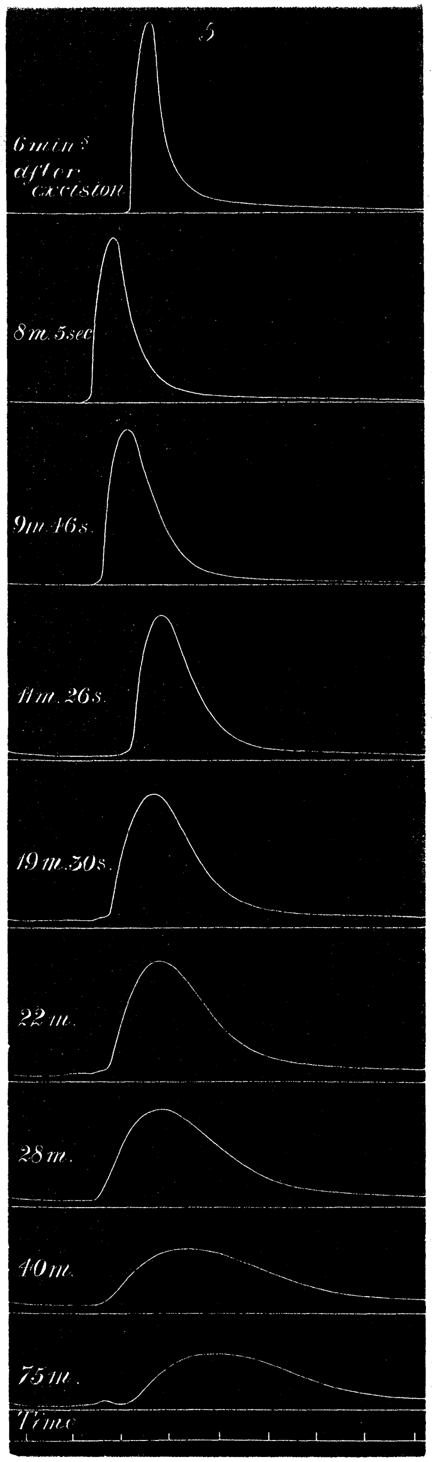
Tracing 4.



Cat's ventricle; record commences 3 minutes post mortem; shows ventricular contractions in groups separated by pauses, each pause being followed by a contraction of unusual strength.

action was normal. Later several auricular beats occurred to one ventricular beat (Table D). Later still the ventricular beat was quite independent of the auricular beat. We incidentally observed the ventricular action to outlast the auricular.

Tracing 5.



Contractions of the excised Rabbit's heart from the 6th to the 75th minute after excision; showing increasing prolongation of the ventricular contraction.

The two points to which we specially directed our attention at this stage were the changes in the duration of the ventricular contraction, both spontaneous and excited, and in the length of the latent period of stimulation. With regard to previous observations, we know of none, with the exception of a very imperfect datum consigned in LANDOIS' "Physiologie," 1880, p. 95, where he gives the maximum duration of the systole of the excised Rabbit's heart as ·48 second, the normal duration being ·25. Of the period of latent stimulation we have nowhere found any mention.*

§ III. *Duration of systole in the excised heart.*

Our observations show that from the moment of excision the ventricular contractions steadily increase in duration to a maximum of about 6 seconds, the normal duration being about ·3 second. This statement holds good for hearts of Cats, Dogs, and Rabbits removed and observed at ordinary room-temperature (16° Cent.). The hearts of these animals at 16° Cent. give contractions far exceeding in duration that of the contraction of the Frog's or Tortoise's heart at the same temperature. Table A and Tracing 5 are illustrative of this statement.

The chief cause of this change is an alteration of temperature, the heart *in situ* being at a temperature of 37° to 40°, while the temperature of the room was between 15° and 18°, or during the winter as low as 12°. That the temperature factor was

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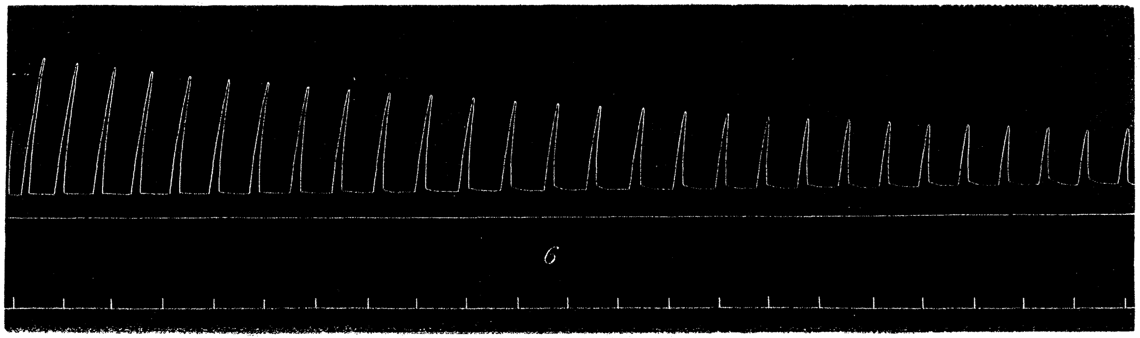
* Three times in the course of our observations we noticed that immediately after decapitation the diaphragm on the left side contracted simultaneously with the beat of heart. In one case the contractions of the diaphragm were so strong as to bend the body of the animal to the left side with each beat of the heart. We supposed that the phenomenon was one of secondary contraction, the left phrenic being excited by the electromotive change of the heart at each contraction. The contractions of the diaphragm were at once arrested by section of the phrenic below the heart.

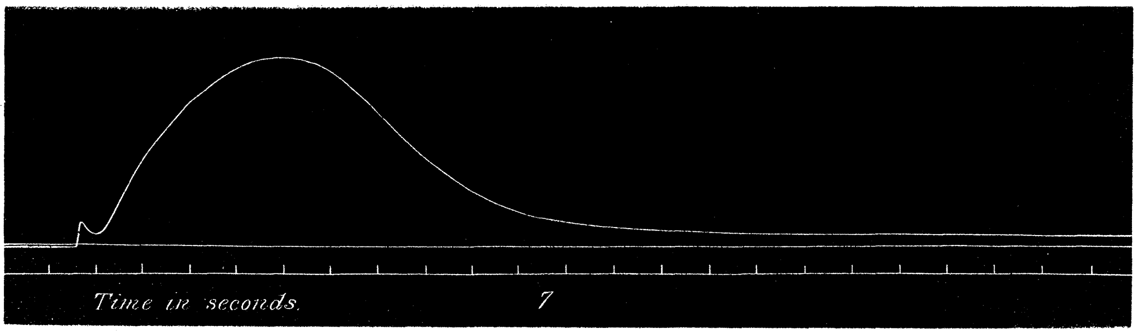
the important one was shown, 1st, by placing the heart in a constant temperature chamber at 40°, 2nd, by placing it in a chamber surrounded by ice. In the first case the characteristic prolongation was almost entirely absent, in the second it was greatly exaggerated. (Tracing 6.)

Tracing 6.



Beats of Dog's heart in warm chamber at 38° C.; the decline is rapid and there is no appreciable prolongation of the ventricular contraction.

Tracing 7.



Effect of cold (8° C.) on ventricular contraction of a Kitten's heart; the latent period is about ·5 second, the duration of contraction is at least 10 seconds (this is the greatest prolongation that we have observed).

We do not think, however, that the temperature is the only condition involved, though it is certainly the chief one, for we have, as a rare exception, observed an alternately longer and shorter contraction, without of course any possible alternation of temperature, and we have found that the hearts of very young animals are more susceptible of this modification than those of fully grown animals. (Tracing 7.) An example of the excessive prolongation of which the contraction is capable under the influence of low temperature is furnished by Tracing 8.

In the course of our experiments regarding the effect of temperature upon the heart's contraction, we made observations showing the great susceptibility of the Mammalian heart to cold, and the preservative influence of cold upon its capacity for action. The effects of cold (surrounding by melting ice) are, in the order in which they occur, 1st, lengthening of the spontaneous contractions; 2nd, abolition of the spontaneous contractions; 3rd, diminished excitability to mechanical* stimuli, with lengthening in the period of latent stimulation; 4th, abolition of excitability to all stimuli. The full effect of cold having been produced, viz., total abolition of contractility, spontaneous and provoked, the application of warmth (by surrounding the vessel containing the heart with water at 40° C.) can restore contractility, spontaneous as well as provoked, the latter reappearing before the former. This abolition of contractility by cold, and its restoration by warmth, may be repeated more than once; we have repeated it as many as three times. The following experiments illustrate these statements:—

Experiment I.—*Kitten's heart.* December 5th, 1885. Record began $1\frac{1}{2}$ minute after decapitation. Heart in small crucible surrounded by ice. The heart at first gave 11 beats during the first 7 seconds; it subsequently gave strong contractions at very long intervals (about 20 seconds) for a period of $4\frac{1}{2}$ minutes.

It continued excitable for 6 minutes longer. During the last minute of this period the prolongation of the latent period and of the contraction were excessive, greater than any we had hitherto observed; the latent period was about 2 seconds, and the length of contraction was so great and its fall so gradual that it could not be exactly measured; it is reproduced in Tracing 8. Its rise to a maximum occupies nearly 4 seconds. At end of 12 minutes post mortem, it was no longer excitable.

The ice was removed and replaced by warm water at 40° C. The heart became excitable, but spontaneous beats did not return.

The latent period was so short that we did not measure it.

The duration of contraction was shortened, and it was noteworthy that the ascent of the curve was rapid while its descent was still gradual.

Experiment II.—*Rabbit's heart.* December 5th. The record began 1 minute after decapitation, the heart being in a cup surrounded by ice.

It only beat for $1\frac{1}{4}$ minute, giving altogether 13 spontaneous beats.

It continued excitable to direct stimulation for about 3 minutes longer.

After the heart had ceased to be excitable it was removed from the influence of the ice, and was subjected to that of warm water at 40° C.

Within 1 minute of application of warmth the heart recommenced to beat *spontaneously*—at first slowly, afterwards more rapidly.

Excited contractions followed so rapidly upon mechanical excitation that we could not measure the latent period with the rate of revolution employed.

Minutes after application of warmth.	Frequency of beat.
3 min.	8 beats per 15 seconds.
5 ,,	6 ,, ,, ,, ,,
8 ,,	3 ,, ,, ,, ,,
11 ,,	2 ,, ,, ,, ,,
14 ,,	2 ,, ,, ,, ,,

Observation stops 30 minutes post mortem, the heart still beating feebly.

It is noteworthy that after the application of cold during 10 minutes, by which spontaneous contrac-

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* We found mechanical stimulation more effectual as well as more convenient than stimuli from the induction coil.

tions and excitability were entirely abolished, the heart, under influence of moderate warmth, recovered so as to beat for 20 minutes, giving during that period more than 150 beats.

Experiment III.—*Small Rabbit.* December 18th, 1885. Decapitated at 12. Heart excised and placed in porcelain crucible surrounded by ice.

Time.	
12.2.	Record begins.
12.7.	Spontaneous beats have ceased.
12.9.	Excitability has ceased.
12.13.	Warmth applied (water at 40°).
12.14.	Spontaneous beats renewed.
12.20.	Cold applied (melting ice).
12.24.	Spontaneous beats ceased.
12.26.	Excitability ceased.
12.28.	Warmth applied (40°).
12.28–30.	Spontaneous beats.
12.40.	Cold applied (0°).
12.44.	Excitability ceased.
12.48.	Warmth (40°).
12.50.	Spontaneous beats.
12.55.	Excitation, followed by "delirium."
1	Cold applied for 10 minutes; quite inexcitable.
1.10.	Warmth applied; a single spontaneous beat.
1.20.	Heart quiescent and inexcitable.

Remark.—The excitability of the heart was three times in succession abolished by cold and restored by warmth.

We have gone further than this: we have frozen the heart until it was quite hard by placing it in a capsule surrounded by ice and salt, and have observed its spontaneous contractions when it has been thawed.

Experiment IV.—December 19th, 1885. A Cat was decapitated at 11.59, the heart quickly excised, and placed in a porcelain capsule surrounded by ice and salt; by 12.14 it was frozen hard; it was removed from the freezing mixture and placed in another capsule surrounded by water at 42° C. It commenced to beat spontaneously at 12.24½, and a lever was at once placed upon it, and a record taken on the smoked cylinder. 20 spontaneous beats were recorded, and the heart remained excitable to the prick of a needle till 12.33.

