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EPILEPSY

AND

OTHER CONVULSIVE AFFECTIONS;

THEIR

PATHOLOGY AND TREATMENT.

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PATHOLOGY AND TREATMENT;

BY

CHARLES BLAND RADCLIFFE, M.D.,
PHYSICIAN TO THE WESTMINSTER HOSPITAL, ETC.

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PREFACE.

In the former edition of this work I introduced the remarks I had to make upon epilepsy and other convulsive disorders by certain considerations respecting the physiology of muscular motion. I did this because I believed that there could be no satisfactory interpretation of those disorders in which muscular contraction is in excess until a radical error in the theory of muscular motion had been corrected. I knew of no better course then; I know of no better course now—for a sound physiology must ever go before a sound pathology. At the same time, it is at the option of any-one, who may so choose, to read the practical portion of this work first, and then to turn back and see how far the conclusions to which he is conducted by arguments of a purely pathological character—

arguments which are complete in themselves—are borne out by the physiology of muscular motion.

In treating of epilepsy and other convulsive affections, I trust that the thought and experience of the last four years will have enabled me to supply some of the deficiencies and to correct some of the errors which existed in the first edition. I do not propose, however, to enter into every part of the subject. On the contrary, I pass by several topics of considerable interest in themselves, but only of secondary importance in the argument, because I do not wish to divert attention from the object I have in view, which is to point out the necessity for a fundamental change in the pathology and treatment of the disorders under consideration—a change which is in accordance with that which would seem to be demanded in the physiology of muscular motion.

C. B. R.

4, Henrietta Street,
Cavendish Square, W.

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PRELIMINARY CONSIDERATIONS

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THE PHYSIOLOGY

OF

MUSCULAR MOTION.

SEVEN years have now elapsed since I first endeavoured to show[1] *that muscle contracts, not because it is stimulated to contract by nervous influence, or electricity, or any other so-called stimulus of contraction, but because something has been withdrawn from the muscle which previously prevented the free action of common molecular attraction.* The argument used at that time was partly physiological and partly pathological; in its best part it was so defective that I can now look back upon it with no feeling of satisfaction; but such as it was, it seemed to show, not only that this view of muscular action was consistent with actual facts, but that it furnished a means of arriving at the physical explanation of three of the most important problems in physiology—of muscular contraction itself, of the rhythmic action of the heart, and of certain independent

[1] 'The Philosophy of Vital Motion,' 8vo, London, Churchill, January, 1851.

movements of the vessels which are of fundamental importance in carrying on the circulation.

Four years later I returned to the subject,[1] and giving more prominence to its pathological bearings, I endeavoured to show that epilepsy and all other disorders in which muscular contraction is in excess, are only intelligible when they are interpreted by such a theory of muscular motion.

All this time, and until a very recent period, I was not aware that the thoughts of any other person had been led in the same direction, but I now find that a similar idea had been entertained by Professor Matteucci, of Pisa, and by Professor Engel, of Zurich—at least, so far as concerns the action of nervous influence in muscular motion.

Professor Matteucci's speculations upon the operation of nervous influence in muscular action, were communicated to the *Académie des Sciences* of Paris, in 1847[2]. Speaking of the nervous *fluid*, he says, " ce fluide développé, principalement dans les muscles, s'y répand, et, doué d'une force répulsive entre ses parties, comme le fluide électrique, il tient les éléments de la fibre musculaire, dans un état de répulsion analogue à celui présenté par les corps éléctrises. Quand ce fluide nerveux cesse d'être libre

[1] 'Epilepsy, and other disorders of the Nervous System which are marked by Tremor, Convulsion, or Spasm; their Pathology and Treatment,' 8vo, London, Churchill, 1854.

[2] 'Comptes Rendus,' March 17th, 1847.

dans le muscle, les éléments de la fibre musculaire s'attirent entre eux, comme on le voit arriver dans la roideur cadavérique. . . . Suivant la quantité de ce fluide qui cesse d'être libre dans le muscle, la contraction est plus ou moins forte." Professor Matteucci appears to have framed this hypothesis partly in consequence of certain considerations which seemed to show that the phenomenon of "induced contraction" was owing to the *discharge* of electricity in the muscle in which the "inducing contraction" was manifested—an idea originating with M. Becquerel—and, partly, in consequence of the analogy which he himself had found to exist between the law of contraction in muscle, and the law of the discharge in electrical fishes; but he does not appear to have attached much weight to the idea even as an hypothesis. Indeed, his own comment at the time is,—"j'ai presque honte d'avoir eu la hardiesse de communiquer à l'Académie des idées si vagues, et apparemment si peu fondées, et contre lesquelles on pourrait faire bien des objections, mais je pense que, parmi les théories physiques les mieux fondées aujourd'hui, il en existe qui ont débuté de cette manière, et il est certain que des hypothèses, aussi peu fondées que celles-ci, ont quelquefois pu produire ensuite des découvertes remarquables."

The views of Professor Engel upon the operation of nervous influence in muscular action are stated in these words. "So hat der Nerve die Aufgabe, nicht die Zusammenziehungen des Muskels zu veranlassen, sondern den Zusammenziehungen bis auf einen geringen Grad entgegenzuwirken. Im lebenden Organismus, in welchem Ruhe etwas Unmögliches ist, ist auch ein ruhender Muskel eben so wohl wie ein ruhender Nerv undenkbar, der Muskel in seinem beständigen Streben, sich zusammenzuziehen, wird vom Nerven daran verhindert, im Nerven macht sich das fortwährende Streben kund, die Zusammenziehung des Muskels auf ein gerechtes Mass zurückzuführen; das Ergebniss dieser zwei einander entgegengesetzen Eigenschaften des Nervens und des Muskels ist das, was man gemeinhin Zustand des Ruhe, Zustand des Gleichgewichtes, oder an Muskeln auch Tonicität nennt. Das Verlassen dieses Gleichgewichts ist die Bewegung einerseits, die Lähmung andererseits. Die Bewegung wird aber erzeugt, indem entweder der Einfluss des Nervens auf den Muskel herabgesetz wird, oder indem die Contractionskraft des Muskels unmittelbar gesteigert wird. Lähmung des Muskels findet sich gleichfalls entweder durch unmittelbare Vernichtung der Contractionskraft des Muskels oder durch eine übermässig gesteigerte Ein-

[1] "Ueber Muskelreizbarkeit." 'Zeitschrift der Kais, Kön. Gesellsch. des Aertze zu Wien,' 1849.

wirkung des motorischen Nervens auf den Muskel. Sollen daher abwechselnde Muskelcontractionen zu Stande kommen, so ist die Gegenwart des lebendigen Nervens im Muskel unerlässlich, und auch bei unmittelbaren Muskelreizen können abwechselnde Zusammenziehungen nur erfolgen, so lange noch die Nerven lebensfähig sind; hört letzeres auf, so zieht sich der Muskel ohne Hinderniss zusammen. Diesen Zustand nennen wir die Todtenstarre." The reasons upon which these views are based would appear to be three in number. The first is to be found in certain facts, many of them very remarkable, which seem to show that the muscles of frogs become more apt to contract under mechanical irritation when they are abstracted from the main sources of nervous influence—as by removing the great nervous centres altogether, or by allowing time for the activity of these centres to become exhausted. The second depends upon the fact that the permanent contraction of *rigor mortis* is the state which supervenes when all signs of nervous influence are completely extinguished. The third is to be found in the fact that cramps and other forms of excessive muscular contraction are often seen to happen spontaneously in paralysed parts.

