PROCEEDINGS

OF THE

PHYSIOLOGICAL SOCIETY,

Dec. 8th, 1900.

Some observations on the nerve cell connection of the efferent vagus fibres in the tortoise. By L. NOON.

It has been shown by various methods that the inhibitory fibres for the heart of the mammal and frog end in connection with nerve cells situated in the heart itself. And by the nicotin method Langley[1] has shown that the efferent fibres of the vagus, which supply the stomach and cardia in the rabbit, probably end in ganglia in close connection with the stomach wall. In both cases, then, the nerve cells are placed near the termination of the nerves.

In Crocodilia and Chelonia the vagus has on its course at some little distance from the heart and stomach a small compact ganglion which has been figured by Gaskell and Gadow[2] amongst others. In the crocodile and alligator Gaskell[3] came to the conclusion that this ganglion contained nerve cells which were on the course of efferent fibres to the œsophagus and stomach.

It seemed desirable that some further observations should be made on the reptilian ganglia. I have taken the tortoise (Testudo græca) as the most readily available animal.

In my experiments the animals were pithed. A one per cent. solution of nicotin, or a weaker solution, was injected through the right jugular vein of the animal. The local application of nicotin gave no good results. The dissection to expose the ganglion, and the branches of the vagus peripheral to it, so injures the circulation of blood in the nerves

[1] Schäfer. *Text-book of Physiology*, II. p. 664.
[2] *Journ. of Phys.* V. p. 362. [3] *Journ. of Phys.* VII. p. 20.

that the nerve fibres themselves were soon paralysed by even ·5 per cent. solutions of nicotin.

In the tortoise the vagus can be stimulated above and below the ganglion. I found that injection of a small dose of nicotin, even 5 mg. into the blood vessels, paralysed the vagus above and below the ganglion, but did not prevent the normal inhibitory effects from being produced by stimulating the walls of the great veins or the nerves peripherally on the heart. I conclude then that the inhibitory vagus fibres end in nerve cells in the heart itself, as they do in other animals that have been investigated.

In the observations of the stomach I began by taking tracings from a strip of the anterior stomach wall. A strip cut in the direction of the transverse muscle fibres, and left attached at both ends so as not to destroy its nerve and blood supply was lifted up on a glass hook. The hook was suspended to the writing lever. The rest of the stomach was held down in place by a glass rod fixed in a clamp. But some contractions of the stomach following on vagus stimulation do not spread over the whole viscus, and hence may not be recorded by such a strip. It was found quite easy to observe the stomach directly, and in this way every contraction could be noted. In the tortoise the contractions of the stomach are very largely peristaltic in nature, and a ring of contraction can often be seen passing down from the cardiac end on stimulation of the vagus. Sometimes the whole stomach passes into a condition of contraction for a minute or two. But tracings taken from a strip show that relaxation begins as soon as stimulation of the vagus is stopped.

The effect of nicotin on the motor vagus fibres to the stomach is not so easily observed as that on the cardiac inhibitors. In the first place the stomach muscle does not remain irritable so long as does the heart muscle. In the second place stimulation of the vagi does not always give any contraction of the stomach. Thus of six left vagi three gave good contractions constantly, one gave small contractions, one gave a single good contraction which was not repeated, and one gave no contraction. Stimulation of the right vagus has a less, and less constant effect on the stomach. This inconstancy of the result of stimulating the vagus makes it necessary to stimulate both before and after the injection of nicotin. Three such experiments were completed.

In the first experiment the left vagus gave small contractions of the stomach, the right vagus none. After injecting 50 mg. of nicotin no contraction was obtained by stimulating central or peripheral to the

ganglia on the vagi. In the second the left vagus gave one strong contraction which was not however repeated, the right vagus gave no contraction. After injecting 10 mg. of nicotin no contraction was obtained. In the third both vagi gave strong contractions. After injecting 5 mg. of nicotin no contraction was obtained. The nicotin was found, in fact, always completely to paralyse the vagus peripheral as well as central to the ganglion, though the secondary coil was pushed up to within 4 cm. or less of its position of maximum efficiency. Before injection a strong contraction was usually obtained with the secondary coil at 8 cm., and if with the coil at 6 cm. no contraction was obtained, then no contraction could be got at all by stimulation of the vagus. One cell was used in every case.