We have gone further still: we have left the freshly excised heart in a freezing mixture for 3 hours. Its contractility did not, however, return. The heart's beat was recorded on the smoked cylinder in all the above experiments.

Warmth, notwithstanding its restorative effect upon a cooled heart, is not favourable to the long persistence of its rhythmic contraction after excision, nor even if it be left in the body of the recently killed animal. We have noticed at the outset that the excised and cooling heart usually beats for a longer period than a heart left in the body; we observed later that an excised heart beats for only a very short period in the warm chamber at 38° C., and we may add that a small, and therefore rapidly cooled, heart (Rabbit) outlasts a large and slowly cooling heart (Sheep). As of other tissues, the excitability of the heart is great, and its decline rapid, at high temperature, while excitability is small, and decline slow, at low temperature.

§ IV. *The latent period of stimulation.*

Our observations show that there is a general correspondence between the duration of contraction of the ventricle, and its excitability, and the length of the latent period of stimulation.

In general, the correspondence is such that, with the lengthening of the contraction, excitability decreases, and *vice versâ.* That these effects are in the main dependent on temperature is shown by the fact that they can be altered at will by variations of the temperature of the surrounding medium. Thus we may alternately obtain with the same heart, 1st, long contraction, long latent period, and obtuse excitability, or 2nd, short contraction, short latent period, and acute excitability, at (1st) lowered or (2nd) heightened temperature respectively.

A heart removed from the body, and examined at 15° to 18° C., shows the gradually increasing changes characteristic of lowered temperature; independently of this factor, however, quite similar changes supervene which are to be referred to the natural decline of action from the life normal to the death zero. This decline is, as for other tissues, not instantaneous, but gradual, and characterised by gradually increasing sluggishness, first, of spontaneous action, and secondly, of responsive action. The complete elimination of the temperature factor did not enter into the plan of our observations, and we give no experiments in support of the above general proposition, although we have observations sufficient to justify our assertion of it as of an experienced fact, not merely of an untested truism. The fact which we desired to clearly demonstrate is the great sluggishness of action to which the Mammalian heart may be reduced before action is extinguished; so that its processes, normally far more rapid than those of the cold-blooded heart, become so protracted as not only to equal, but to exceed, the latter in slowness of accomplishment. Our observations furnished the demonstration, and clearly show that the Mammalian heart is more susceptible to differences of temperature than is that of the Frog.[*]

Of the statements regarding the three features referred to above, viz., length of systole, length of latent period, degree of excitability, the first has been considered in § 1; with regard to the third, we have no evidence to give beyond the statement that

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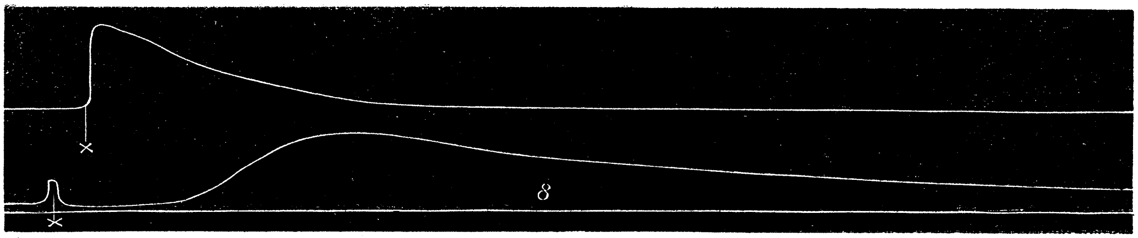
[*] Normal duration of Frog's systole at
27°—0·9
24°—1
21°—1·3
18°—1·6
15°—1·9
12°—2·1
BURDON SANDERSON, 'Journal of Physiology,' vol. 2, p. 384.

Frog's systole ten times as long at 3° to 7° as at 18° to 20°. HERMANN, vol. 4, p. 372.

we could roughly estimate excitability by the greater or less energy with which a mechanical stimulus is required to be applied to provoke contraction; with what we have termed "acute" excitability, the lightest superficial touch was sufficient to discharge a beat; with "obtuse" excitability, pricking or scratching with a needle was required to produce any effect; between these two extremes we could and did test gradations which justified our statement. To the second feature, viz., length of latent period, we paid particular attention; a summary of our results is given in Table E, from which it appears that the latent period is prolonged with prolongation of the heart's contraction. There is a rough relationship between the two magnitudes, the former being to the latter about one-tenth; but the relationship is only a rough one, owing to the fact that when the contraction is very prolonged it is impossible to determine where it ends on the tracing, and its true time-value cannot, therefore, be exactly given. But the chief point is unmistakably clear: the latent period of stimulation may be extraordinarily prolonged, a very usual length being about ·5 sec., and an exceptional maximum being as great as 2 secs. (see Tracing 8). This, as far as we know, exceeds any value that has been observed for the Frog's heart, in which the latent period is usually between ·1 and ·2 sec.

Tracing 8.



Rabbit's heart; effects of cold and heat on latent period; in the cooled heart the latent period is about 2 seconds, the ventricular contraction rises slowly to its maximum; in the case of the warmed heart the latent period is very short, and the ventricular contraction very rapidly reaches its maximum; the decline is in both cases gradual, most so in the case of the cooled heart. The heart was placed in a porcelain capsule and cooled or warmed by being surrounded by ice or with water at 40° C. × denotes instant of stimulation.

The above remarks are applicable to the ventricular beat. We have also made one or two observations on the auricular beat with analogous results, viz., prolongation of the latent period and of the duration of contraction.

That lowered temperature is the chief factor in the prolongation of the latent period as in that of the duration of the contraction, we learned by experimentally varying the temperature, alternately cooling and warming the heart.

The effects as regards the latent period are especially striking; the same heart may respond to a stimulus only after a period of one second or more while it is in a relatively cold medium (12° to 0° C.), and when this is replaced by a relatively warm medium (38° to 40° C.) response becomes almost immediate.*

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* We use this indefinite expression because our tracings were taken on a slow-rate cylinder, and

§ V. *The Wave of Contraction.*

It is not known whether or no there is any wave of contraction in the normal systole of the Mammalian heart, nor has it hitherto been sought for in the excised organ. There are no data to show whether, in the spontaneous or excited beat of the heart, its individual elements contract simultaneously or successively.

Our knowledge of the wave of contraction in the heart rests entirely upon observations made upon the excised organ of cold-blooded animals (Frog, Tortoise); the method employed has been to follow by rheotome and galvanometer the electromotive signs of an excitatory state at points more or less distant from a point of excitation. By this method, as applied by ENGELMANN, MARCHAND, and BURDON SANDERSON, the diphasic variation indicative of the passage of a wave of negativity has been plotted out, and its time-relations determined; the results thus obtained have been by BURDON SANDERSON controlled and confirmed by photo-electrometric records. The velocity of the wave thus determined has been given at 20 to 40 mm. per sec. (ENGELMANN), 100 mm. per sec. (MARCHAND), 125 mm. per sec. (B. SANDERSON and PAGE).* These results apply to the excited beat of the "stanniused" Frog's heart. With regard to the spontaneous beat of the Frog's heart, a double variation indicative of negativity, first at base, then at apex, has been seen, and is properly received as evidence of the passage of a wave from base to apex in the spontaneous systole of the excised heart. No attempt has been published, as far as we know, to determine a wave of contraction in the beat of the Mammalian heart, whether spontaneous or excited, nor has the wave of contraction been recognised on the heart of any animal by a mechanical method analogous to that which AEBY † first applied to skeletal muscle.

We have examined the Mammalian heart for the phenomenon in question both by electrical and by mechanical methods. Deferring consideration of our results by the galvanometer and electrometer, we here briefly give the results of our application of a mechanical method, viz., application to the heart of a double myograph so as simultaneously to record the movements of its different parts. With regard to the instrument, we need only say that it consisted of two levers, 1 to 8 cm. apart, which were applied to the excised heart just as in AEBY'S experiment they are applied to voluntary muscle. It will be well to give separately (*a*) the results we obtained upon the quiescent heart by excitation nearer to one or other of our levers; (*b*) the results we obtained on spontaneously beating hearts.

(*a*.) Excitation of a quiescent heart, upon which rested the two levers of our double cardiograph, at once revealed the fact that the total coordinated contraction of the

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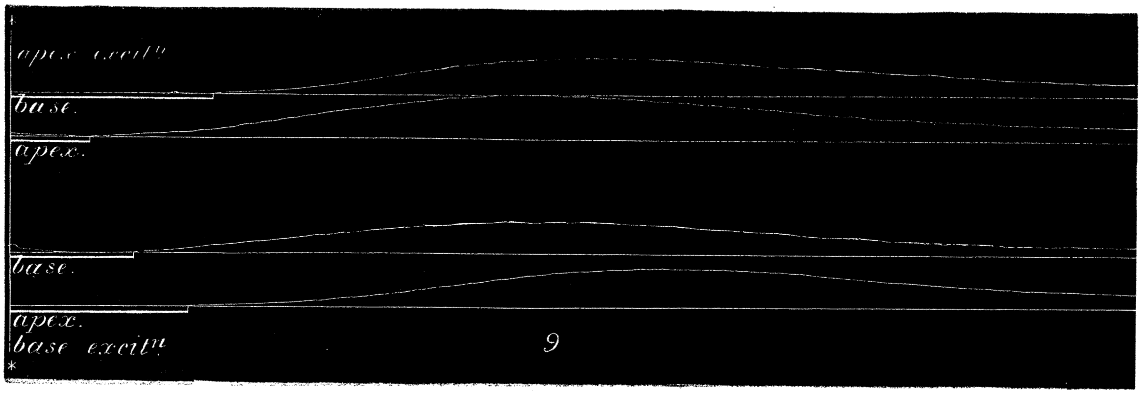
because we used mechanical stimuli. This was necessary, but did not give us the opportunity of taking fine measurements of time, which indeed we did not require.

* ENGELMANN; PFLÜGER'S 'Archiv f. Physiol.,' vol 17; MARCHAND, *ibid.*, vol. 15 and 17; BURDON SANDERSON and PAGE, 'Journal of Physiology,' vol. 2, p. 384.

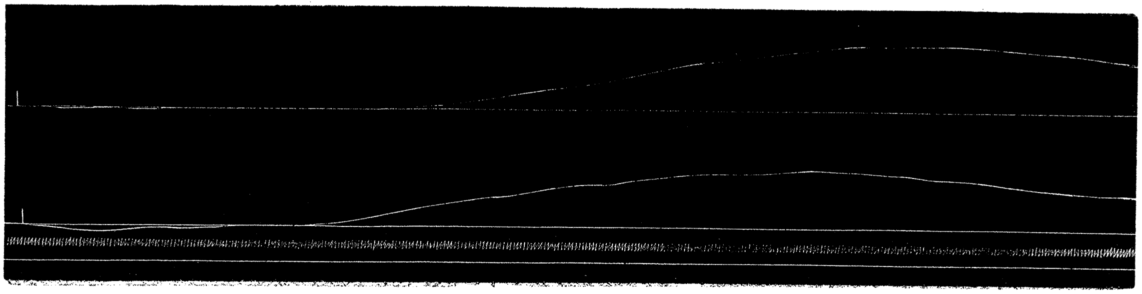
† AEBY, 'Archiv Anat. Physiol.,' 1860, p. 253.

Tracing 9.