Later still—later than the time when I published my own views on the subject of muscular action,—I also find that Professor Stannius, of Rostock, reflecting upon the way in which *rigor mortis* is seen to be relaxed by blood,[1] has been led to a similar conclusion respecting the action of nervous influence upon muscle. Reflecting upon this fact, this physiologist considers—"dass es eine wesentliche Aufgabe der sogenannten motorischen oder Muskelnerven sei, die natürliche Elasticitäts grosse der Muskelfasern herabzusetzen und ihre Elasticität vollkommener zu machen; dass anscheinende Ruhe des Muskels, zum Beispiele, während des Schlafes, das Stadium solchen regen, den Muskel zu seinen Aufgaben weider befähigenden Nerveneinflusses anzeige; dass active Muskelzusammenziehung einen geregelten und begrenzten momentanen Nachlass des Nerveneinflusses auf den Muskel bezeichne; dass endlich die Nachweisung einer Muskelreizbarkeit, in der üblichen Auffassungsweise, ein durchaus vergebliches Bemühen sei." And afterwards he adds, "Ich muss es mir vorbehalten, später den Beweis zu führen, das diesse Anschauungsweise, so paradox sie immer auf den ersten Anblick sich anlassen mag, mit unserem thatsächlichen Wissen über Nerven und Muskelthätigkeit keineswegs im Widerspruch steht."

— I do not stand alone, then, in thinking that a great change is demanded in the theory of mus-

[1] 'Untersuchungen über Leistungsfähigkeit der Muskeln und Todtenstarre; Vierordt's Archiv für Physiol. Heilkunde,' Stuttgart, 1852.

cular action—a change which amounts to no less than a complete revolution—and hence I am encouraged to take up the subject anew, and point out the numerous particulars in which subsequent inquiry has shown that the old argument requires to be modified and expanded before it can hope to demand the attention of physiologists and pathologists.

In the present instance, I propose to confine myself, simply and exclusively, to the *Physiology of Muscular Motion*, and, leaving the pathology for the body of this work, I shall endeavour to establish the three following propositions:

1. That muscular contraction is *not* produced by the stimulation[1] of any property of contractility belonging to muscle.

2. That muscular elongation[2] is produced by the simple physical action of certain agents, electricity and others, and that muscular contraction is the simple physical consequence of the cessation of this action.

[1] The recognised opinion is that muscle is endowed with a vital property of contractility, and that muscular contraction is brought about when this property is called into action by certain agents, such as electricity or nervous influence. It is supposed, indeed, that this vital property of contractility is roused or excited or *stimulated* into a state of action when the muscle contracts, and hence the agents which thus rouse or excite or stimulate are called *stimuli*.

[2] The term *elongation* is used in preference to the term *relaxation* for reasons which will appear in the sequel.

3. That the special muscular movements which are concerned in carrying on the circulation—the rhythm of the heart, and the movements of the vessels which are independent of the heart—are susceptible of a physical explanation when they are interpreted upon this view of muscular action.

I. The First Proposition.

That muscular contraction is not produced by the stimulation of any property of contractility belonging to muscle.

1. *That muscular contraction is not produced by the stimulation of electricity.*

In order to understand the mode in which muscle is affected by *electricity*, it is necessary to know something of those electrical actions of which living muscle, in common with several other living structures, is the subject.

It is no new idea that muscle is the subject of electrical actions. Thus, Galvani explained the contractions which arise in a recently killed and prepared frog, when its nerves and muscles are connected by a conducting arc—" arco conduttore "—by supposing that *animal electricity* had been *discharged* by the arc. Soon, however, the discovery of Volta, that electricity was produced by the contact of dissimilar metals, gave rise to the idea that these contractions were due, not to the discharge of animal electricity, but to the passage of a weak current from the arc itself; and this opinion continued to gain ground, until Humboldt gave additional weight to the claims of animal electricity, by showing that similar contractions were produced when the muscles and nerves of the frog were connected by a piece of recent nerve. A little later, and animal electricity was once more thrown into the shade by the discovery of the voltaic battery. Then followed a long period of forgetfulness, only broken by Nobili's discovery of the "current of the frog," and not terminated until Professor Matteucci took up the subject and demonstrated most conclusively the existence of electrical currents in several tissues, and in muscles among the rest. It is to Dr. du Bois-Reymond,[1] however, that belongs the honour of having investigated most successfully the difficult subject of animal electricity, and there is no better course open to us than to follow in the track which he has opened out.

In his investigations upon the "muscular current" (to use the term which Professor Matteucci has applied to the current of animal electricity developed in living muscle), Dr. du Bois-Reymond made use of a galvanometer, the general arrangement of which is very similar to the sinus-galvanometer of Poggendorff. In the first instance, he used an instrument, the coil of which consisted of 3,280 feet of wire, and with this he also discovered the "nerve current," of which more will be said presently; but subsequently he used a far more

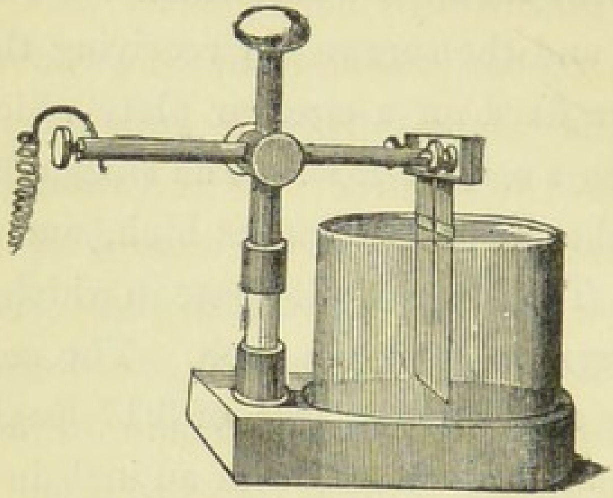
[1] 'Untersuchungen über thierische Electricität,' Berlin, 1848.

sensitive instrument, of which some particulars must be mentioned.

In this instrument, the frame upon which the wire is coiled, the arch from which the needles are suspended, and the screws for receiving the ends of the coil, are fixed on a circular plate which can be rotated upon a central axis. The sides of the frame are 4·6 inches long, 3·1 inches high, and 1·6 of an inch apart. The depth of the space in which the lower needle swings, is 0·1 of an inch. The copper wire forming the coil is 5584 yards, or 3·17 English miles in length, and about ·0055 of an inch in diameter. The number of coils is no less than 24,160. The needles are cylindrical with each end sharpened out into a long point. They are 1·5 of an inch in length, ·03 in diameter, and the weight of the two is 4 grains. They are connected by a thin piece of tortoise-shell, nearly 1·6 of an inch in length, and weighing not more than 0·9 of a grain. Away from the coil, the astatic system was adjusted so as to make a single vibration in 33 seconds.

Each end of the coil is connected with a peculiar electrode which requires to be described in order to make some of the following remarks intelligible.