From these experiments I conclude that the motor vagus fibres to the stomach have their cell connection in the stomach wall. No other ganglia can be found between our ganglion and the stomach, on the course of the vagus. The vagus of the tortoise appears also not to supply fibres to the gut below the stomach, nor to the œsophagus. No contractions of these viscera were observed. The œsophagus was watched with especial care because in the crocodile Gaskell found that cells in the ganglion trunci vagi sent motor fibres to the œsophagus. The ganglion in the tortoise, then, does not quite correspond in its functions to the similarly placed ganglion in the crocodile and alligator. It seems to contain no motor relay cells, and will therefore probably be found to contain sensory cells.

The experiments of which I have given an account above were made in the Physiological Laboratory of the University of Cambridge.

I am indebted to Dr Langley for much advice and assistance.

Observations on the metabolism of creatinine. By J. J. R. MACLEOD.

(*Preliminary Communication.*)

During investigations on the metabolism of creatinine several facts have presented themselves which are of peculiar interest, and this is so especially with regard to the variations in the quantity of this substance excreted in the urine in various diseases.

Several experiments were performed on a normal subject in order to estimate the average amount of this body excreted in the urine, *firstly* during a flesh or creatine-containing diet, and *secondly* during a

creatine-free diet. During both these periods the diet contained approximately the same amount of N. and of Calories.

It was found that in the former case the average was 2·098 gr. in 24 hours, and in the latter 1·064 gr. During these two periods the total N. and the urea were estimated, the former by Kjeldahl's method, the latter by that of Mörner and Sjöqvist. It was found that during the first period an average of 13·7 gr. N. and 28·2 gr. urea were excreted, and during the second period 14·7 gr. N. and 26 gr. urea.

These results confirm the results of Voit and others, that urea does not arise from creatinine, for, had this been the case, a greater diminution of urea would have been expected during the second period.

The creatinine excreted by the urine may therefore be divided into an *endogenous*, and an *exogenous* quotient, the former arising from the metabolism in the animal's own tissues, the latter from the creatine and creatinine introduced in the food. Since the total amount of creatinine excreted, therefore, must vary with the diet in omnivorous animals, it is necessary, in studying the variations in the excretion of this body in disease, to place the patient on a creatine-free diet.

I have done this in several diseases, but more especially in muscular atrophy and in splenic enlargement. In the former disease I did not find any diminution.

In cases of splenic enlargement, however, a very remarkable result was obtained, namely a diminution in the excretion by about 50%.

The first case was one of *Hypoleucocytosis*, the leucocytes numbering on an average 2,000 per cubic mm., and the spleen being very much enlarged. The endogenous creatinine amounted in this case to ·332 gr. in 24 hours (five estimations).

The second case was one of splenic leucocythæmia where the endogenous creatinine amounted to ·530 gr. in 24 hours (five estimations).

The diminution, therefore, is probably not dependent on the number of leucocytes, but on the size of the spleen. In other words it would appear that the spleen exercises some influence on the metabolism of creatinine. In order to determine if this be so, I am at present engaged in studying the effect of splenectomy on the excretion of this body in animals.

The estimations were made by Salkowski's method, controls being carried out by a modification of Johnson's method.

Ether and chloroform extraction apparatus for liquids. By W. A. OSBORNE.

1. Ether extraction apparatus for liquids. The principle of this apparatus will be readily understood from the accompanying diagram, Fig. 1. It differs from the ordinary Soxhlet in having the siphon tube starting from a point *A* high up in the tube. *D* is a thistle funnel which collects the ether falling from the reflux condenser *C*. From its obliquely pointed extremity *B* the ether bubbles up through the aqueous solution *E*.

The advantage of having a thistle funnel of considerable capacity as compared with an ordinary Y funnel is this, that when the siphoning process has commenced the entire contents of the bulb *D* pass rapidly through the solution *E* stirring it up vigorously.

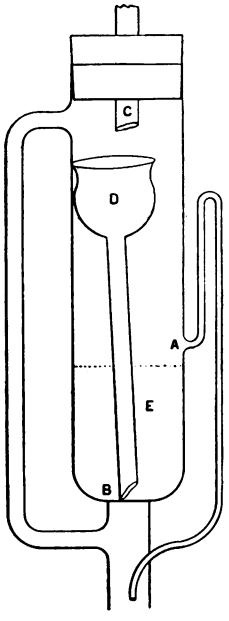


Fig. 1.