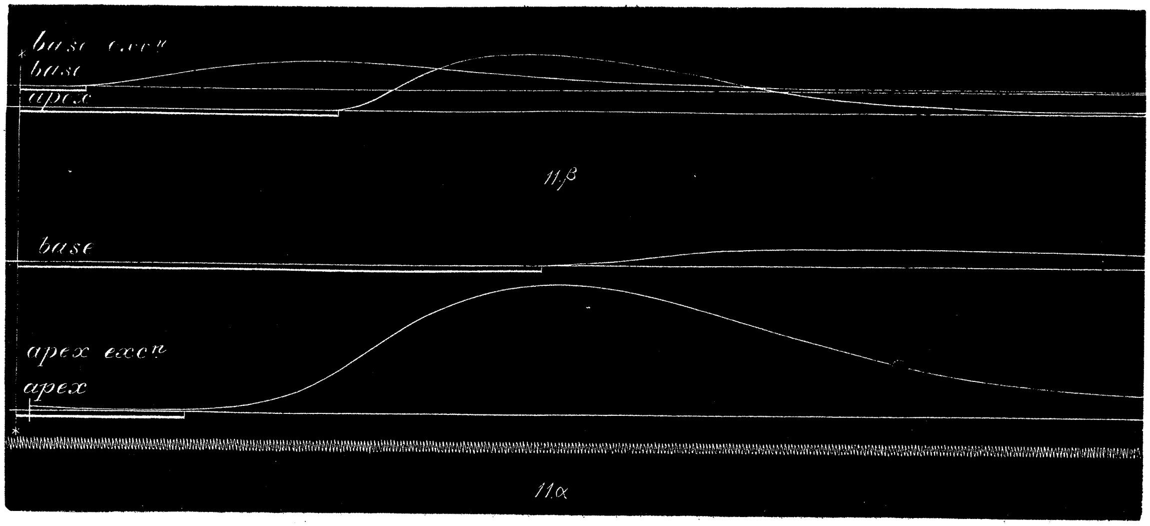
Wave of contraction in ventricle of Cat's heart, 30 minutes post mortem; taken by double cardiograph; mechanical stimulation near the base gives a ventricular contraction, in which the lever resting on the base commences to rise earlier than the lever resting near the apex; mechanical stimulation near the apex causes the lever near the apex to rise before the lever near the base.

Tracing 10.



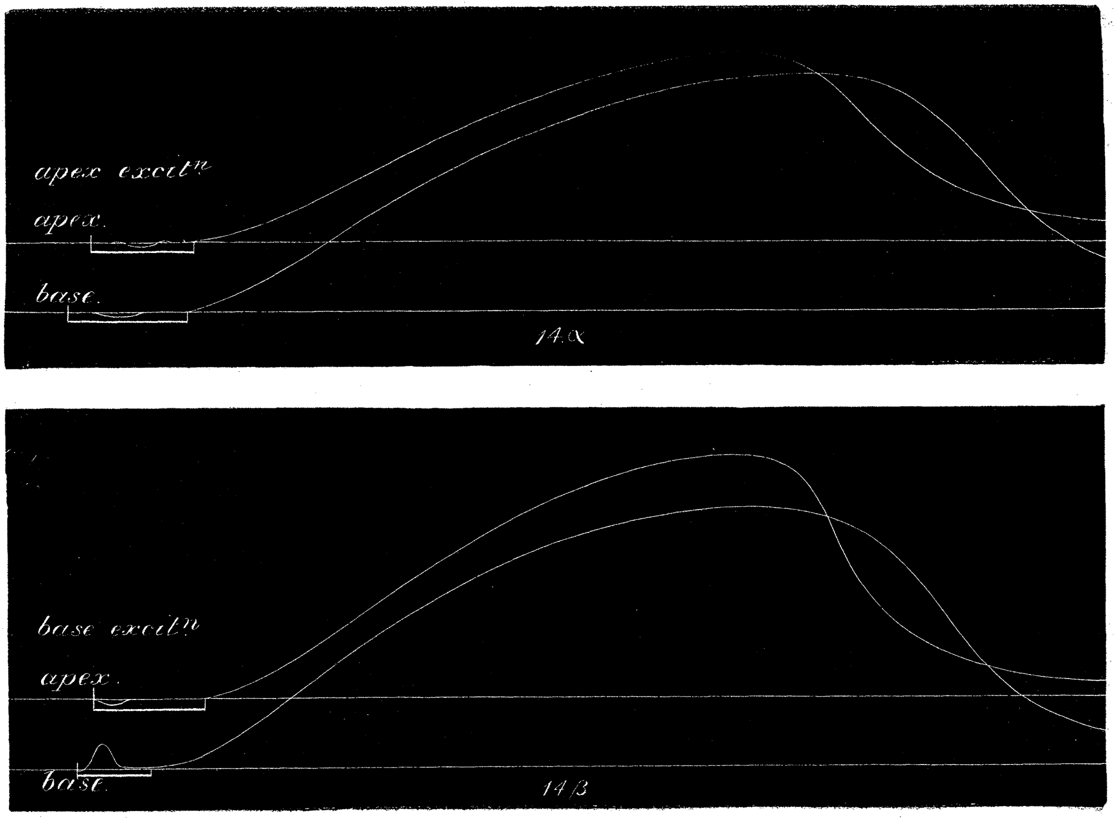
Wave of contraction in a strip cut obliquely from the right ventricle of a Dog, 10 minutes post mortem. Mechanical stimulation. The proximal lever is raised before the distal lever.

Tracing 11.



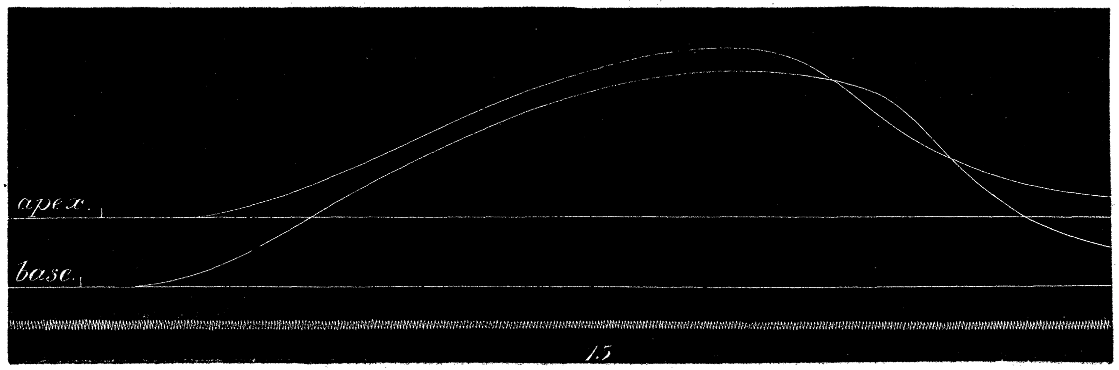
Wave of contraction in right auricle of Dog's heart. Upper line base, lower line apex. Excitation near base (β). Ditto. Excitation near apex (α).

Tracing 14.



Wave of contraction in Frog's ventricle. Upper line apex, lower line base. Excitation near apex (α). Excitation near base (β).

Tracing 15.



Wave of contraction in spontaneous beat of Frog's heart taken by double cardiograph. The movement of the lever resting on the base precedes that of the lever resting on the apex. It is noteworthy that in all cases the contraction at the base outlasts that at the apex.

organ occurred as a wave starting from the point of excitation. The lever nearest to the excited point commenced to rise sooner than the more remote one, the difference being sometimes so considerable as to be plainly visible without record.

This, which is the fundamental fact, was clear and unmistakable in ventricle and in auricle, and we may anticipate upon a future section of our paper by adding that the galvanometric indication of the passage of a wave of negativity was equally clear and unmistakable in the form of a diphasic variation.

We thus learned that the wave known to occur in the excited beat of the "stanniused" Frog's heart occurs *under similar conditions* in the Mammalian heart, *i.e.*, in the excised quiescent organ. As may, however, be expected, the experimental shortcomings of the Mammalian heart are more frequent than in the case of the cold-blooded organ; complete and typically illustrative results are not obtained without fail, and variations are so great that a normal time-value of the wave cannot well be given.

But we have obtained records conclusively showing the existence of the wave in the ventricle and in the auricle, both in the entire organ and in strips of muscle cut therefrom. The experiments we have made in relation to this point are given in the accompanying *résumé* (Table F). Tracing 9 is an example of the wave of contraction in the left ventricle. Tracing 10 is an example of the wave of contraction in a strip of the right ventricle. Tracing 11 is an example of the wave in the right auricle of the Dog's heart. Table G contains the results of the few measurements we have made of this wave.

The results thus obtained on the Mammalian heart led us to apply the same method to the heart of the Frog; for this purpose we employed levers 5 mm. apart. As far as we knew, this had not yet been done: MARCHAND had attempted to obtain evidence concerning the rate of the contraction wave by alternately exciting the ventricle, near and far, from a part upon which a single lever had been adjusted; his attempt failed, as was to be expected, from the variability of the latent period. The double lever, by means of which the contractions of two parts near and far from a point of excitation are simultaneously recorded, avoided this source of error, and by it observations may be multiplied. Our measurements thus made give for the rapidity of the contraction wave in the Frog's ventricle, at between 8° and 12° C., a rapidity of between 30 mm. and 90 mm. per sec. These measurements are given at the end of Table F, and are illustrated by Tracing 14.

(*b.*) *The Spontaneous Beat.*—The evidence which we had obtained of the occurrence of contraction consequent upon excitation of the quiescent organ in the form of a wave starting from the excited point naturally led us to inquire whether or no a similar wave of contraction takes place in the normal spontaneous contraction. The testimony of the galvanometer is to the effect that such a wave does occur in the spontaneous beat of the excised Frog's heart; the rheotome is here inapplicable, but the double variation observed from the beating heart, when led off at base and apex, is such as to indicate the two phases: (1) negativity of base, (2) negativity of apex, from which the conclusion is drawn that contraction proceeds from base to apex. With regard to the rate of propagation of the excitatory state in the spontaneous beat we have no information.

The application of two levers, 5 mm. apart, to the ventricle of the spontaneously beating Frog's heart at once shows that the contraction is from base to apex, and the difference in time between the rise of the lever nearer to the base and that of the lever nearer to the apex can be measured with reasonable accuracy; this time-difference shows that the wave of spontaneous contraction occurring from base to apex has a rapidity of about 100 mm. per sec. at a temperature of 9° C. (Tracing 15). As regards the spontaneous beat of the Mammalian heart, a similar method led us to results markedly different from those we had obtained in the Frog's heart. We have examined the hearts of Cats, Rabbits, Dogs, and Sheep, and find that in the spontaneous beat of the excised organ the contraction of the apex generally appears to precede that of the base; in some cases we have been unable to detect any difference between base and apex: in only two cases have we seen the contraction of the base precede that of the apex. The accompanying Table, G, summarises our results upon this point, and we give examples of the records we have taken in Tracings 12 and 13. It may not be superfluous to add that we have exercised all possible caution and care, paying due regard to correspondence of levers, pressure of pens, and comparative size of contractions.

It appears from our Table, firstly, that the rapidity of the wave is in general much greater in the hearts of warm-blooded animals than in that of the Frog. Secondly, that in Mammals it is more rapid in large hearts than in small; the maximum rapidity which we have accurately observed was in a Sheep's heart, the record being taken 4 minutes after death by bleeding, the levers being 8 cm. apart; the time-difference was ·01 sec., giving a rapidity per sec. of 8 metres. Thirdly, the rapidity of the wave progessively decreases after excision of the heart.*

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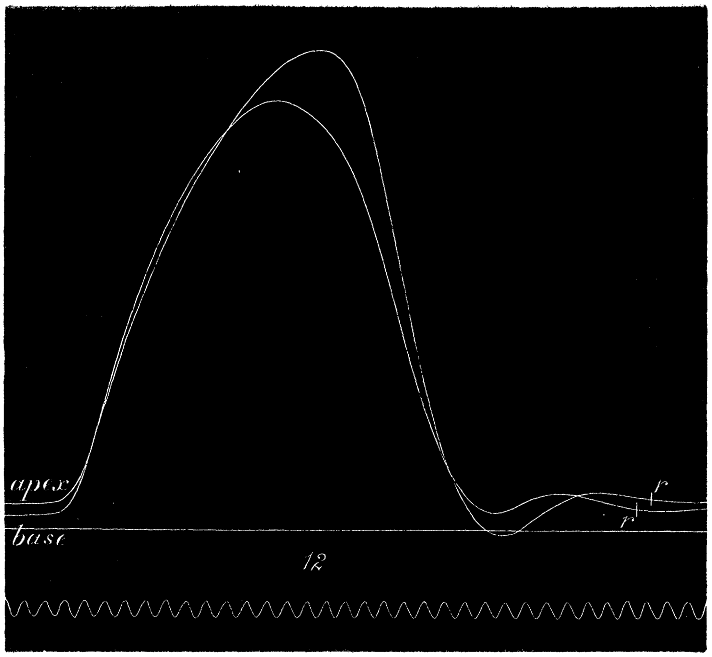
* When we made our observations we were not acquainted with those of F. KLUG (DU BOIS-REYMOND'S 'Archiv für Physiologie,' 1881, p. 265, and 1883, p. 398). In his first paper he stated as the probable conclusion to be drawn from his observations that apex precedes base in the normal systole of the hearts of Frogs and of Rabbits. No records are given. His method of observation was, however, not free from objection, and in his second paper he states that the evidence derived from his previous observations is of no value. The observations were made upon hearts *in situ*, with intact circulation, so that the discharge of blood by the auricle caused a movement of the ventricle which prevented the determination, with a sufficiently rapidly travelling surface, of the commencing ventricular contractions.