This electrode consists of a horizontal arm, a pair of platinum plates, a glass trough containing a saturated solution of common salt, a cushion of blotting paper (of which the upper end is made to bend over the edge of the trough while the lower end is made to rest upon a ledge which projects inwardly from the wall of the



trough), a vertical pillar, and a wooden stand. The horizontal arm supports the platinum plates at one extremity, and at the other extremity it receives the wire of the galvanometer. The arm itself is moveable upon the pillar. The platinum plates are fixed to the arm by an appropriate clamp, and they hang so as to be immersed in the solution with which the trough is filled. Each plate is 2·5 inches in length, and 1 inch in breadth. Great care is taken in the preparation of these plates. It is absolutely necessary that they should be perfectly homogeneous (for any heterogeneity will give rise to currents when the circuit is closed), and in order to secure this they are cleansed successively with a mixture of alcohol and sulphuric ether, with nitro-muriatic acid, with distilled water, and last of all they are heated to incandescence for half a minute in the flame of a Berzelius' lamp. Great precautions are necessary, moreover, to preserve as well as to procure this homogeneity. A transitory current may be produced by the immersion of homogeneous plates, if the immersion of the plates of the two electrodes be not absolutely simultaneous; and to prevent this source of confusion, the plates of each electrode are kept continually immersed in the saline solution. A current might also arise from different conditions of different parts of the same plates if these plates were only partially immersed in the solution; and to obviate this difficulty, the parts above the solution are kept continually moistened by being wrapped in pieces of blotting paper of which the lower portion is immersed in the solution. Each electrode is furnished with two plates, partly to increase the surface by which any current may be able to enter the coil, and partly as an additional precaution against heterogeneity in the plates themselves,—for it is found that this cause of disturbance is less likely to operate when two plates are used than when only one plate is used. The cushion of blotting-paper is about 1·25 of an inch in breadth, and 2 inches in length, and when swelled out by absorbing the solution—about 0·5 of an inch in thickness. It is always soaked in the solution in which its lower portion is immersed; and its moist surface is therefore continuous with the surface of the solution. This cushion, indeed, is the real electrode, for it is through it that any current enters into or returns from the coil. The pillar which supports the horizontal arm and the parts attached to it, is insulated by a glass foot. Such is the electrode to which each end of the wire of the galvanometer is attached.

In completing the circuit, the cushions of the two electrodes may be placed in contact, or they may be connected by a third cushion, itself soaked in the same saturated solution of salt. When the instrument is not in use, the circuit is closed in this manner, and also (for additional security) by a piece of wire. Indeed, every precaution is taken to diminish the chances of any heterogeneity arising in the electrodes by making the circuit as complete as possible.

The galvanometer which I have used in repeating Dr. du Bois-Reymond's experiments, and for other experiments to which these have led me, was made[1] after

[1] This instrument, which is the first of its kind constructed in this country, was made by Mr. Becker, of Newman Street. Mr. Becker, moreover, entered with much spirit into the inquiries for which it was destined, and I derived very material assistance from his kind co-operation in some of the earlier experiments. Indeed, I shall always congratulate myself on the fact that I had recourse to his skilful assistance.

the pattern of the one which has been just described, or nearly so. The guage of the wire forming the coil in this instrument is number 38, or as nearly as possible that of the pattern coil, the weight of the wire entering into the coil 1 lb. 11 oz., the layers of the coil 154, the number of coilings 20,020. The coilings, therefore, are not quite so numerous as in the pattern coil—a difference owing in all probability to the silk winding around the wire being somewhat thicker ; but the instrument to all appearance is not a whit less sensitive on this account. The needles were copied with as much exactness as possible, and the only difference in the astatic system is in the fact that the connecting piece is made of aluminium instead of tortoise-shell. The whole system was a little lighter—4·5 grains, instead of 4·9 grains. The degree of astaticism was such as to make the needles arrange themselves at right angles to the magnetic meridian, or thereabouts.

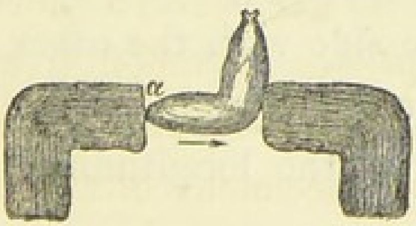
When in actual use, the galvanometer is placed upon a solid support where there will be as little vibration as possible, insulating pieces of glass are placed under the adjusting screws, and the coil is arranged so as to be across the magnetic meridian, or nearly due east and west. The electrodes may be on the same support as the coil or upon an adjoining table. In performing an experiment, the intermediate cushion, and the connecting wire, are removed, and the circuit is completed by placing the muscle upon the cushions, after having first covered the parts upon which it will actually rest with a small fragment of pig's bladder well moistened with white of egg. The bladder is used to prevent the direct action of the saline solution upon the muscle—an action which may of itself be sufficient to produce contraction.

Dr. du Bois-Reymond has examined the muscular current in man, rabbits, guinea-pigs, and mice; in pigeons and sparrows; in tortoises, lizards, adders, slow-worms, frogs and toads, tadpoles and salamanders; in tench; in freshwater crabs, and in earthworms;—and always with the same result. The animal which he ordinarily used was the water frog (*rana esculenta*), and this animal is undoubtedly the best suited to the purpose; but it is not necessary to be at the trouble to obtain these frogs from the Continent, for every experiment may be performed upon the common land frog (*rana temporaria*) of this country, if the galvanometer be as sensitive as the one above described, and if the frog be full grown. Indeed, I have not only repeated all Dr. du Bois-Reymond's fundamental experiments upon the common frog, but I have obtained more marked results than were obtainable from certain specimens of the *rana esculenta* which I had procured from the neighbourhood of Hamburg.

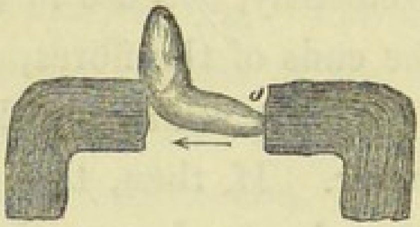
The primary phenomena of the muscular current, may be studied in any muscle, but most conveniently, perhaps, in the *adductor magnus* of the frog—most conveniently, because in this muscle it is easy to bring the ends of the fibres, or the sides of the fibres into separate relation with the cushions of the galvanometer. If, then, this muscle be laid upon the cushions, the results are these :—

If the two ends of the muscles are so laid; thus—

the needle of the instrument is deflected very slightly or not at all. In other words no sensible current is derived from the muscle. If the two sides of the muscle are placed in connexion with the cushions, the result is the same. But if the muscle be bent upon itself, and the cushions of the galvanometer so placed that one is in contact with the red flesh, and the other with the tendinous extremity; thus—



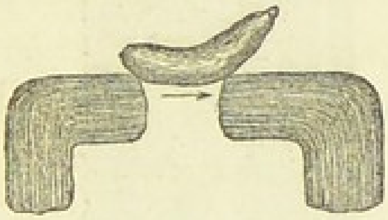
the needle of the galvanometer immediately gives evidence of a current in the direction of the arrow, that is, from the tendinous end. And, again, if the muscle be turned round so as to bring the other tendinous extremity to the cushion; thus—



there is still a current, and this current still sets from the tendinous end. And thus it appears that the muscular current is not fixed in its course, in as much as it is made to set in the contrary direction when the muscle is turned round upon itself.