An apparatus of this construction, four centimetres in diameter will hold about 50 c.c. solution.

The advantages claimed for this apparatus are its extreme simplicity, its cheapness and its reliable working.

2. Chloroform extraction apparatus for liquids, Fig. 2. This apparatus is a little more complicated than the preceding one but its principle may be easily explained by the procedure adopted before and

during its use. First a few c.c.s of chloroform are poured in so as to fill the lower bend of the siphon tube *A*. Then the liquid to be extracted is added until its surface reaches the opening of the second tube at *D*. The chloroform as it drips from the condenser falls through the solution

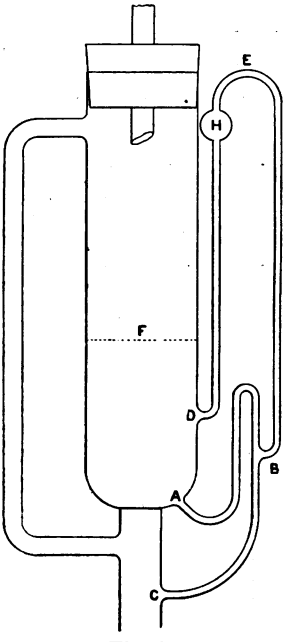


Fig. 2.

and collects at the bottom. When the level of the solution has reached a point represented by the dotted line *F* siphoning will commence through the tube *ABC*. This will stop however as soon as the opening *D* is uncovered for then air will be admitted through the upper into the lower tube.

The bulb *H* in the upper tube is necessary as at the end of the siphoning process there is a tendency for the column of chloroform in the lower tube to suck a little of the solution through *DEB*.

To prevent splashing of the solution by the falling chloroform a thin glass bulb with a diameter exceeding the radius of the extractor may be kept floating on the surface of the former.

Both apparatus may be had from Müller, High Holborn, the ether apparatus at the price of 4*s*., the chloroform at 5*s*.

Note to Fig. 2. This figure is drawn very diagramatically. In reality the siphon tube *ABC* should be close to the body of the extractor, *D* being placed a little to the side.

New form of pendulum contact clock[1]. By C. F. PALMER. Communicated by T. G. BRODIE.

This instrument was made for me by C. F. Palmer. It has proved to be so efficient both as a time-marking and as an exciting clock, that I have thought it desirable to draw the attention of other physiologists to its advantages. The whole credit of the design is due to the maker.

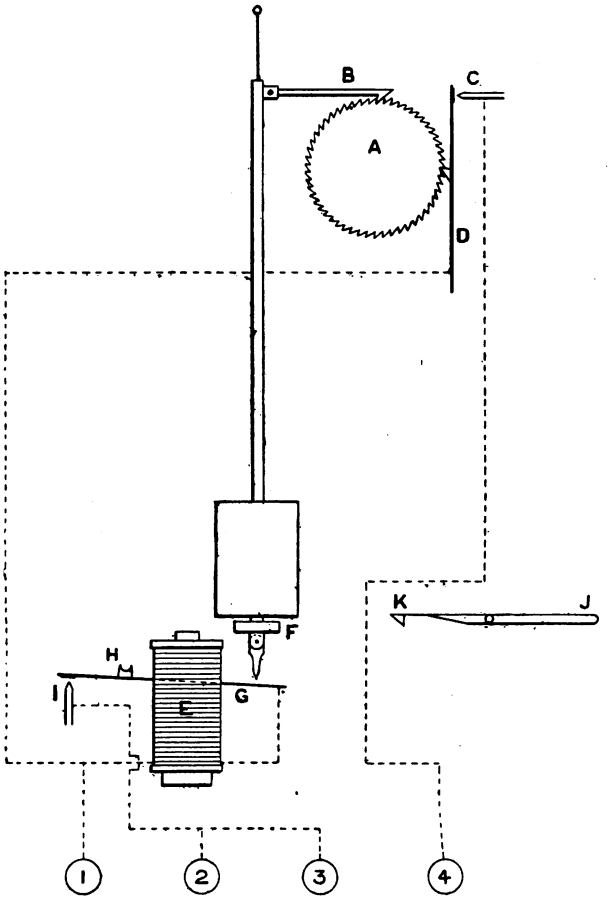
Description. This is an application to physiological purposes of an older form of electrical clock. It gives a contact either every second or, by a simple movement, every 2—10—30 or 60 seconds. The pendulum is kept in motion by means of an electro-magnet *E*, acting on the armature *F*, and will run, if necessary, without attention until the battery is exhausted, which would not happen for some weeks at least, as by an ingenious device the electro-magnet *E* only gives an impulse to the pendulum occasionally as required. This is accomplished by means of a little tongue of steel hanging from armature *F*. When the pendulum is swinging its normal beat, the little tongue passes over the notched piece *H*, carried on the spring *G*, but as soon as the length of stroke becomes less than normal, the little tongue catches in the notch *H*, which depresses spring *G* making contact at *I*, thus exciting the electro-magnet *E*. It is so arranged that the contact is broken again just before the armature reaches the centre of magnet, thus allowing pendulum to continue its beat.