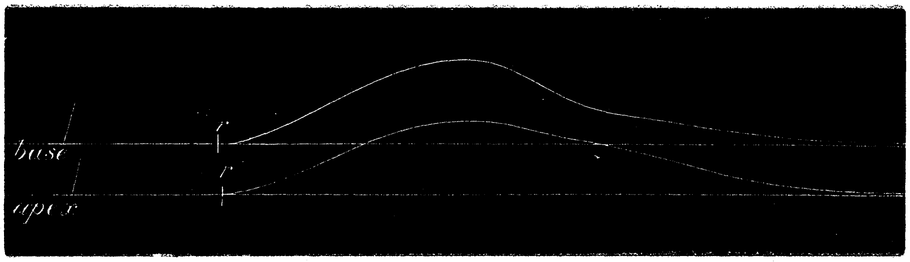
The opinions of HALLER, SENAC, ARNOLD, and of a British Association Committee in 1843 (quoted by KÜRSCHNER in WAGNER'S 'Handw. der Physiol.,' 1844, vol. 2, p. 35), were various; they were based on simple inspection of the heart's movements.

Tracing 12.



Wave (?) of contraction in ventricle of Cat's excised heart, beating spontaneously, 2 minutes post mortem. The vertical lines *r*, *r* indicate corresponding positions of levers, that resting on the apex being a little in advance of that resting on the base.

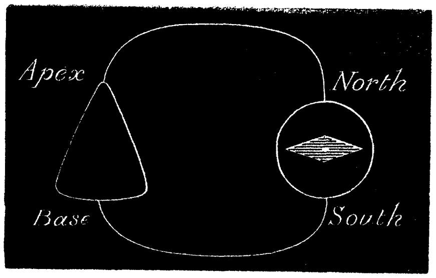
Tracing 13.



Spontaneous contraction of Cat's heart, recorded by double lever, 3 minutes post mortem. The movement of the lever resting on the apex precedes that of the lever resting on the base. The vertical lines *r*, *r* mark correspondence of levers.

§ VI. *Galvanometer Indications.*

It is necessary first to describe the plan of our apparatus, and the meaning of the abbreviations we shall have to use.



The heart, having been excised, was laid upon an insulating support, slightly hollowed to receive it, and the ventricle, right or left, led off by unpolarisible electrodes applied near base and near apex respectively. The disposition adopted was such that the apex was always in connection with the north screw of the galvanometer, the base being in connection with the south screw; we call these points respectively A and B. Current through the galvanometer from A to B is indicated by a deflection south of the magnet, current from B to A by a north deflection: it is thus apparent that a north deflection indicates that A becomes negative to B; that a south deflection indicates that B becomes negative to A. To denote the deflections north and south respectively, we use the letters N and S for large deflections, *n* and *s* for small deflections. We used a very delicate THOMPSON galvanometer ($R = 13{,}000$ ohms); the periodicity of its oscillations was such as not to allow us to make observations at intervals of less than half a minute.

This was in some cases a disadvantage; it was, however, soon apparent to us that the condition of the heart itself is generally such as to forbid the too frequent repetition of excitations.

In observations of the kind which we had to make it would have been useless to take exact measurements in number of degrees, and we contented ourselves with the occasional use of the qualifications "large" and "small" to express the relationship to each other of the two phases of the diphasic variation which usually came under our observation.

Any electrical inequality between the two led-off points was compensated in the usual manner by means of a SANDERSON'S potentiometer; we used the same instrument to take measurements of such differences of potential.

Our observations by the galvanometer are in the main confirmatory for the Mammalian heart of the fundamental facts established on the Frog's heart by the researches of ENGELMANN, BURDON SANDERSON, MARCHAND, and others, but with certain reservations and amid frequent irregularities, owing to the presumably more mobile nature of the Mammalian organ. We know already, from the observations of

Kölliker and Müller,* that the spontaneous systole of the Mammalian heart is accompanied (or rather preceded) by an electromotive change; to this we have to add that the electromotive change is frequently diphasic, and entirely similar to the diphasic variation of the spontaneously beating Frog's heart. The second chief fact relating to spontaneous action is that electromotive changes, such as ordinarily belong to visible contraction, frequently persist in the absence of such visible contractions, and continue long after these have entirely ceased, *i.e.*, invisible molecular changes outlast visible changes of form.

(*a*.) *Excited contractions.*—As regards excited beats, one fundamental result is that excitation applied near to one of two points, by which any two parts of the excised, but otherwise uninjured and quiescent, heart are led off to the galvanometer, gives rise to a diphasic variation the direction of which is such as to indicate (1) negativity of the proximal electrode; (2) negativity of the distal electrode. These are the most important points which we have been able to satisfactorily establish. To these may be added a fourth statement, viz., the effect of local injury long after the heart has become quiescent, *i.e.*, inexcitable, and is apparently dead, is to develop a local alteration of potential, the injured part becoming negative to all other parts.

The details of our experiments show many irregularities, some of which we can only partially understand, but which are doubtless attributable to irregularities and inequalities in the dying organ; these may have been due to differences of temperature or accidental injuries, or other spontaneously occurring inequalities of excitability at different points. It might be expected that an explanation of these should be found by the experimental establishment of such irregularities: we have sought for such an explanation and failed to find it; and we attribute this failure to the great susceptibility of the excised Mammalian heart to experimental interference. Our laboratory notes contain abundant examples of this extreme susceptibility to apparently trivial causes. To mention an example, we have frequently noticed that a slight touch with a blunt pointed instrument near one of our two electrodes was sufficient to develop *permanent* negativity at the part, indicative of a slight degree of injury. It consequently happened, as the most frequent exception to the classical effect, that instead of the diphasic variation, indicative of negativity at the first and second contacts respectively, we obtained only a simple variation. We think that the apparent local negativity developed under these conditions was in different cases attributable to either of two causes: (1) injury; (2) the excitatory state. The first cause is indicated by a single permanent deflection, the second by a single swing; and that this is an excitatory effect is indicated by the fact that the first variation consequent on excitation is often followed by a second or a third such variation, and that the series of single variations thus initiated may terminate with a typical diphasic variation. We have exceptionally noticed that local excitation might give rise to a single variation, indicating that the electrode furthest from the point of

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* 'Würzburg, Phys. Med. Gesell. Verhandl.,' vol. 6, 1856, p. 529.

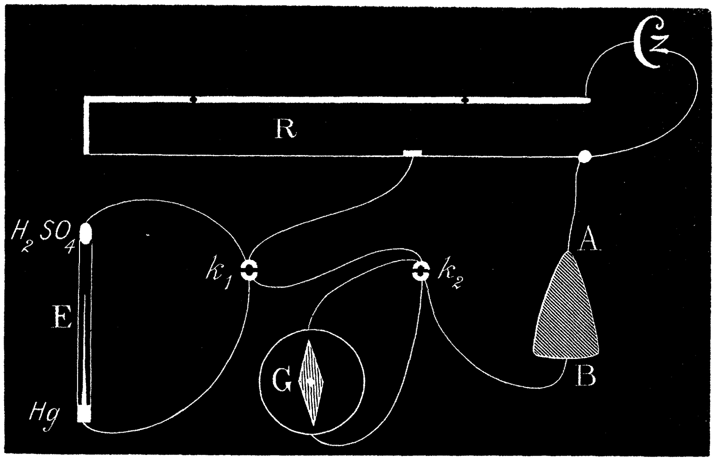
excitation becomes negative to the proximal electrode. This result is exceptional, and, although at first sight anomalous, can, we think, be reasonably explained. We think it is due to unequally excitable tissue at the two led-off points, and that the inequality consists in diminished excitability near the first lead-off in comparison with greater excitability near the second lead-off. Our notes contain several experiments, the analysis of which justifies this conclusion, and we may refer to Experiment No. 31 (Remarks on Galvanometer Experiments) as an instance in point.

We have never seen a diphasic variation such as to indicate negativity starting from the distal electrode. These results are in agreement with those previously described under § V. (*a*). We have usually applied our electrodes near the base and near the apex of the ventricle, both right and left; we have also applied them laterally to the right and left border of each ventricle. We have also examined the variations with an electrode on each ventricle, and with one electrode upon the auricle, the other on the ventricle. As regards all positions of the electrodes on the two ventricles, we have observed that the diphasic variation is the rule by excitation near either electrode. Our observations go to show that the ventricular portion of the excised Mammalian heart is an indifferent physiological conductor of the excited impulses in all directions. There are in the ventricular mass no indications of directions of greater or less resistance to the passage of the excitatory state. As regards the junction between auricle and ventricle, we have never seen any evidence of the passage of negativity in either direction. When an electrode is placed on either auricle and ventricle respectively of the same side, and excitation is applied to either cavity, the result has always been a single variation indicative of negativity of the electrode applied to it. We have never obtained a diphasic variation, nor have we ever observed a diphasic variation with electrodes on each auricle. These statements are based on experiments made after spontaneous contractions had ceased, viz., half to two hours after excision of the heart.

(*b*.) *Spontaneous contractions.*—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 which we obtained by the graphic method. The direction of the deflection when the heart was connected with the galvanometer by base and apex was very variable, and indicated no regular origin or mode of progression of the excitatory process. We can say no more on this point than that out of 62 experiments we observed N in 17 cases, S in 17, NS in 16, and SN in 12. Under these circumstances, and in the anticipation that this difficult question may be further pursued, and the conditions of variety in results determined either by ourselves or by others, we think it best to place on record a tabular summary of our results (Table I.), in the hope that the data therein consigned may prove to be of further use. We intend to pursue this question in a future investigation.

§ VII. *Electrometer Indications.*

An examination by the electrometer of the electromotive changes, as revealed by the galvanometer, is of obvious importance. The capillary electrometer of LIPPMANN gives indications which follow rapid changes of potential far more faithfully than do the indications of the galvanometer, whether "dead-beat" or freely oscillating. We arranged our connections according to the following diagram :—



The heart is led off at A and B. By the two short circuiting keys, K_1 and K_2, its current can be sent either through the electrometer E or through the galvanometer G. The current is compensated from the battery and rheochord R (its key is not represented in the diagram). The electrometer E is fixed on the stage of a microscope, the tube of which projects into a dark chamber, and the image of the field of the microscope, with the capillary column of mercury, is thrown upon a sheet of ground glass, on which its movements can be observed through an aperture in the dark chamber. The movements of the electrometer are recorded, when desired, by substituting a travelling sensitive surface for the ground glass, all light, with the exception of that passing through the capillary, being shut off from the plate by a screen in which a vertical slit is cut. Our photographs, except where otherwise stated, were taken with a ½-inch objective, and the image was formed 90 cm. behind it. We made use of sunlight for our photographs, reflected from the ordinary sub-stage mirror of the microscope.

The movements of the mercury in the capillary—advance and retreat—are in the same direction as the direction of current; hence, when A is negative to B, the movement of the mercury is northwards in the field of the microscope, and, the column being arranged to block the light focussed through the capillary on to the travelling sensitive surface, this movement appears on the photographic negative as a white projection into a darkened area; the reverse occurs if B becomes negative to A, and a black projection into a light area is recorded on the negative. Of course this is again reversed in the positive prints of such negatives.*

We did not, as in the case of the galvanometer, adopt only one mode of connection of the electrometer, but varied it so that sometimes A was in connection with H_2SO_4, sometimes with Hg; this we considered advisable, seeing that the movements of the capillary column do not take place with equal facility in both directions, but more easily towards than from the end of the tube. But, to preserve uniformity in the registration of our results, we give them as if the connection were throughout as in the diagram; negativity of A is then denoted by a north variation, negativity of B by a south variation (apparent variation in field of microscope).