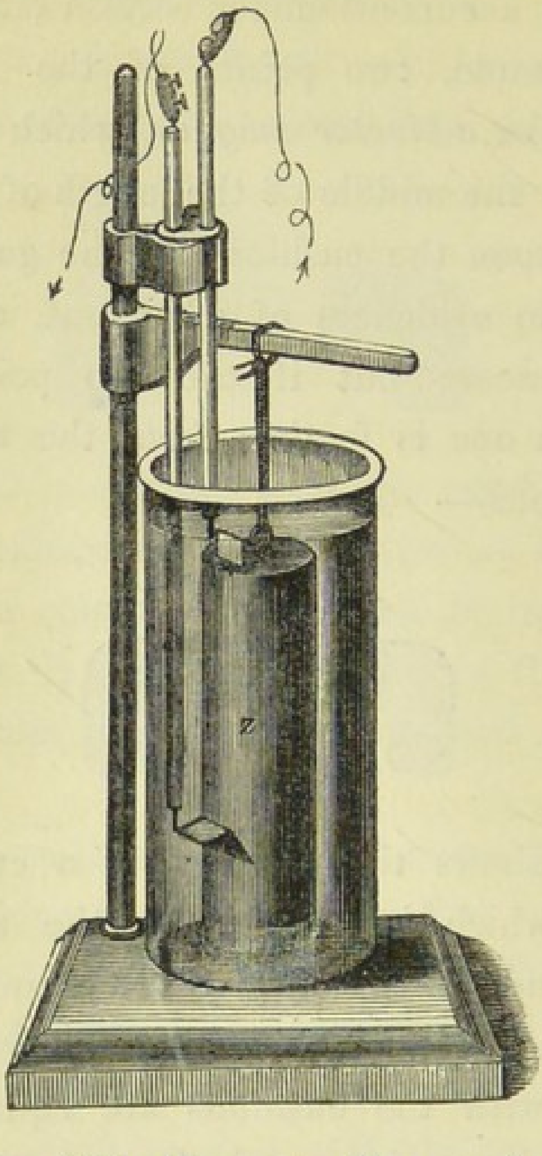
Similar results are also obtained by repeating these experiments after having first cut off the tendinous extremities of the muscle, for, on doing this, there is still no current, or a very feeble current, so long as the two ends only, or the two sides only, are in contact with the cushions; and there is a current, now somewhat stronger, which invariably sets from the end to the side, when the end of the muscle is placed in contact with one cushion, and the side with the other cushion.

On further examination, it is found that the different points of the longitudinal and transverse surfaces of a muscle, whether natural or artificial, possess electrical differences, and that these differences give rise to a current under certain circumstances. If, for example, two points of the longitudinal surface of the *adductor magnus*, which are equally distant from the middle of the length of the muscle, are placed upon the cushions of the galvanometer, there are no evidences of a current, or but very slight evidences; but if the two points are so chosen, that one is further from the middle than the other, thus—



the needle shows the presence of a current from the point which is nearest to the end of the muscle. And so also with the transverse surface. There is no current if the two points in connection with the cushions are equally distant from the median point; and there is a current if one point be nearer to this point than the other. But when most decided, the current which is derived from different points of the longitudinal or transverse surfaces is very weak when compared with the current which passes between the longitudinal and transverse surfaces.

In order to explain these phenomena more clearly, Dr. du Bois-Reymond has constructed an



apparatus in which they are all reproduced, and by which it is possible to attain to a very exact idea respecting them.

One of these instruments, and this the most satisfactory, consists of three parts—an electro-motive cylinder, a glass vessel containing water, and an appropriate stand. The electro-motive cylinder is copper, covered with a coating of amalgamated zinc, except at the two ends. It is 6·1 inches high and 2·1 inches in diameter. The vessel for containing this cylinder is 3·5 inches in diameter, and sufficiently high to allow the electro-motive element to be fully immersed in it. The stand consists of a wooden base, a vertical pillar, and two clamps—one for supporting the cylinder and the other for carrying the two wires by which the current is carried from the cylinder to the galvanometer. The stand is simply a square piece of wood upon which the glass vessel rests, and from which the pillar rises. The pillar is a metal rod upon which the two clamps may be made to slide up or down. One of the clamps, a well varnished piece of brass, projects horizontally, and to it the electro-motive cylinder is tied by a piece of string; the other clamp carries a cork which is perforated, so as to carry the conducting wires. The wires themselves are terminated at their lower extremities by small pieces of platinum (0·6 of an inch), and, in order to insulate them more effectually, they are enclosed for some distance in portions of glass tubing, of which the lower extremity is hermetically sealed upon them. These tubes and the contained wires may be pushed up and down, and moved about, so as to allow the platinum ends to be applied to different parts of the electro-motive cylinder.

On connecting the upper ends of the conducting wires with the galvanometer, the needle is acted upon or not acted upon according to the position of the platinum ends upon the electromotive cylinder. If one end be placed upon the zinc surface and the other upon the copper extremity, as in the figure, the needle immediately diverges to the extent of from 15° to 20° degrees. If both ends are placed upon the zinc surface at an equal distance from the two extremities of the cylinder, where consequently the electric tension arising from the reaction of the zinc and copper at these extremities is equal, there is no current; but if the ends be drawn upwards or pushed downwards, a current begins to be manifested as soon as their relations to the extremities of the cylinder begin to be unequal, — a current which may move the needle from 5° to 10°. There is no current, also, when the two ends are placed so as to be equidistant from the middle of the extremity of the cylinder, and there is a current when they are moved, so as to be unequally distant from this point. In a word, all the electrical phenomena of a muscle of which mention has yet been made, are fairly represented in this apparatus.

— But there are other phenomena of the muscular current, some of great interest, which have yet to be noticed.

In the *gastrocnemius* of a frog, for instance, the muscular current does not return to the sides of the fibres from both ends of the fibres, or from each end indifferently, as it did in the *adductor magnus*, but it returns from *one* end, and however the muscle be turned about on the cushions (except for a short time before its final extinction), the current always passes in an *upward* direction from the tendo Achillis to the head of the muscle. There is also an upward current in the *extensor cruris* of the thigh, and in several other muscles. In some muscles, on the contrary, there is a current in a contrary direction. It is so also with that general current which is derived from large groups of muscles; thus the general current of a limb has an *upward* direction in the leg of a frog, and a *downward* direction in the arm of a man, while in the pigeon the direction is *downwards* in the thigh, and *upwards* in the leg.

Now in order to explain these differences it is necessary to follow Dr. du Bois-Reymond a little further, and study the electrical condition of the tendon and the manner in which this condition may react upon the muscular current—a not very difficult task.