A ratchet wheel *A* having 60 teeth is mounted so as to move 1 tooth at a time by means of the paul *B*. As this moves it depresses spring *D* which makes contact at *C*. This contact is very quick and therefore gives a good tracing with any suitable chronograph. There are other wheels having 30—6—2 and 1 teeth on the same axle as wheel *A* which can be made to gear with contact spring *D* at will, thus the different periods of contact are obtained.

There is also another simple but ingenious device for starting and stopping the pendulum. It is first hooked up on spring hook *K*, then by depressing lever *J* the pendulum is set in motion. To stop it, it is only necessary to raise lever *J* again when the pendulum will hook itself up ready to start next time. It is so arranged that the same battery that works the chronograph maintains the action of pendulum. The connections being from terminal 1 to electro-magnet *E*, thence to

[1] The instrument can be obtained from C. F. Palmer, 6, Upper Tulse Hill, Brixton Hill, S.W., at the price of £4. 0*s*. 0*d*.

spring G and from contact I to terminal 2. The chronograph circuit being from terminal 1 to spring D and from contact C to terminal 4. Terminals 2 and 3 being connected together. Battery (2 Leclanché cells) being connected to terminals 1 and 2. Chronograph to 3 and 4.

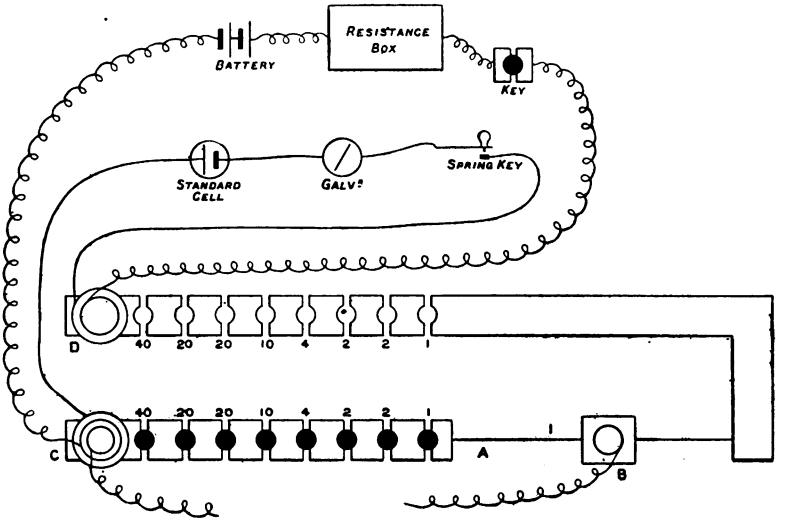


A potentiometer for physiological use. By W. M. BAYLISS.

This instrument was designed to combine the advantages of the resistance box pattern of Latimer Clark and the wire and slider pattern of Du Bois Reymond.

A platinum-iridium wire A (see fig.) 30 cm. long is stretched

between brass blocks, has a scale beside it divided into 100 parts and a slider *B*. Each end-block is connected with a series of eight resistances with plugs to short-circuit them; these resistances are of the relative values shown in the diagram, *e.g.* the one next the wire at both ends is of the same resistance as the wire itself, the next twice the resistance and so on, the resistance of the whole instrument, with plugs in on one side, being 100 times that of the wire alone.



A known potential difference is produced in Latimer Clark's manner between the end-terminals *C* and *D* as shown in the diagram; I find the most convenient standard cell to be Carhart's zinc chloride and mercury cell of 1 volt E.M.F.