(*a*.) *Of spontaneous contractions.*—Our first trials showed at once that the readings of the electrometer agree with those of the galvanometer, but that, as might be expected, rapid changes are read by the former instrument which escape observation by the latter. Thus, *e.g.*, in Experiment 42 the spontaneous variation galvanometrically was N, while electrometrically it was SN, and in Experiment 44 it was galvanometrically S, electrometrically NS. The superiority of the electrometer is not, however, fully made use of without having recourse to the photographic method, by which a permanent record of the variations is made. Whereas by the swinging magnet or swinging coil of a galvanometer magnitude and duration of electromotive changes, such as occur with action of the heart, are compounded and cannot be separated, their separate estimation by the electrometer is to some extent possible; how far this is so we do not know, doubtless the curve described by the mercury column does not give the absolute course as to duration and magnitude of a very rapid electromotive change; different electrometers, or the same electrometer at different times, may have a different amount of deforming effect. This is, however,

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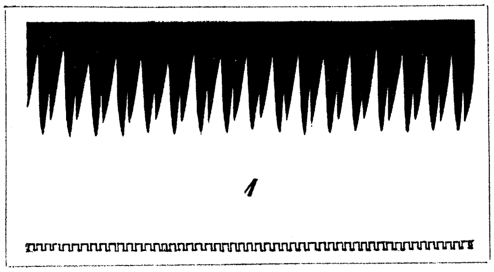
* The following key will be of service for the reading of our photographs :—

With A to H_2SO_4 and B to Hg (as in diagram).—*If* A *becomes negative to* B, the mercury moves towards the end of the capillary, *i.e.*, northwards in the field of the microscope; the photographic negative shows a light projection into a dark area, the positive shows a dark projection into a light area. *If* B *becomes negative to* A, all is reversed; the mercury moves southwards in the electrometer image, and the negative shows a dark projection into a light area, the positive shows a light projection into a dark area.

With A to Hg and B to H_2SO_4.—*If* A *becomes negative to* B, the photographic negative shows a dark projection into a light area. *If* B *becomes negative to* A, the photographic negative shows a light projection into a dark area. In all our photographs the upper border of the figure corresponds with the north of the microscope field; the time-tracing is recorded at this border. In the woodcuts, which are the reproduction of photographic positive prints, the lower border of the figure corresponds with the north of the microscopic field; the time-tracing is recorded at this border. The black portion of the woodcuts reproduces blocking of light by the mercury column, but in the woodcuts North movement of the mercury is represented by black projection towards the time-tracing, South movement by white projection in the opposite direction. All photographs read from left to right. The rates of movement of the sensitive surface are recorded on each plate. Their actual values are given with photos. 3 and 4.

out of our control: we can only submit to the criticism of our readers examples, the data of which we have obtained by its means. All these data have been furnished by the same capillary tube. The review of these examples shows (1) that our instrument was perfectly capable of giving the record of diphasic variations,* (2) that in some cases only a monophasic variation is revealed. Our opinion is that Fact 1 is sufficient argument for accepting Fact 2 as a true indication of a single phase, and not regarding it as the compound indication of two phases. Our experiments showed us that a double variation seen by the galvanometer is also seen and recorded to be double by the electrometer (Photos. 1 and 2); they showed further, as we expected, that a variation seen as simple with the galvanometer could be shown to be composed of two phases when the electrometer was put in circuit instead of the galvanometer. The fact, however, for which our previous experiments had not prepared us, was that

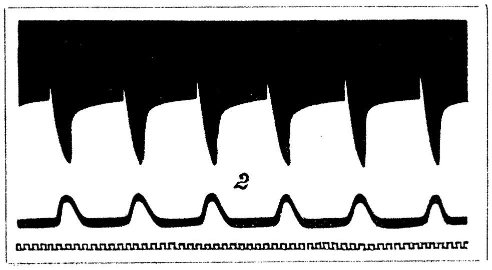
Photograph 1.



Frog's heart spontaneously beating, showing auricular variation, followed by ventricular.

NOTE.—All photographs read from left to right. The rates of movement of the photographic plate are indicated on the plates themselves. Two rates were usually employed, the slower of about 1 minute = ·44 second, the quicker of about 1 minute = ·08 second.

Photograph 2.



Shows double variation of spontaneously-beating Frog's ventricle.

The electrical phases are in opposite direction at beginning and at end of beat; first phase, base negative to apex; second phase, apex negative to base.

A simultaneous record is made of the movements of a lever resting on the ventricle. (Apex to H_2SO_4, variation SN.)

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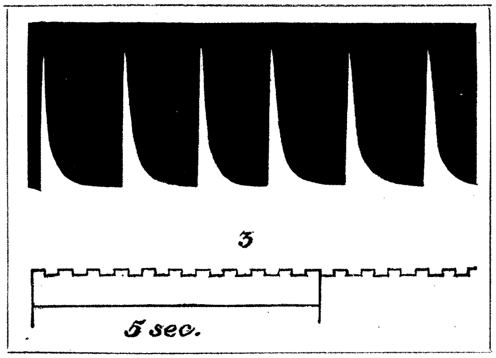
* We also obtained the diphasic variation of a single twitch of the Frog's gastrocnemius excited by an induction shock.

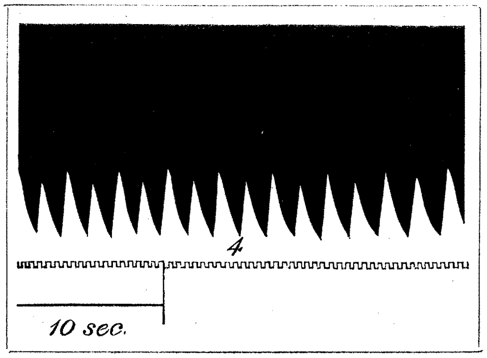
the electromotive change accompanying the beat of the ventricle is not always composed of two phases, it is sometimes expressed in its photograph as a single variation (Photos. 3 and 4). This then is the fact; our opinion concerning it is that, having regard to the rapidity with which the instrument may be shown to act to very brief changes in very rapid succession, the single variation observed under these circumstances is proof of a practically single and simultaneous change taking place throughout the ventricle, and disproof, or at least failure of proof, of the passage of a wave of excitation in the contractile substance. Our previous experiments by mechanical and by galvanometric methods had furnished the demonstration under certain conditions of the passage of a wave of excitation and of contraction at rates slow enough to be measurable, and the demonstration is confirmed by the electrometer (*diphasic variation*, Photo. 5). But our previous experiments by mechanical

Photograph 3.



Spontaneous variations of Rabbit's ventricle immediately after excision. These are in this case monophasic, and such as to indicate that the apex became negative to the base at each beat. (Apex to Hg. Variation N.)

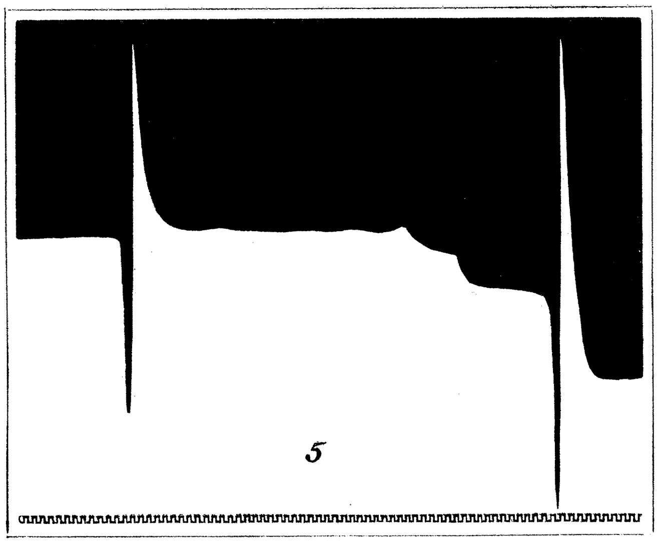
Photograph 4.



Spontaneous variations of Cat's ventricle. These are monophasic, and indicate that the base becomes negative to the apex. The electromotive variations show dicrotism which corresponded with the dicrotism of visible beats. Time post mortem 5 minutes. (Apex to H_2SO_4. Variation S.)

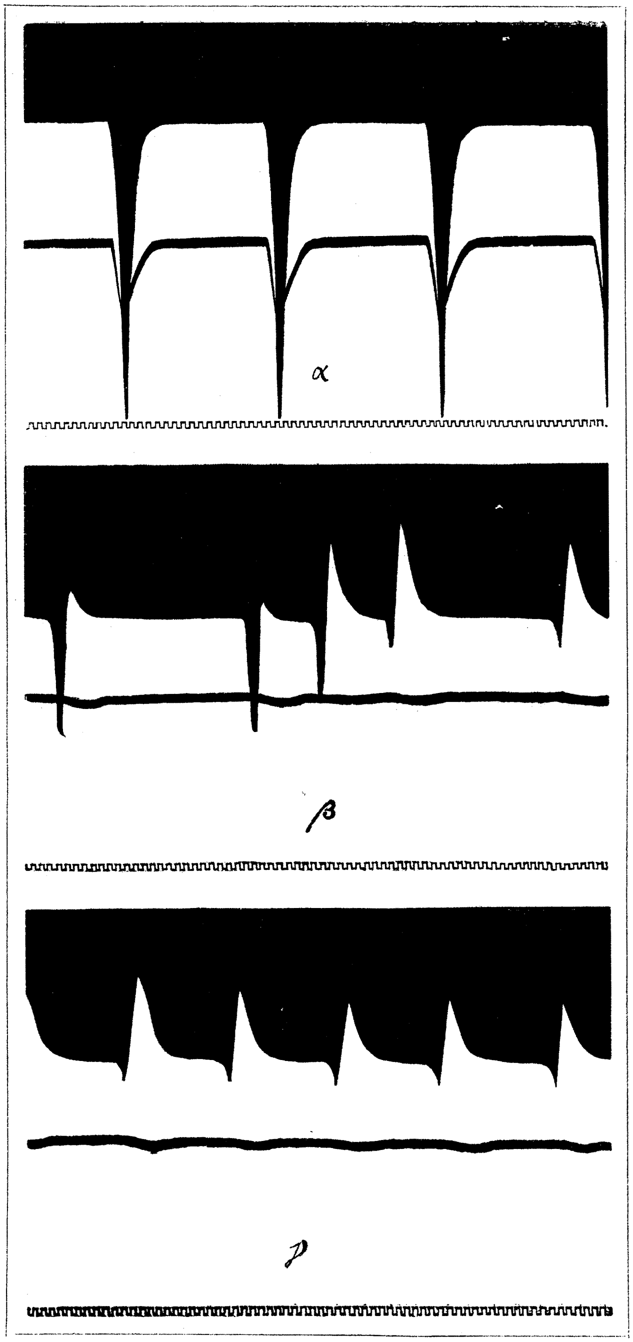
and by galvanometric methods had in many cases failed to furnish this demonstration, and the failure is repeated by the electrometer (*monophasic variation*). The conclusion we draw from this is that in such cases the ventricles contract in their several parts with a synchronism such that no evidence of wave as regards the excitatory process is obtainable by any method we have used. Whether the synchronism be absolute or not, we cannot say; it does not affect the conclusion we are about to draw, viz., that the excitatory change must, under such conditions, be practically simultaneous in all parts of the ventricle, and that such simultaneity postulates the existence of nervous channels of conduction. What are the conditions of the difference in the two cases? To this question we can only answer that we have, in general, observed *the monophasic variation during early moments after excision of the heart, the diphasic variation subsequently to these earlier moments;* more definite expressions we cannot use on account of the differences of individual hearts. We have further observed more than once the diphasic variation to supervene upon the monophasic in one and the same heart, as time advanced, without any alteration in the position of the electrodes (Photo. 6). As time advances, however, a diphasic may give place to a *late* monophasic variation, which is, to our mind, suggestive of a

Photograph 5.



Puppy. Two double variations in ventricle. The first is of a spontaneous contraction and indicates: first phase, apex negative; second phase, base negative; the second variation is caused by mechanical excitation of the apex, and also shows first phase apex negative, second phase base negative. The mechanical excitations to produce this contraction were three in number; the first two were ineffectual, *i.e.*, produced no visible contraction, they are, however, visible upon the tracing, the apex negativity having been permanently increased by each excitation. Apex negativity remained permanently increased after the diphasic variation had come to an end. (Apex to H_2SO_4. Variation NS.)