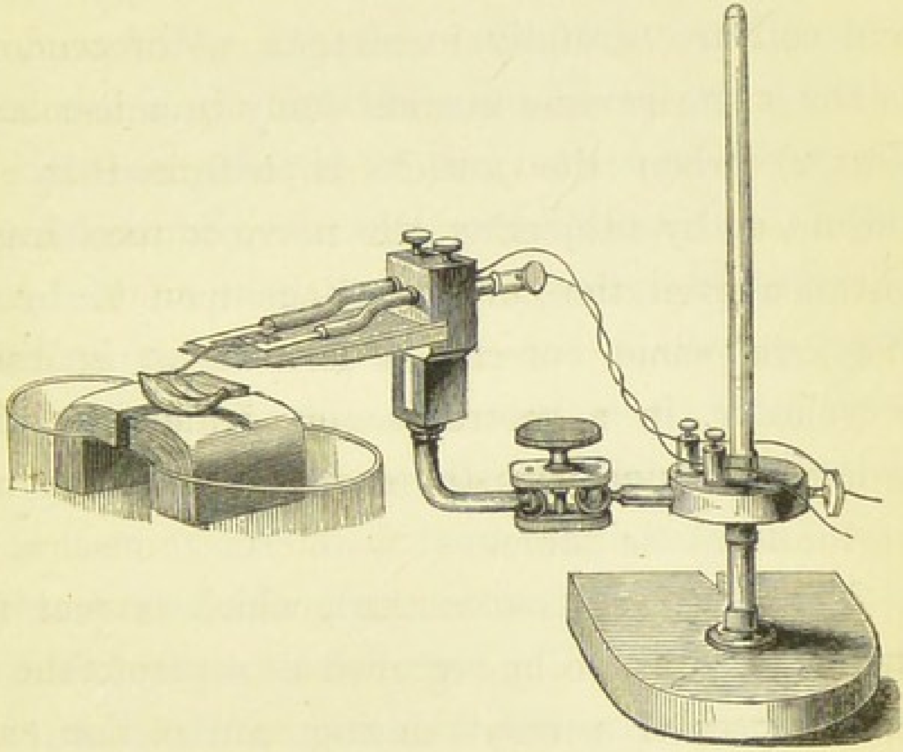
Tendon, like proper muscular fibre, is found to be the seat of evident electrical currents. If, for example, the tendon of a rabbit be so placed that its natural longitudinal surface is in connection with one cushion and the artificial transverse section with the other, the needle of the galvanometer is seen to move through a few degrees—seldom more than 5° and at most not more than 8°. It is also seen that this current moves in the same direction as the muscular current, *i.e.*, from the end to the side, that it readily becomes reversed, and that it dies out more slowly than the muscular current. If the tendo Achillis of a frog be examined, the results are the same, only here the evidences of the current are very indistinct; and similar results are also obtained when a portion of elastic tissue from the ligamentum nuchæ of a recently killed wether is put in connection with the galvanometer.

This current of the tendon is indeed feeble, but it is sufficiently active to react positively upon the muscular current, and this reaction will explain some of the difficulties with which we are at present concerned. It will explain, for instance, how it is that the muscular current in the gastrocnemius of the frog becomes reversed when the muscle has been subjected for some time to a freezing temperature. It is still Dr. du Bois-Reymond who has discovered the fact of this reversal, and supplied the explanation. He has found, indeed, that the previous *upward* current of the gastrocnemius is *brought back again*, although in greatly diminished force, when the tendinous expansion is removed by the knife or destroyed by chemical or other means, and at the same time he has proved that this bringing back is really due to the removal or destruction of the tendinous expansion, by showing that no such effect is brought about by cutting or otherwise destroying the sides of the proper muscular fibres of the muscle. In other words he has traced the feeble *downward* current of the frozen gastrocnemius to the tendon, and shown, not only that as this time the current of the tendon is contrary to that of the true muscle, but also that the muscular current may become so much enfeebled as to be overruled by the current of the tendon.

In the same way, also, we may in all probability explain why it is that a feeble *downward* current takes the place of the previous *upward* current of the hind limb of the frog (a fact I have repeatedly noticed), and passes for a short time before the final extinction of all electrical actions; and that this reversal happens equally when the previous upward current has been allowed to die out gradually, or when it has been suddenly exhausted by a series of alternating shocks from an induction coil; for if the current of the tendon comes into play when the muscular current has been weakened by cold, it may also be looked for in any case in which the muscular current has become sufficiently weakened.

If, then, this be the effect of the tendon, it follows, almost as a necessary consequence, that the course of the muscular current in particular muscles will be affected by the arrangement of tendon belonging to the muscle, and that the muscular current, *cæteris paribus*, will escape from that end of the muscular fibre at which it encounters least resistance from the current of the tendon. And as the arrangement of tendon is different in different muscles, or even in the same muscle in different animals, it may be inferred that the course of the current will be different in different muscles, or in groups of muscles,—for the general current which is derived from groups of muscles, as from the muscles of a limb, is only the resultant of the current of the individual muscles, or rather, it is only the resultant of the currents of the individual muscular fibres, for evidences of a current obeying the same law are found in the smallest as well as in the largest portions of muscular tissue.

— It would also seem as if the muscular current were *weakened* during ordinary contraction; indeed, there can be no reasonable doubt that this is the case. In demonstrating this very important fact, Dr. du Bois-Reymond makes use of the gastrocnemius of a frog with a long portion of its nerve attached. This muscle is placed upon the cushions of the galvanometer, and the nerve is arranged in such a manner that a series of shocks from an induction coil can be passed through a portion of it. As soon



as the muscle is laid upon the cushions, the needle of the galvanometer is deflected to some distance from zero, and it continues to be deflected, though not to same extent, so long as the muscle remains in a state of rest; as soon as the muscle is tetanized by passing the interrupted current through a portion of the nerve, the needle immediately travels back again and oscillates on the other side of zero. These are the simple facts. As soon as the muscle is tetanized, that is to say, *the needle is acted upon by a reverse current.*

Now it cannot be supposed that this current is due to the irruption of a current, from the induction apparatus by which the muscle is tetanized, into the circuit of the galvanometer, for both galvanometer and coil are carefully insulated. Moreover, there is the same reverse current (only in a less marked degree) when the muscle is tetanized by other means, as by subjecting the nerve to mechanical or chemical irritation, or by acting upon it by heat. Nay, the same current is seen to be produced by strychnine in a gastrocnemius which is still in living connexion with the nervous centres by means of the ischiatic nerve.

Nor is the reverse current, which is manifested during tetanus, to be regarded as a proof that there is a muscular current during contraction, whose direction is contrary to that of the current which plays during the state of rest. On the contrary, it is possible that the muscular current, before the state of tetanus was induced, may have given rise to a reverse current within the circuit of the galvanometer by evolving the secondary polarity of the platinum plates, and that this reverse current may be manifested during tetanus, in consequence of the proper muscular current having become *weakened*. And that this is the true explanation may be seen by the following modification of the same experiment.

The only difference between the experiment as modified and the original experiment is this—that one of the electrodes, instead of being connected with the galvanometer by a continuous piece of wire, is connected by a broken wire, of which the two ends are made to dip into a small cup of mercury. The difference, that is to say, is in a simple contrivance for rapidly breaking or closing the circuit; for it must be plain that the circuit is broken when an end of the wire is raised out of the mercury, and closed when the same end is replaced in the mercury. In performing the modified experiment, the muscle is first laid upon the cushions, and a note made of the degree to which the needle of the galvanometer is deflected. Then the muscle is removed from the circuit of the galvanometer, and the instrument depolarized by raising one end of the wire out of the mercury. In the next place, the muscle is tetanized, and while it is in this state it is included in the circuit of the galvanometer by replacing the end of the wire in the mercury. In this way then, the current of the tetanized muscle is for an instant separated from that reverse current of secondary polarity, which is caused by the continued action of any current upon the instrument, and the result is that the needle travels in the *same* direction as that in which it travels under the current of the untetanized muscle, *but not to the same distance from zero.* In other words, the muscular current of the tetanized muscle is found to be *weakened,* but not changed—as it would appear to be if no care be taken to eliminate the influence of secondary polarity from the experiment. In several instances in which I repeated this experiment upon the ordinary frog, the primary deflection of the needle, under the current of the elongated muscle, was from 40° to 60°, and the permanent deflection from 5° to 7°, whereas the primary deflection of the needle, under the current of the tetanized muscle, was from 8° to 15°, and the permanent deflection from 1° to 2°.