The E.M.F. to be balanced is connected to *C* and the slider *B*. When all plugs are in on lower side, as in diagram, the E.M.F. between *C* and *B* is that indicated by reading of scale of wire alone and, since the whole resistance of potentiometer is 100 times that of wire, the E.M.F between extreme ends of the latter is $\frac{1}{100}$ volt and each scale-division has the value of $\frac{1}{10000}$ volt; if the E.M.F. to be balanced is greater than $\frac{1}{100}$ volt all that is necessary is to take out one or more of the plugs in the lower row and place in the corresponding holes in the upper series of resistances; if for instance we transfer plug 1 to upper row we do what is equivalent to adding to the portion of wire lead off a length of wire equal to the whole length of wire *A*,

i.e. $\frac{1}{100}$ volt, so that, if the original reading was say ·005 volt, by taking out plug 1 we make it equal to ·01 + ·005 = ·015 volt.

It will be seen that by changing over a resistance from one end of the wire to the other end no alteration is produced in the total resistance of the potentiometer, and therefore the difference of potential between its terminals remains as before.

The advantage of this pattern is that it combines considerable range with delicacy of adjustment, it is, in fact, equivalent to an E.M.F. of 1 volt between the ends of a wire 30 cm. × 100 = 30 metres long.

The instrument was made by Nalder Bros. and Co. of Westminster at a cost of £11. 0*s.* 0*d.*

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The frog's skin as an electrical organ. By A. D. WALLER.

At the last meeting (November) of the Society I showed the effects of homodrome and heterodrome (positive and negative) excitation of the Frog's Eyeball, and called attention to the close similarity between these effects and the effects of homodrome and heterodrome excitation of an electrical organ.

The purport of the present communication is to make comparison between similar effects of excitation of the Frog's skin and organ response; taken in conjunction with the previous comparison between eyeball and organ response, it is clear that the comparison of eyeball and of skin respectively with an electrical organ as their common standard of reference, implies their comparison *inter se*.

Skin currents have been a subject of investigation by many previous observers (du Bois-Reymond, 1849; Engelmann, 1871—2; Roeber, 1869; Hermann, 1878; Bach and Oehler, 1880; Bayliss and Bradford, 1886; Waymouth Reid, 1893; Biedermann, 1893) with various results and various interpretations. Deferring for a future occasion the consideration of the historical aspect of the questions raised, I shall for the present confine my remarks to a description of facts observed by one particular method, with a single layer of skin between electrodes.

The method is similar to that described and figured in a recent communication to the Royal Society on the blaze-currents of the Frog's Eyeball (*Proceedings R. S.*, Dec. 6th), the skin led off from internal and external surface taking the place of the eyeball led off from fundus

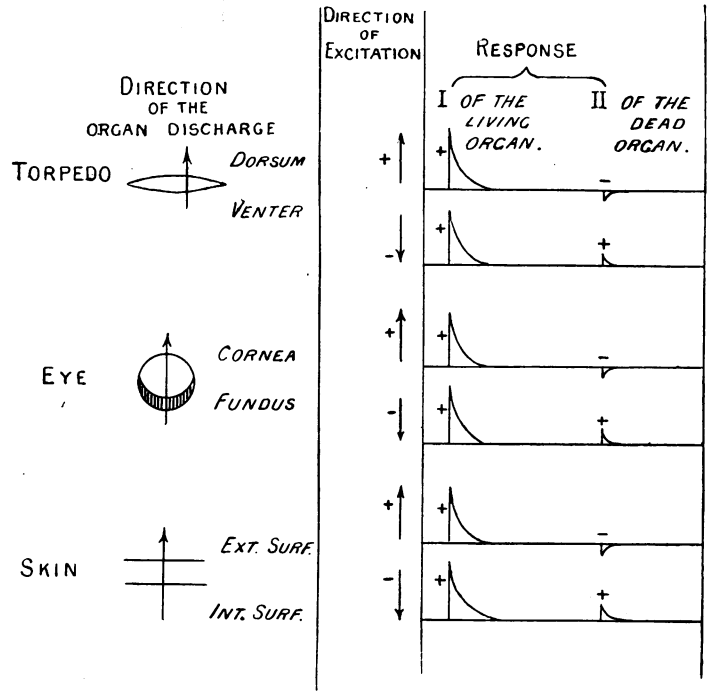
and cornea. "Positive" direction is thus "outgoing," and negative direction "ingoing."