Photograph 6.



A series of three photographs of variations of a Dog's heart taken without moving the electrodes.

α. Five minutes after death. The variation is monophasic, indicating negativity of apex.

β. Forty-five minutes after death. The variation is becoming diphasic; first phase apex negative to base; second phase base negative to apex.

In some of the variations the first phase is much more marked than the second, in others the second phase is the more marked.

γ. Sixty minutes after death. The variation has become uniformly diphasic, first phase apex negative to base; second phase base negative to apex, and the second phase is more marked than the first (Apex H_2SO_4. Variation N and NS.)

different cause; whereas we regard the early monophasic variation as the expression of local predominance of a change taking place throughout the whole ventricle, we think that the late monophasic variation is in reality due to local activity at and near one of the two leading-off electrodes; this, indeed, is often demonstrable at this period by local excitation, which then causes contraction restricted to definite spots.

(*b.*) *Excited contraction.*—As regards the excitatory variation, our results with the electrometer were entirely similar to those which we have already described with the galvanometer. The accompanying Table summarises our electrometer observations. (Table K.)

§ VIII. *Conclusion.*

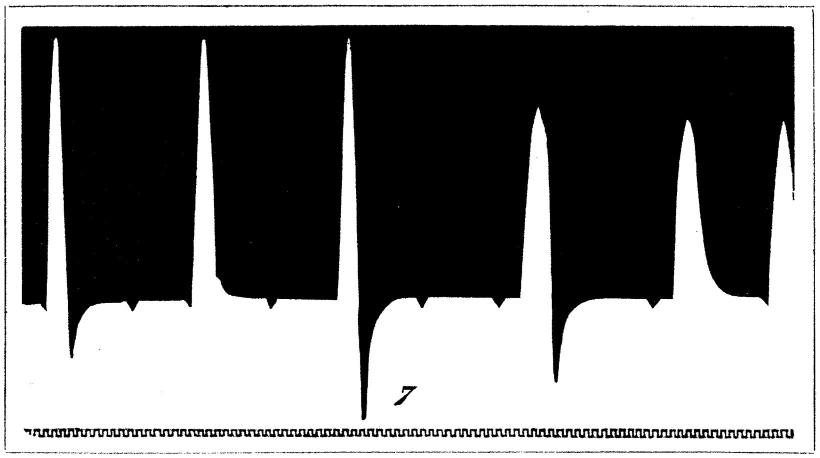
It would be superfluous to repeat, in summary, the facts relating to the mode of decline of the excised Mammalian heart which are described in the second, third, and fourth paragraphs of our paper. The extraordinary sluggishness of action, of which these showed us that the Mammalian heart is capable under the conditions of our experiments, tempted us to analyse as far as possible the mode and mechanism of cardiac contraction in Mammalia.

In this direction the chief result, which we regard as established, is that on the Mammalian heart, under conditions similar to those in which the Frog's heart has been examined in this respect, the passage of an excitatory wave is demonstrable as a diphasic variation with the galvanometer and the electrometer, and that the passage of a corresponding wave of contraction is demonstrable by mechanical methods.

As regards the Mammalian heart, neither of these facts have, so far as is known to us, been hitherto observed; as regards the Frog's heart, we have incidentally added to the classical phenomenon of the diphasic variation a demonstration of its mechanical aspect as a wave of muscular contraction. The similarity between the excised Mammalian heart and the excised Batrachian heart is thus far complete. But there are points of divergence. Analysis of the spontaneous beat both by mechanical and by electrical methods brings these into evidence. The diphasic variation of the spontaneously beating Frog's heart is uniformly indicative of negativity of base, followed by negativity of apex; to this we add the statement that muscular contraction of base precedes, and is followed by, muscular contraction of apex. All our experiments and measurements are thus confirmatory of the doctrine that the contraction takes place from base to apex, and that the wave of excitation and of contraction takes place by muscular channels. It is otherwise with regard to the Mammal heart. The variation is not always diphasic; immediately after excision it is more frequently monophasic and thereafter becomes diphasic. The movement of galvanometer and electrometer indicates for the monophasic variation negativity predominant either at apex or at base; for the diphasic variation (when it is present) negativity of apex, followed by negativity of base, or the reverse. There is no constant rule, in fact, and we saw no use in further multiplying observations, seeing that we had to do with the irregularities due to an excessive susceptibility to local injury; of such susceptibility we had abundant evidence; the delirium, which is so apt to supervene, is its extreme instance.

We see no escape from the conclusion that the Mammalian ventricle is an organ not only controlled by nerves, but also co-ordinated as to the action of its several parts through intra-muscular nervous channels. A monophasic variation can consist only with simultaneity of action throughout the organ, or with the successive action of its several parts so rapid as not to be revealed by either galvanometer or electrometer, or with action confined to one part of it, or with action predominant at one part; it is not consistent with the comparatively slow successive action of various parts of the ventricle by muscular transmission. Such simultaneity or approximate simultaneity can, we think, only be effected by nervous channels, and that conduction by nervous channels plays a part in the simultaneous and co-ordinated action constituting a beat is supported by other considerations, by measurements of the rate of conduction, for instance, such as we have given above, and still more strongly, we think, by the electrometer sequence repeatedly observed by us when the heart is dying and its parts becoming evidently asynchronous in their action, viz., negativity of auricle, followed by negativity of apex, followed by negativity of base. This can consist only with the existence of nervous channels along which excitatory impulses have passed from auricle to the apex. (Photo. 7.)

Photograph 7.



Variations accompanying spontaneous beats of Puppy's heart. The variations of the auricle are visible as small black teeth on the tracing, and it may further be seen that the variations of the ventricle are in two instances monophasic, indicating negativity of apex, and in three instances diphasic, with first phase = apex negative to base, second phase = base negative to apex. (Apex to Hg. Variation of auricle S; of ventricle N and NS.)

Usually it is not till several minutes have elapsed after excision that the diphasic variation is seen. It doubtless depends on the fact that with the slowness of action asynchronism of acting parts becomes manifest, but whether we have to do with a much retarded transmission from part to part along nerves or with muscular conduction is difficult to say with certainty; the entire similarity of the wave with a muscular wave is, however, sufficiently obvious. Finally, when transmission can no longer be effected, the heart responds to local excitation by a monophasic variation which may be temporary—indicative of local discharge of action,—or permanent—indicative of injury of tissue.

No portion of our inquiry gave us more trouble and doubt than the results of the mechanical exploration at base and apex of the spontaneously beating heart. Almost without exception we obtained apparently movement at apex antecedent to movement at base. All possible care was exercised, and we have given our results. We have been careful to say no more than that "the movement of the lever resting near the apex precedes that of the lever resting near the base."

Sometimes the contraction of the apex was visibly antecedent to contraction at the base—exceptionally, the precedence was with the base. In the case of the Frog's heart this was always the case.

A desire to obtain the variation of the absolutely normal and unexposed heart led us to the exploration of the human subject with the following result, viz., auricular followed by ventricular negativity anteceding respectively the auricular and ventricular events. We could obtain no evidence of a diphasic ventricular variation, and we have yet to exclude the possibility of the observed variation being caused by alteration of contact by the heart's impulse.*

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* I have since ascertained that the variation on Man is in reality due to an action current, and not to alteration of contact. The variation precedes the cardiac impulse; it may also be observed when the hand and foot are dipped into two vessels of salt solution connected with the electrometer. Under these circumstances the variation is still observed preceding the cardiac impulse.—A. W., July 5, 1887.

TABLE A.—Cat's Heart. November 30, 1885. Decapitated 3.2 P.M. Record starts 3.3 P.M.

Time P.M.	Number of beats.	Duration.
minutes.		seconds.
1 – 1¼	20·5	·2
1¼ – 1½	17·5	·3
1½ – 1¾	16	·37
1¾ – 2	16	·45
2¼ – 2½	13½	·67
2½ – 2¾	13½	·82
2¾ – 3	12	·9
3 – 3¼	10	1
3½ – 3¾	7½	1·2
3¾ – 4	8½	1
4 – 4¼	7	1·27
4¼ – 4½	7	1·27
Time lost in changing cylinders, ¾ minute.		
5½ – 5¾	5	1·5
5¾ – 6	4½	1·5
6 – 6¼	4½	1·65
6¼ – 6½	4	1·8
6¾ – 7	4	1·8
7 – 7¼	3	1·95
7¼ – 7½	3	2·1
7½ – 7¾	3½	2·1
8 – 8¼	2	2·1
8¼ – 8½	3	2·17
8½ – 8¾	2½	2·25
8¾ – 9	2½	2·25
9¼ – 9½	3	2·25
9½ – 9¾	2	2·4
9¾ – 10	2	2·4
10 – 10¼	2	2·4
Time of changing cylinder (new paper cut and smoked), 5¼ minutes.		

Time P.M.	Number of beats.	Duration.
minutes.		seconds.
15¾–16	0	
16 –16¼	2	2·55
16¼–16½	1	2·7
16½–16¾	2	2·85
17 –17¼	1	3
17¼–17½	1	3
17½–17¾	1	3
17¾–18	1	3
18¼–18½	1	2·85
18½–18¾	1	3·15
18¾–19	1	3
19 –19¼	2	3·15
19½–19¾	1	3·3
19¾–20	1	3·3
20 –20¼	5 } delirium	Beats ·6 to 1·2 second
20¼–20½	18 }	
20¾–21	5 (subsequent to delirium)	1·5, 1·8, and 2·2 seconds
21 –21¼	1	3·75
21¼–21½	1	3·75
21½–21¾	0	
22 –22¼	1	4·5
22¼–22½	0	
22½–22¾	0	
22¾–23	1	4·5
23¼–23½	2	3
23½–23¾	1	4·5
23¾–24	1	4·2
24 –24¼	0	
24½–24¾	2	4·5
24¾–25	0	
25 –25¼	0	
25¼–25½	0	
Excitation commenced after this.		

TABLE B.—Cat's Heart. December 2, 1885.

Minutes P.M.	Number of beats.	Minutes P.M.	Number of beats.
3¼–3½	16	8¼– 9¼	12
3½–3¾	15	9½–10½	9
3¾–4	14	10¾–11¾	7
4 –4¼	13	12 –13	6
4½–4¾	12	13¼–14¼	5
4¾–5	11	14½–15½	4
5 –5¼	9	15¾–16¾	4
5¼–5½	9	17 –18	3
5¾–6	7½	18¼–19¼	3
6 –6¼	7	19½–20½	2
6¼–6½	6	20¾–21¾	1
6½–6¾	6	22 –23	1
7 –7¼	5		
7¼–7½	4		
7½–7¾	4		
7¾–8	4		

TABLE C.—Table showing Beneficial Effect of Pause.

Number of the contraction.	Height.	Interval.	Number of the contraction.	Height.	Interval.
	millims.	seconds.		millims.	seconds.
1	25		14	13	
		9			18·75
2	14		15	17·5	
		10·8			19
3	19		16	14	
		13·5			4·8
4	16·5		17	9	
		12·3			30·3
5	17		18	20	
		16·5			140·21
6	17		19	16·5	
		17·5			84·15
7	17		20	17	
		16·2			21·60
8	16		21	14	
		29·25			7·5
9	22		22	9·5	
		30·9			60·9
10	18·5		23	17	
		70·5			238·2
11	21		24	15	
		100·65			11·25
12	19		25	9	
		6·45			
13	9·5				
		5·25			

TABLE D.—Rabbit. December 4, 1885. Auriculo-ventricular Block.

Minutes P.M.	
3– 6	All auricular contractions pass over.
6–12	Every other one passes over.
12–14	Every 3rd–4th passes over.
14–15	Every 4th–5th passes over.
15–16	Every 8th–10th passes over.
17–18	Every 8th–10th passes over.
18–19	Every 16th passes over.
20	Every 16th passes over.

After 22 minutes none get over.
The block increases very rapidly after about 14 minutes P.M.

TABLE E.—Table of Latencies. (Mammalian Hearts.)