Dr. du Bois-Reymond, however, is of opinion that it is not enough to look at the needle of the galvanometer in deciding this question, and he is of this opinion because the phenomenon of *induced* contraction (which phenomenon is manifested, as Professor Matteucci was the first to show, by placing the nerve of a second muscle upon a muscle in which contraction is to be brought about) appears to point to some *oscillation* in the current of the primarily contracting muscle, which oscillation is not revealed by the needle of the galvanometer. He is of this opinion, because the phenomenon of *induced* contraction, in being oscillatory and not continuous, appears to point to some oscillation in the current of the muscle in which the *inducing* contraction is manifested. Dr. du Bois-Reymond supposes further that the induced contraction is due to these *oscillations* in the muscular current of the muscle in which the *inducing* contraction is manifested; and supposing this, his next inference is that there could be no contraction in the muscle in which the *inducing* contraction is manifested unless its current had taken upon itself a similar oscillatory movement. Arguing from this phenomenon of induced contraction, he thinks, indeed, that the condition of the muscular current in muscular contraction is one of *oscillation* and not of simple *failure* (as the needle of the galvanometer would seem to show); and he concludes that there could be no contraction if the current remained *constant*. But it is not easy to perceive the force of this mode of reasoning. It is easy to understand that the needle of the galvanometer may be too sluggish to oscillate with every single oscillation of the current, but it is not easy to allow that the needle is too sluggish to take up a position in the mean of oscillation. And if the needle is not too sluggish for this, then the muscular current must be *weakened* if the current fails to keep the needle at the same degree of divergence as that at which it stood before the muscle began to contract,—for certainly the fact of trembling does not alter the fact of weakness. At any rate, this theory of oscillation is not necessary to account for the phenomenon of induced contraction, and that it is not will be sufficiently obvious hereafter.

— And, lastly, it is found that all evidences of the muscular current have disappeared when the muscle has passed into the state of *rigor mortis.* It is found, indeed, that the muscular current is most active when the "irritability" of the muscle is most perfect, that it fails *pari passu* with this "irritability," and that it has died out altogether when the muscle has passed into the state of *rigor mortis.*

— On turning from these considerations to those which concern the action of the ordinary galvanic current upon muscle, it is to be expected that the existence of the muscular current is not to be ignored. And further, it is to be expected that certain reactions between the two currents will arise out of the particular course of the muscular current. It is no doubt true, as Dr. du Bois-Reymond represents, that the muscular current passes from the end or ends to the sides of the muscular fibre; but it is also true that this current must complete its circle and return to the point from which it started. It is certainly true that the muscular current cannot have reached the ends of the fibre without having first reached the interior of the fibre, and that it cannot have reached the interior without having first passed through the exterior of the fibre. In other words, it is certainly true that the muscular current, in passing from the sides to the ends of the fibre, must have reached the interior by passing in a more or less *transverse direction across the exterior parts of the fibre.* Now, if such be the course of the muscular current, it is evident, first of all, that there is no way of passing the galvanic current through a muscular fibre in which this current will not cross the course of the muscular current. The galvanic current, it is true, will not always cross to the same extent, and to a certain point it may coincide with the course of the muscular current, as it will do when it passes along the interior of the fibre in the direction in which the muscular current is predominant — when it passes, for example, in an *upward* direction through the gastrocnemius; but if it coincides with the muscular current in its course along the *interior* of the fibre, it must cross the muscular current in that part of its course where, in order to reach the interior, it passes more or less transversely through the *exterior* of the fibre. And if the two currents cross in this manner, then must they clash; and (to an equivalent degree) neutralize each other. In a word, it is not enough to regard the muscle as a simple conductor, through which the galvanic current has to pass and take possession; but it is necessary to regard it as the seat of a current which must be overcome before the galvanic current can pass and gain possession. Nor it is not enough to say that the muscular current returns when the galvanic current is suspended. The muscular current does return at this time—of this there can be no doubt, but there is also another current which springs into existence at the same time, and this is the ordinary *reverse* current which arises out of the secondary polarity of some part of the circuit. Just, then, as the muscle could not be looked upon as a simple conductor at the moment when the primary galvanic current began to pass, so now, it cannot be looked upon as a simple conductor at the moment when the muscular current returns to the muscle,—for before the muscular current can regain possession of the muscle it must contend with and overcome the reverse current above mentioned. It is evident, indeed, that there will be a clashing and equivalent neutralization between the muscular current and the reverse galvanic current at the moment when the circuit is opened, which is in every respect similar to that clashing and neutralization which took place between the muscular and primary galvanic current at the moment when the circuit is closed; and hence a moment in which electrical action is neutralized —a *moment of inaction*, as it may be called—may be supposed to precede both the establishment of the galvanic current and the return of the muscular current.

— What, then, it now remains to ask, are the phenomena which attend upon the passage of the galvanic current through the muscle, and how are they to be accounted for? The principal fact is, that a muscle contracts when the circuit is closed, and when the circuit is opened, but not during the time that the current is passing through it. Thus, in an experiment of Professor Matteucci in which a galvanic current was passed through the pectoral muscle of a pigeon from which every visible trace of nervous tissue had been carefully removed, the muscle was found to contract at the moment the circuit was closed, and at the moment the circuit was opened, but not during the passage of the current, and this order was found to be altogether irrespective of the direction of the current. In this experiment it was also found that the contraction on opening the circuit was less marked than the contraction on closing the circuit, —that both contractions became progressively less and less marked until they ceased altogether,—and that one or both of the contractions might be revived for a short time after their extinction by increasing the strength of the current.

Now in seeking for the explanation of these phenomena it is difficult, if not impossible, to find any reason for supposing that the contractions are due to any direct action of the current, natural or artificial. For what is the case? The case is that the muscle does *not* contract so long as the galvanic current continues to pass through it. The case is that the muscle does *not* contract so long as it is left to the undisturbed possession of the muscular current. But on the other hand there is no difficulty in connecting the contractions with that clashing and mutual neutralization of the muscular and artificial current, direct or reverse, of which mention has been made as attending upon the commencement and cessation of the passage of the artificial current through the muscle,—for if contraction is absent when the artificial or natural current is manifestly present, and if contraction is present when the artificial or natural current is manifestly absent, then there appears to be only one course open, and that is to connect the contraction with the *absence* of the current.

Upon this view, also, it is intelligible that the contractions should become less and less marked as the experiment proceeds, and that the contraction on opening the circuit should be less marked than the contraction on closing the circuit. Removed from the body, the muscle is found to yield fainter and still fainter indications of a current until these indications cease altogether, and hence it follows that the neutralizing reaction between the muscular current and the artificial current will fail more and more as the experiment proceeds,—will fail, because to diminish this current is evidently to diminish the amount of reciprocal neutralization,—and, so failing, that the contractions will become progressively less and less marked, for if the contraction is connected with the neutralization of electrical action in the muscle, the degree of the contraction must be directly proportionate to the degree of this neutralization. It is, also, intelligible that the contraction on opening the circuit should be less marked than the contraction on closing the circuit, for the lapse of time and the disturbing influence of the artificial current upon the electromotive elements of the muscle, will combine to weaken the muscular current—will combine, that is, to render the contraction on opening the circuit less marked than the contraction on closing the circuit, for—to repeat what has just been said—to weaken this current is to diminish those neutralizing reactions between the two currents, which, upon this view, are directly measured by the degree of the contraction.

Nay, it is in some degree possible, upon the same view, to explain the fact that the contractions should be reproducible, after they have once ceased, by increasing the strength of the artificial current; for in order to explain this difficulty, it is only necessary to suppose—and the supposition is certainly allowable—that the weaker current had only penetrated the muscle superficially, and that the stronger current had penetrated more deeply and reacted with the current of muscular fibres which had been beyond the range of the weaker artificial current. This supposition is certainly allowable, for current electricity, although of very low tension, must yet possess some tension, and on that account it must agree in some degree with electricity of tension in preferring to occupy the surface of the body which serves as a conductor.