Current of rest. With regard to direction, my observations agree with those of all previous observers to the effect that it is ingoing (negative) and that it increases during observation. Occasionally it may be outgoing (positive) at the very outset of observation, by reason of rough manipulation, and in such case the positive declines to and through zero to the increasing negative state. There is thus no essential difference between the so-called currents of rest in the two cases, although the one (skin) is generally observed as increasingly negative from the outset, while the other (eyeball) is generally observed as diminishingly positive, then increasingly negative. But in my view "current of rest" as applied to this falling state is a misnomer, and the declining current, whether decreasingly positive or increasingly negative, is in each case the subsiding current of excitation caused by manipulation of the organ. Having thus recognised the excitatory origin of these so-called currents of rest, we must, however, further recognise that in the state of rest to which both organs gradually approximate by gradual subsidence of manipulation blaze, there is negative current, from cornea to fundus in the case of the eyeball, from external to internal surface in the case of the skin.

Current of action. The principal facts according to my observations, are that any and every kind of stimulus of a fresh eyeball is followed by a positive electrical response (*i.e.* by a negative variation of the negative current of rest); and that any and every kind of stimulus of a fresh skin is followed by a positive electrical response (*i.e.* by a negative variation of the negative current of rest).

To which may be added a third statement, which I have fully substantiated in the case of the eyeball, but only partially studied in the case of the skin, to the effect that compression and rough manipulations of the two organs respectively 1° elicit positive current (manipulation blaze), 2° disfavour the normal (positive) action current produced by a single electrical stimulus of whatever direction, and 3° unmask a negative component, clearly and regularly in the case of the eyeball, less obviously in the case of the skin. In this condition, the eyeball and the skin give respectively positive interrupted by negative response (triphasic response + − +), or in the case of the eyeball pure negative response. Pure negative response in the case of the skin has not yet come under my observation, but I do not now wish to commit myself to any denial of the effect, and intend to carefully investigate this point further. It is, however, in any case a rare and exceptional effect.

The normal and regular electrical response of the skin to any sort of disturbance, mechanical, chemical or electrical, consists in a positive (outgoing) current. This outgoing direction is in my view that of the normal organ discharge analogous with the normal direction of organ discharge in the case of electrical organs, which is the type of such effects, and in that of the eyeball, as shown at a previous meeting of the Society. And the electromotive value of the effects in the three cases is of the same order, viz. 0·03 to 0·05 volt[1]. The analogy will be



Positive response to positive excitation is "homodrome"; positive response to negative excitation is "heterodrome". After death the slight effect when visible is in the direction opposed to that of excitation, *i.e.* an ordinary polarisation counter current.

most clearly appreciated by reference to the accompanying diagram, giving a comparative view of the phenomena in the three cases of the electrical organ of Torpedo (after du Bois-Reymond), the frog's eyeball and the frog's skin.

[1] On one occasion I observed a value of 0·09 volt for a homodrome (positive) skin response. But I think such a high value is exceptional. Ill-nourished frogs give very low values.

Some data regarding magnitudes of skin effects of electrical excitation.

	Excitation.		*Response.*
1.	Break induction current	100 +	+ 0·0050 volt
		100 −	+ 0·0010 ,,
		1000 +	+ 0·0550 ,,
		1000 −	+ 0·0850 ,,
		10,000 +	+ 0·0700 ,,
		10,000 −	+ 0·0900 ,,
2.	Break induction shock	1000 +	0·0330 ,,
		1000 −	0·0420 ,,
		5000 +	+ 0·0260 ,,
		5000 −	+ 0·0320 ,,
3.	Make induction shock	1000 +	+ 0·0045 ,,
		1000 −	+ 0·0015 ,,
	Break induction shock	1000 +	+ 0·0140 ,,
		1000 −	+ 0·0160 ,,
4.	Make	500	nil
		500	nil
	Break	500	+ 0·0085 ,,
		500	+ 0·0135 ,,
	Make	1000 +	+ 0·0150 ,,
		1000 −	+ 0·0065 ,,
	Break	1000 +	+ 0·0370 ,,
		1000 −	+ 0·0500 ,,
5.	Condenser discharge	8 volts 1 mF. + (= 640 ergs)	+ 0·0100 ,,
		,, ,, − ,,	+ 0·0100 ,,
		8 volts 0·1 mF. + (= 64 ergs)	+ 0·0015 ,,
		,, ,, − ,,	+ 0·0008 ,,

N.B. The + sign signifies outgoing direction, the − sign ingoing direction.