Date.	Animal.	Time P.M.	Latent time.	Length of contraction.
		minutes.	seconds.	seconds.
1885.				
Nov. 27	Rabbit	11	·25	
Nov. 28	Rabbit	60·5	·45	
„	„	62	·45	
„	„	63·5	·49	
„	„	65	·45	
„	„	66·5	·6	
„	„	68	·45	
„	„	69·5	·6	6
Nov. 30	Cat	25·5	·3	
„	„	27	·45	4
„	„	30	·6	
Dec. 4	Cat	27·5	·27	4
„	„	32	·45	9
Dec. 4	Rabbit	Later than 45	·5, ·6, and ·87	5
Dec. 5	Kitten	11·5	2·	
Dec. 5	Rabbit	3	·15	
„	„	3·5	·225	
„	„	4	·37	
Dec. 8	Cat	30	·37	3·7
„	„	Later	·5	
„	„	„	·5	
„	„	„	·85	
„	„	„	·775	
„	„	„	1·075	
„	„	35	1·725	5
Dec. 9	Rabbit	8·5	·25	2·5
„	„	9·5	·25	
„	„	11	·275	
Dec. 12	Rabbit	10	·2	2·5
Dec. 14	Cat	26·5	·6 and ·9	4·5
„	„	31·5	2·	
1886.				
Jan. 1	Kitten	11	·175	2
„	„	Later	·2	
„	„	„	·225	
„	„	„	·25	
„	„	21	·35	3·75
„	„	22	·5	
Jan. 2	Cat	10	·1	·6
Jan. 7	Cat	18	·275	2·75
„	„	19	·325	3·12
„	„	23	·57	3·75
„	„	24	·625	5
Jan. 22	Cat	3	·150	1·62
„	„	4	·15	1·87
„	„	10	·225	2·25
„	„	11	·3	3
„	„	12	·3	1·5
„	„	12	·375	3
Feb. 5	Cat	12	·4	4
„	„	17	·5	5·5
Feb. 6	Dog	29	·44	
„	„	28	·40	1·4
Feb. 9	Sheep	19	·08	·8
March 1	Rabbit	10	·225	2·25
March 15	Dog	. . . . . .	·22	2

TABLE F.—Rate of Wave in Excited Beats of Ventricles.

Animal.	Time after death.	Rate of wave per second.	Remarks.
	minutes.	mms.	
Cat	..	40	*Vide* tracing 9.
Rabbit	5	364	
Kitten	11	∝	
"	21	1200	
Cat	..	1200	
Dog	2 min. 30 sec.	857	*Vide* tracing.
Cat	14	272	From base to apex.
"	12	218	From apex to base. (Latency = ·4 sec.)
"	18	54	From apex to base.
Dog	10	∝	
"	9	295	From apex to base.
"	10	369	From base to apex.
"	12	378	Strip of right ventricle.
"	13	284	" "
"	14	252	" "
"	24	142	" "
"	26	75	" "
"	10	367	" " *Vide* tracing 10.
"	12	312	" "
"	22	189	" "
Frog	..	70	
"	..	91	*Vide* tracing 14.
"	..	76	
"	..	30	
"	..	44	
"	..	88	

TABLE G.—Wave of Contraction in Right Auricle of Dog.

Time P.M.	Excitation.	Rate of wave per second.	Length of contraction.	
minutes.		mms.	seconds.	
33	B	45	2	
35	A	32	2·86	
36	B	26	2·3	
37·5	A	24	2·3	
60	A	14	4	Tracing 11α. Dog. Temp. 12° C.
5	B	25	..	Cat.
41	B	18	2	Tracing 11β. Dog.

TABLE H.—Rate of Wave in Spontaneous Beats. Mammal.

Animal.	Time P.M.	Rate of wave per second.	Lever rising first.	Remarks.
	minutes.	mms.		
Rabbit . . .	4	450	A	A > B.
,, . . .	4–13	∝	=	A slightly precedes B, where A > B.
,, . . .	1·5	1395	A	A > B.
Cat	3	1000	A	A = B. Tracing 13.
,, ...	3	2400	A	A > B.
Rabbit . . .	3	1000	A	A > B.
Cat	2, 4, 5, and 6	∝	=	A = B.
Kitten . . .	2, 3, 4, 5, and 6	∝	=	A = B.
Cat	5	1200	A	A > B.
,, ...	. .	∝	=	A = B.
,, ...	2	400	A	A > B.
,, ...	4	∝	=	A = B.
,, ...	4·5	∝	=	A > B.
Dog	1	1200	A	A < B.
Cat	2	2400	A	A > B. Tracing 12.
,, ...	9	444	A	A < B.
,, ...	4	300	A	A < B.
,, ...	4	600	A	A < B.
Dog	4	2400	A	
Sheep . . .	4	8000	A	A > B. Duration of contraction ·12 sec.
Dog	3–4	2320	A	A > B. Duration of contraction ·36 sec.
Guinea-pig .	2·5	900	A	A > B.
Cat	4·5	400	B	A > B.
Kitten . . .	5	300	B	A = B.
Cat	5	1875	A	A > B.

Rate of Wave in Spontaneous Beats. Frog.

		76	B	
		35	B	
		40	B	
		20	B	Tracing 15.
		12	B	
		90	B	
		100	B	
		100	B	
		100	B	
		79	B	
		133	B	

NOTE. $\left.\begin{array}{l}A > B\\ A = B\\ A < B\end{array}\right\}$ Signify that the curve of the apex lever was higher than, equal to, or less than that of the base lever.

Table I.—Abstract of Galvanometer Experiments.

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
1	1885 Oct. 31 .	Cat . .	2 hrs. 15′	N				
2	Nov. 30 .	Cat . .	1 hr. 30′	N	..	..	N ·002 D	90′
3	Dec. 2 .	Cat . .	1 hr. 15′	..	N	S	·0032 D	53′
4	Dec. 3 .	Cat . .	45′	..	*n*S	S	S	45′
5	Dec. 4 .	Rabbit .	1 hr. 15′	..	..	..	N ·0136 D	60′
6	Dec. 4 .	Cat.						
7	Dec. 4 .	Rabbit.						
8	Dec. 14 .	Cat . .	..	N	N	N	S	

Remarks to Galvanometer Experiments of Table I.

Experiment 1.—Our first observation was made upon the heart of a Cat, which had been killed by chloroform 2 hrs. 15 mins. previously; we were looking for the effects of injury upon its E.M.F., and observed spontaneous variations similar to the variations which accompany the normal rhythmic beat; the heart was motionless during these variations, which were such as to indicate negativity of apex.

Experiment 3.—This heart showed no spontaneous variations 53 mins. after death; beats had ceased to be visible 35 mins. post mortem. Excitation gave rise not merely to a single variation, but to a series of variations, indicating negativity of the excited part; we noticed this at the following periods post mortem: 1 hr. 18 mins., 1 hr. 53 mins., and 2 hrs. Thermal excitation of apex 2 hrs. 10 mins. post mortem gave S variation; at 2 hrs. 13 mins. post mortem excitation at base and apex gave S.

Experiment 4.—Electrode A was at apex of left ventricle; electrode B was at base of right ventricle.

Experiment 5.—The demarcation current was such as to indicate negativity at apex; it rapidly diminished from the moment when it was made.

1 hr. 10 mins. post mortem the demarcation current was . . .	·0056D **N**
1 hr. 30 mins. post mortem ,, ,, . . .	·0032D
Section at apex now caused it to rise to	·0192D
A few minutes later it stood at	·0184D
And a section at the base reduced it to	·0128D

The injury variation was thus considerably greater at apex than at base.

The excitatory variations were unsatisfactory, showing great want of uniformity, partly due, no doubt, to shifting of contacts.

Spontaneous variations were observed 1 hr. 20 mins. post mortem, there being at this time no trace of visible movement of the heart; they were subsequent to thermal excitation.

Experiment 6.—Cat's heart. 3 hrs. 21 mins. post mortem. Electrodes applied to base and apex were iso-electric; section of apex gave a N deflection of ·0036D. The entire heart was in rigor, and we could not satisfy ourselves that excitation had any effect.

Experiment 7.—Rabbit's heart. 5 hrs. post mortem. The left ventricle was in rigor, the right ventricle flaccid; electrodes on middle of right and left ventricle respectively showed no difference of potential.

Apex and base of right ventricle gave a current of ·0011D N. On left ventricle a current of ·00016D N.

Section of apex of left ventricle increased this to ·001D N.

Section of base converted it to ·024D S.

On the right ventricle section of apex increased current from ·0011D to ·016D N.

Experiment 8.—The demarcation current was such as to indicate negativity of base, viz., S; all variations spontaneous, excited from apex, or excited from base, gave a variation in the same sense, viz., N, which we could not attribute to shifting of contacts.

Section at base gave a small S deflection in comparison with a large N deflection caused by section at apex. It is allowable to conclude from the original direction of the demarcation current that the base was more injured than the apex at the commencement; this is confirmed by the effects of section at the two points, the effect being greater at the apex than at the base.

As regards the excitatory variations, the results are paradoxical, but intelligible on the supposition that the excitatory variation is manifested by the less injured part alone, viz., in this case the apex; and upon this supposition it is intelligible that the spontaneous beat should have given a similar variation.

TABLE I.—Abstract of Galvanometer Experiments—(continued).

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
9	1885 Dec. 14 .	Kitten .	0–1 hr. 30′	N	S	S	N before excision	
”	”	” .	(After	excision)	S	S	S ·0344 D	120′
10	Dec. 16 .	Kitten .	. .	. .	. .	. .	N before excision	
”	”	” .	. .	N	N	N	S ·0005	
”	”	” .	(Position of electrodes altered)	S & *n*S	NS	SN		
”	”	” .	Later	. .	N	S		
11	Dec. 17 .	Kitten .	. .	SN	NS	. .	Iso-electric	

Experiment 9.—Stimulus, with heart *in situ*, whether at base or at apex, gave S deflection; the demarcation current at the time showing apex to be most injured.

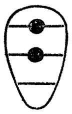
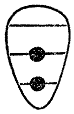
After excision the demarcation current was reversed and equal to ·0344 D S, and still excitation, whether at base or apex, gave S deflection.

The first part of the experiment showed very clearly the persistence of spontaneous electromotive variations after the complete cessation of visible beats; the effects of excitation are paradoxical, and we cannot explain them.

Experiment 10.—Kitten heart in body almost iso-electric; apex slightly negative to base.

Heart excised. Demarcation ·0005 D S.

Spontaneous variation; base and apex excitation alike gave N.

Position of electrodes was altered from  to 

Spontaneous beats are now S and occasionally *n*S.

Base excitation gave SN.

Apex excitation gave NS.

Later.—Base excitation gave S.

Apex excitation gave N, and injury near A gave permanent deflection N, injury near B permanent deflection S.

The first part of the experiment, *i.e.*, with the electrodes in the first position, was paradoxical, but intelligible upon a supposition similar to that made in case of Experiment 8, viz., that the excitatory variation is pre-potent at the less injured part, in this case nearer to the apex.

The effects observed in the second position of the electrodes do not contradict this supposition; they are obtained upon the portion of ventricle which is presumably least removed from the normal, and the results of excitation conform with the normal course of the excitatory variation which takes place in the stanniused Frog's heart; it was the first instance which we observed of a regular diphasic variation indicative of the passage of the excitatory state from B to A or from A to B. The direction of the spontaneous variation in this case is such as to indicate that the negativity is most manifest at B, but that it is first manifested at A. It is probable that we have to do here with a weak first and an intensified second phase of a diphasic variation proceeding from A to B.

Experiment 11.—Spontaneous variations are such as to indicate an excitatory change (negativity) commencing at B and terminating at A. Excitation at A gave the reverse effect, viz., a diphasic variation NS. We attempted to follow the variation by the rheotome in this case, but met with the difficulty that we were unable to obtain unfailing effects with short intervals between successive excitations, and that the prolongation of the experiment allowed of a too considerable decline of excitability for it to be possible to take observations with several positions of the rheotome. The necessity for allowing considerable intervals between test excitations was a noteworthy feature in all our experiments; the refractory period of excitation appears to be greatly prolonged in the excised Mammalian heart. For these reasons, and also because of the difficulty of knowing beforehand what intervals and what periods of closure to adopt for rheotome investigation, we did not pursue our experiments with this instrument.

TABLE I.—Abstract of Galvanometer Experiments—(continued).