— There is, no doubt, much that is not altogether satisfactory in some of these speculations, and it cannot well be otherwise until a clearer knowledge of electricity is brought to bear upon the subject; but at the same time it must be evident that there is no reason for believing that muscle is stimulated into the state of contraction by electricity, and that this state of contraction passes off when this stimulation is at an end; and it may also be allowed that there is some reason for concluding that muscular elongation, and not muscular contraction, is the state which is produced by the action of electricity upon the muscle.

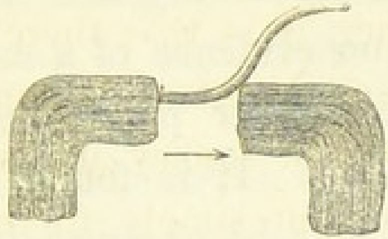
— Nor is there any objection to this view in the physical condition of the contracted and uncontracted muscle. At first sight, indeed, it would appear as if a stimulus had ceased to act when the muscle passes out of the contracted state, and it is no easy matter to get rid of this idea; but Professor Ed. Weber has shown, not only that the hardness of muscle in a state of contraction is simply owing to the straining of the muscle upon its attachments, but also that the contracted muscle is even softer than the uncontracted muscle when it is cut loose from these attachments. In other words, he has shown that the condition of the uncontracted muscle may be more correctly represented by the term *elongation* than by the term *relaxation*,—and hence the mere physical condition of the muscle does not oppose any objection to the idea that muscular elongation, and not muscular contraction, is produced by the action of electricity upon muscle.

2. *That muscular contraction is not produced by the stimulation of "nervous influence."*

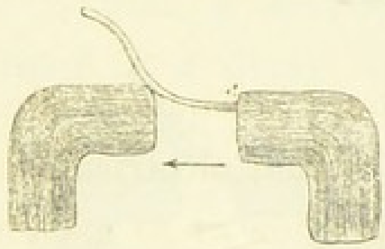
"Nervous influence" is a convenient term, which includes more agencies than one. It includes certain agencies which are thought to be vital, and it includes other agencies which come within the scope of physical inquiry. It is not necessary, however, to endeavour to determine how much is vital, and how much is physical, for if one thing is more probable than another, it is that there is one common force underlying all agents, vital and physical, and that the differences of agency are owing, not to differences of force, but simply to the differences of circumstance in which one and the same force is manifested. Now of the several agencies included in the term "nervous influence," electricity is the one which is most easily investigated, and hence we will begin to interpret the mode in which muscle is affected by "nervous influence" from an electrical point of view.

Dr. du Bois-Reymond was the first to demonstrate the existence of actual electrical currents in nerves, and his demonstration is so masterly and complete as to leave very little to be done by others. The original discovery of these currents was made by means of the smaller galvanometer, which had been used in investigating the "muscular current," and the subsequent investigations were made, partly with this instrument and partly with his large instrument. The experiments are, in many respects, similar to the experiments upon muscle, and the results are strictly analogous.

Placing a piece of the ischiatic nerve upon the cushions, so that it touched one cushion with the artificial transverse section, and the other with the natural longitudinal surface, thus—

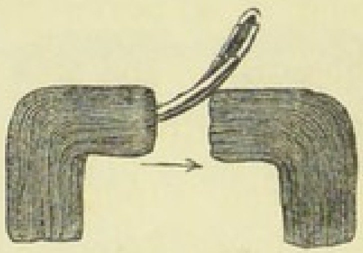


the needle of the galvanometer reveals the presence of a current from the end to the side of the nerve. Reversing the position of the nerve so that the other end is brought in contact with a cushion, thus—



the current still sets from the end to the side of the nerve. But if the nerve be placed so that the two ends or the two sides are in contact, one end or one side with each cushion, there is no current. Moreover, if two points at equal distance from the middle of the nerve are placed upon the cushions, there is no evidence of a current; but, if one point is nearer to the middle than the other, then there are signs of a current which passes from the end towards the middle. Indeed, these phenomena of the "nerve-current," as this current has been called, are precisely similar to the phenomena of the "muscular current," with this exception—an exception arising in all probability from the smallness of the parts—that no evidence of a current could be found between dissimilar points in the transverse section of the nerve. It is found, also, that the same phenomena may be observed in purely motor and sensory nerves, as well as in mixed nerves, like the ischiatic.

Dr. du Bois-Reymond has also shown that a current sets from both ends when both ends were brought together by doubling the piece of the nerve into a loop, thus—

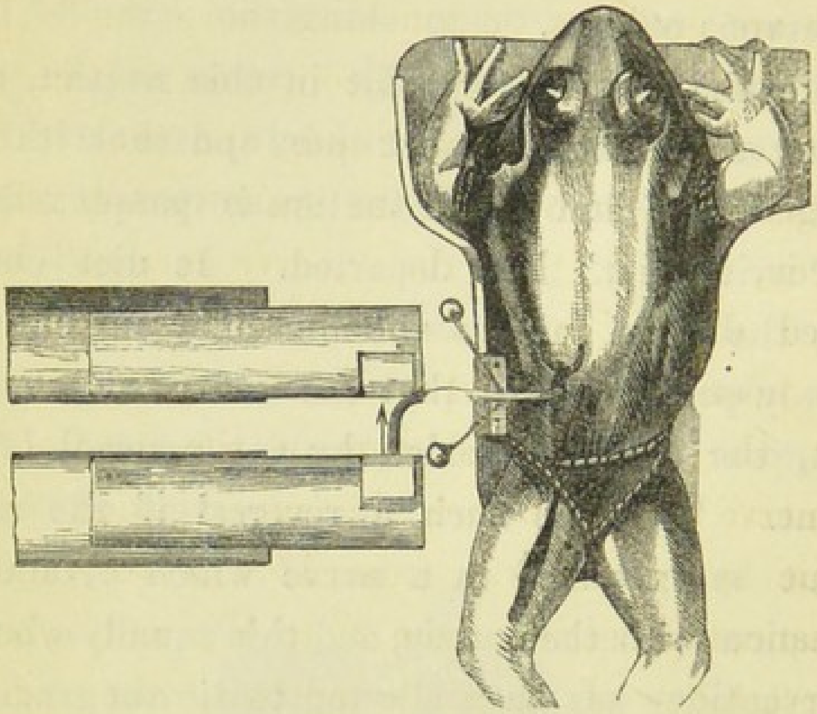


In the undisturbed condition of the nerve, however, as in the undisturbed state of the muscle, the nerve current is found to set in a particular direction, which, under ordinary circumstances, appears to be constant. Thus I find the nerve-current of the sciatic nerve to be in the same direction as that of the general current of the limb—that is, from the toes upwards, in the ischiatic nerve of all the animals I have examined, viz., the water-frog and land-frog, the mouse and rabbit. This may be easily seen in the frog, by laying the nerve of a galvanoscopic limb across the cushions, or by the more cruel expedient of bringing the same nerve into the same relations while it retains its connection with the nervous centres.

Like the muscular current in this respect, also, the nerve current dies out *pari passu* with the "irritability," and the extinction is complete when the "irritability" has departed. It dies out at about the same rate as the muscular current, and the only peculiarity is, that the act of death does not appear to be preceded by any reversal of the current. There is such a reversal if the nerve current be examined in a nerve which retains its connection with the muscle, and this equally whether the irritability has been allowed to die out gradually or whether it has been suddenly exhausted by a series of alternating shocks from an induction coil; but I find that this reversal is in some way connected with the reversal of the muscular current, for it ceases when the nerve is removed from the muscle, and it is never manifested when the nerve-current is investigated from the beginning in a separated nerve.