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
12	1886 Jan. 2 .	Cat . .	30′	S & SN	NS & N	SN & S	N ?	
,,	,,	,, . .	Later	NS	NS	SN		
13	Jan. 4 .	Cat . .	30′	*n*S & S	NS	S		
14	Jan. 5 .	Cat . .	5′–30′	S	NS	S		
,,	,,	,, . .	1 hr.	,,	S	SN		
15	Jan. 6 .	Cat . .	. .	S	NS	*s*S		
16	Jan. 6 .	Cat . .	. .	S	NS			
17	Jan. 7 .	Cat . .	. .	. .	NS	S	N	

Experiment 12.—The observations within an hour after excision were very uniform as regards excitatory effects, viz., diphasic variations according to the point of excitation; in the second half of the first hour we noticed that, whereas a stronger stimulus was still capable of giving typical diphasic variations, viz., NS by excitation at A, SN by excitation at B, a weaker stimulus gave only a single variation, viz., N by excitation at A, S by excitation at B. Presumably, the excitability was not far removed from uniformity, and with this agrees the fact that the two contacts were practically iso-electric. The spontaneous beats indicated the passage of an excitatory change from B to A.

Later, in the second half-hour, the excitatory effects continued uniform, viz., NS by excitation at A, SN by excitation at B; the noteworthy point during this period was the appearance of spontaneous variations NS; these were not properly spontaneous, but consequent upon excitation, each stimulus at A giving not merely one variation NS, but a series of two, three, or more such variations at intervals.

Experiment 13.—This agrees with the supposition that the apex is more injured than the base: we omitted to take the demarcation current.

Experiment 14.—The above remark applies to this experiment during the first half-hour.

Experiment 15.—Our results are compatible with the supposition that A is warmer than B, and that the excitatory variation at B appears twice.

Our attempt to reproduce the effect by warming B or cooling A failed.

Experiment 16.—We tried on this heart the effects of thermal injury, bringing a heated point near the apex; it gave a variation S, viz., apex positive; thermal injury near the base gave an N variation, *i.e.*, base positive; these anomalous results we afterwards found to be due to thermoelectric currents.

Experiment 17.

Cat's heart.—Galvanometer observations begin 25′ post mortem. Apex to N screw of galvanometer.

I. Electrodes on *left ventricle*. Demarcation current N.

Apex excitation	NS	
Base ,,	S	
Apex ,,	NS	
Base ,,	S	followed by delirium.
Weak apex excitation	N	only.
Apex excitation	NS	
Right ventricle excitation		no effect.
Base excitation	SNS	
Base ,,	SNS	
Apex ,,	NS	
Base ,,	SNS	
Weak base excitation	S	
Strong base ,,	SNS	
Apex excitation	NS	
Both auricles excited		no effect.

II. Electrodes on *right ventricle*

Apex excitation	N	
Base ,,	SN	followed by delirium.
Apex ,,	NS	
Base ,,	SN	

TABLE I.—Abstract of Galvanometer Experiments—(continued).

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
	1886							
18	Jan. 15	Cat	..	S	S	S	N	
19	Jan. 15	Cat	..	SN	NS & N	S		
20	Jan. 13	Dog	2 hrs.	NS	N	S	N	120′
21	Jan. 22	Cat	5′–15′	SN	NS	SN	S	5′
22	Jan. 22	Cat	8′	SN	NS	SN		
23	Jan. 27	Dog	10′–15′	..	NS	S	N	10′
24	Jan. 28	Dog	2′	N	N	SN		
25	Jan. 30	Dog	..	N	N	SN		
26	Feb. 5	Cat	25′	..	N	S		
27	Feb. 9	Sheep	..	..	NS	SN	N ·012 D	26′
28	Feb. 12	Rabbit	25′	S	NS	S & SN		

Experiment 17 (continued).

Spontaneous beat . . . . NS
Base excitation . . . . SN
Base „ . . . . S
1 hour post mortem excitatory effects uncertain.
Injury at *base* . . . . S
Injury at *apex* . . . . N
Change caused by apex injury less than that caused by base injury.

Experiment 19.—We noted that 15 mins. post mortem the spontaneous variation was SN, at 34 mins. post mortem it was N.

Experiment 21.—We cooled this heart by placing it in a vessel surrounded by melting ice; in the course of cooling we observed the following effects of excitation at A: NS, NSN, N, and nothing. The variation NSN is compatible with A cooler than B; the variation N is due to the excitatory process not reaching to B.

Experiment 23.—These effects are compatible with greater injury at apex than at base. We tried the effects of excitation with the electrodes on the two borders of the left ventricle, and obtained by excitation near N the variation NS; near S the variation SN.

On stimulating midway between N and S, we obtained no appreciable variation.

On exciting nearer to N, we obtained NS; on exciting nearer to S, we obtained SN.

Similar effects were obtained on the right ventricle.

Spontaneous effects were now observed N, N and S being on apex and base.

Apex excitation gave N.

Base excitation gave SN.

Later (56 mins. post mortem) we observed spontaneous variation SN on the L.V.

59 mins. post mortem we observed spontaneous variations on the R.V., N; on the L.V., S.

Experiment 24.—Tested laterally on the L.V., we obtained from excitation near A a variation NS, from excitation near B a variation SN.

With A and B at apex and base of R.V., we obtained, 2 hours and 20 mins. post mortem, a series of spontaneous variations SN.

Experiment 25.—We took the spontaneous variation leading off from the right and left borders of the L.V., left border to S electrode, right border to N electrode, and observed it to be SN; this variation indicates negativity, beginning on the left side, ending on the right side.

2½ hours post mortem, spontaneous beats NS were still apparent, the R.V. being led off in the usual manner from A and B.

Experiment 26.—Extreme susceptibility to injury; the slightest touch gave permanent deflections; the current of injury was ·014D.

Experiment 27.—Tested laterally, A being on the right border and B on the left border of the L.V., we obtained, by excitation near A, NS; by excitation near B, SN; spontaneous variation, S.

On the R.V., laterally, we obtained similar effects by excitation near A and B respectively, viz., NS from excitation of A; SN from excitation of B. Spontaneous variation was NS, A being on the left border, B on the right.

TABLE I.—Abstract of Galvanometer Experiments—(continued).

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
	1886							
29	Feb. 12 .	Dog . .	6′	SN	NS	S		
			8′	S*n*S				
30	Feb. 13 .	Cat . .	38′–47′	. .	NS	SN		
31	Feb. 15 .	Dog . .	. .	SN	NS & N	SN		
32	Feb. 18 .	Dog . .	20′	N & NS	N 40′ P.M.	. .	S	
33	Feb. 22 .	Cat . .	. .	S LV	N	SN	S	
,,	,,	,, . .	. .	N RV	NS	SN		
34	Feb. 25 .	Guinea-pig	. .	. .	NS	SNS	N ·016 D	
35	Feb. 26 .	Cat . .	. .	S	. .	. .	N	
36	Feb. 27 .	Rabbit .	10′	. .	S	S	N ·0388 D	5′
,,	,,	,, .	20′	. .	NS	S		
,,	,,	,, .	30′	. .	N	S		

Experiment 29.—The chief peculiarity in this experiment was the varying character of the spontaneous variation; we noted it as SN, N, NN, NS SN, and NS.

Experiment 30.—With lateral position of electrodes, A being on the right border and B on the left of L.V., spontaneous beats gave SN.

On the right ventricle with same disposition, the same, SN.

Excitation near A gave NS.

Excitation near B gave SN.

Experiment 31.

Dog.—Decapitated 1.10 P.M. Apex to N screw of galvanometer.

I. Electrodes to base and apex of *left ventricle.*

Spontaneous beats . . . . SN 1.16
Base excitation . . . . S
Spontaneous beats . . . . SN 1.20
Apex excitation . . . . NS and N 1.25
Base ,, ... SN

II. *Electrodes laterally placed.* N electrode to right border; S to left border.

Excitation near S electrode . . . . SN 1.30
,, N ,, ... NS

III. Electrodes laterally placed on *right ventricle.* N electrode to left border; S electrode to right border.

Excitation near S electrode . . . . S 1.35

IV. Electrodes to base and apex of *right ventricle.* N electrode to base; S electrode to apex.

Apex excitation . . . . NS and N 1.47
Spontaneous beats . . . . N
,, ,, ... NS 1.53

V. Electrodes laterally placed on *left ventricle.* N electrode to right border; S electrode to left border.

Excitation near S electrode . . . . SN
,, N ,, ... NS followed by
Spontaneous. . . . . NS
Excitation near S electrode . . . . SN followed by
Spontaneous. . . . . SN

Later. 3 P.M.

Excitation near S electrode . . . . SSS followed by SN.
,, N ,, ... N followed by NS.
,, N ,, ... NNN followed by NS. Time, 3.15.

Experiment 32.—We took the excitatory variation in the excised right auricle. Excitation near A gave NS, excitation near B gave SN. We attempted to follow the effects of local heating of base and apex, but without result.

TABLE I.—Abstract of Galvanometer Experiments—(continued).

Experiment.	Date.	Animal.	P.M.	Spontaneous beats.	Apex excitation.	Base excitation.	Demarcation.	Time P.M. of registered demarcation.
	1886							
37	March 1	Rabbit	..	..	N	S	N ·0112	14′
38	March 2	Rat	..	...	N	S	N ·006	
39	March 8	Kitten	..	..	N	S	S	
40	March 8	Kitten	..	S	NS	SN		
41	March 8	Kitten	..	NS	NS	S & S*n*S	N	
42	March 15	Dog	..	N	NS	S	S	
43	March 20	Dog	..	..	NS	S	S	
44	March 26	Rabbit	..	S	NS	S		
45	March 27	Cat.						
46	June 6	Dog	..	S	NS	SN	N	

Experiment 44 furnished us with good data for the comparison of galvanometric with electrometric indications. The electromotive change accompanying the spontaneous beats was galvanometrically S, electrometrically NS; the latter we take to be the correct expression of the actual change: it proves beyond a doubt that the excitatory change (negativity) manifested itself at apex and then at base. When we shifted the electrode B to the auricle, which was giving two, three, or four beats to one ventricular beat, the electrometer showed a small South variation, corresponding to each beat of the auricle alone, followed by a larger double variation North-South with each ventricular beat. On moving electrode B to its original position at the base of the ventricle, the auricular beats ceased to affect the electrometer. The result obtained above we regard as most significant: it shows auricular negativity, followed by a biphasic ventricular negativity, at apex first, then at base; and it proves beyond doubt that the transmission of the excitatory state takes place, otherwise than in the Frog's heart, probably by nervous channels. If the excitatory state had passed from auricle to adjacent part of the ventricle, and thence to the apex by muscular continuity, its E.M. expression would necessarily have been a diphasic variation South-North.

Experiment 45.—The spontaneous variation immediately after excision was *South*, *i.e.*, base negative; the excited variation was North-South with excitation of apex, South-North with excitation of base.

TABLE K.—Abstract of Electrometer Observations.

No. of experiment.	Animal.	Spontaneous.	Apex excitation.	Base excitation.	Remarks.
42	Dog . .	SN	NS	S	
44	Rabbit .	NS	NS	SN	
45	Cat . .	S	NS	SN	
46	Kitten .	N and NS			
47	Rabbit .	N	. . . .	. . . .	*Vide* Photo. 3.
48	Rabbit .	N			
49	Cat . .	NS			
50	Puppy .	NS	NS	S	
,,	,, .	. . . .	. . . .	. . . .	Lateral excitation. Near N electrode NS. ,, S ,, SN.
51	Dog . .	S			
52	Puppy .	*s*N			
,,	,, .	*s*N			
53	Puppy .	NS and N	. . . .	. . . .	*Vide* Photo. 7.
54	Puppy .	SN			
55	Puppy .	N	N		
56	Rabbit .	. . . .	NS		
57	Puppy .	N	. . . .	. . . .	*Vide* Photo. 6a.
,,	,, .	NS	. . . .	. . . .	*Vide* Photos. 6β and 6γ.
58	Dog . .	. . . .	N	SN	
59	Puppy .	NS	NS	. . . .	*Vide* Photo. 5.
60	Cat . .	S	. . . .	. . . .	*Vide* Photo. 4.
,,	,, . .	NS	NS	S	
61	Cat . .	S and SN	N and N*s*N	SN	
62	Rabbit .	N and NS	N and NS	SN	
63	Cat . .	SN	NS	S	
64	Cat . .	N and NS alternately	N and N*s*N	SN	Lateral excitation S on R.V. Sx—SN. N on L.V. Nx—NS.