Dr. du Bois-Reymond has also shown that the "nerve current" agrees with the "muscular current" in that it affords evidence of enfeeblement *during muscular contraction.*

A frog is fastened upon a suitable frame, and then, after tying its common iliac artery, the ischiatic nerve is cut low down in the ham and



dissected out up to the vertebral column. After this the lower end of the nerve is bridged over the cushions of the galvanometer, so as to touch one cushion with its end and the other cushion with its side, and a note is taken of the degree to which the needle of the galvanometer is deflected by the "nerve current." The animal is then poisoned with two or three drops of a solution of nitrate of strychnia, and the effect of the tetanus upon the "nerve current" is attended to by watching the movements of the needle of the galvanometer. The experiment is simple and the result unmistakeable, for when the tetanus occurs *the needle recedes three or four degrees nearer to zero,* and this not only during the principal attacks, but also during those single shocks which are produced on touching the animal. It is seen further that the needle continues at this point so long as the tetanus continues, and that it again diverges from zero when the spasm passes off. It is seen, indeed, that the "nerve current" is enfeebled during muscular contraction. In some frogs upon which I repeated this beautiful experiment, the primary deflection of the needle under the nerve current *before* the supervention of the tetanic symptoms was from 15° to 20°, and the permanent deflection from 2° to 4°; and, *after* the supervention of these symptoms, the primary deflection was from 3° to 4°, and the permanent deflection scarcely perceptible. In these experiments I took the nerve from the cushions after testing its natural current, and replaced it in the wound. I then poisoned the animal with the strychnia, and when the spasms were fully established, I replaced the nerve upon the cushions. I did this in order to remove the nerve as much as possible from the desiccating influence of the atmosphere—an influence which might have put an end to the nerve current if the nerve had been allowed to remain upon the cushions in the interval during which the strychnia was taking hold upon the system.

— In turning from these considerations respecting the nerve current to those which concern the mode in which muscles are acted upon by galvanic electricity *through the instrumentality of the nerves*, there is one remarkable fact which claims immediate attention, and this is—*that the same results are produced by passing the current through a portion of a nerve as are produced by passing the current through the whole length of the nerve.* In the case of the galvanoscopic limb, for example, the same results are produced by including a small portion of the ischiatic nerve between the poles of the battery as are produced by placing one pole upon the extremity of the trunk of the nerve and the other upon the toes.

It is easily demonstrable, however, that the portion of the nerve which is not included between the poles of the battery is in the same electrical condition, or rather furnishes evidence of a current in the same direction, as the portion which is included between these poles, and hence it may merely be a natural consequence of the peculiar molecular structure of the nerve that the galvanic current cannot act upon a portion of a nerve without acting upon the whole nerve.

This is seen to be the case, by taking the sciatic nerve of a frog, and arranging it so that a portion of it is included within the circuit of a properly insulated galvanometer, and another portion between the poles of a galvanic battery, also insulated. A galvanic current is then passed through the portion of the nerve which is included between the poles of the battery, and upon doing this *a current is found to pass in the same direction through the portion of the nerve which is included in the circuit of the galvanometer*,—if, that is, the galvanic current be sufficiently strong to overrule the " nerve current" when this current is contrary. If the galvanic current be very weak, and the " nerve current" be in a contrary direction, it may fail to set up a current in the same direction as its own in the portion of the nerve which is included in the circuit of the galvanometer, but if it fails to do this, it will not fail to *weaken* the " nerve current." Indeed, there is no exception to the statement here made—that the effect of passing a galvanic current of sufficient strength through any portion of a nerve, is to set up a current in the same direction in every part of the nerve.

Now, it cannot be that the galvanic current has spread directly into every part of the nerve, and thence into the circuit of the galvanometer, for the insulation of the galvanometer and battery requires that this current should be confined to that portion of the nerve which is included between the poles of the galvanic battery; and, therefore, it is necessary to suppose that the galvanic current has determined a corresponding arrangement in the electromotive elements of the nerve itself; but be the explanation what it may, the fact is undeniable, and there can be no doubt that a portion of a nerve cannot be acted upon by a galvanic current without a corresponding action being set up in other parts of the nerve.

What, then, it may now be asked, are the phenomena which are manifested in the muscles when a nerve, or a portion of a nerve, is acted upon by the galvanic current, and how are they to be accounted for?

When a nerve, or a portion of a nerve, is included in the circuit of a simple galvanic apparatus, the results, as seen in the muscles, are very remarkable. If the experiment be performed upon the nerve of a recently prepared frog's leg, the muscles of the limb are seen to contract when the circle is opened and closed, and to remain elongated so long as the current continues to pass; and, at first, no very marked differences in the degree of these two contractions are produced by changing the direction of the current. Afterwards, when the excitability of the nerve has become diminished, there begin to be certain differences in the degree of these contractions, which differences are determined by the direction of the current in the nerve,—the law being (1) that the contraction on opening the circuit is more marked than the contraction on closing the circuit, *when the current is said to be "inverse"*—when, that is, it passes from the expansion to the origin of the nerve; and (2), that the contraction on closing the circuit is more marked than the contraction on opening the circuit, *when the current is said to be "direct"*—when, that is, it passes from the origin to the expansion of the nerve. At a later stage of the experiment the contractions are found to become fainter and fainter, until they cease altogether, the rule being that the contraction which was most marked is continued for some time after the other has disappeared. In this experiment, the galvanic current may be passed in either direction, but not up and down the nerve alternately or indiscriminately, and this is a point of considerable importance. Indeed, on reversing the current it is found that the muscles will again begin to respond to the current, even after they have altogether ceased to respond to the former current. It is also found that the contractions which arise on reversing the current in this manner, will attend for some time upon the closure and disruption of the circuit; but not for so long a time as that during which they attended upon the previous current. It is even

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possible to reproduce the contractions more than once on reversing the current in this way, and some time may elapse before these remarkable alternations, or *voltaic alternatives* as they have been called, will come to an end. These peculiar alternations are to a certain extent independent of the direction of the current in the nerve, but at the same time the direction of the current has a very appreciable influence upon the results. If the current be *direct*—that is, from the origin of the nerve to its expansion, and if it be passed for some time continuously, and the circuit then opened and closed, the opening and closure of the circuit are not attended by any contractions in the muscular fibres; but if the current be *inverse*,—that is, from the expansion to the origin of the nerve, this is not the case, and provided the galvanic current be not too powerful, the current may be passed through the nerve for a much longer time than in the last instance, and the contractions may still attend upon the opening and closure of the circuit. Instead of exhausting the excitability of the nerve, as did the *direct* current, it would indeed seem as if the *inverse* current had augmented this excitability; and this was the opinion of Professor Matteucci, who first directed attention to these differences in the effects of the direct and inverse currents. At any rate, there is a *marked* contraction on opening the circuit after the *inverse* current has been passing for some time through the nerve.

Now, in explaining the action of the artificial current upon the nerve, as in explaining the action of the same current upon the muscular fibre, the key to every difficulty would appear to be in the recognition of certain reactions between the "nerve current" on the one hand, and the artificial current on the other hand; and all that is necessary is to use this key with sufficient carefulness.

At first sight, perhaps, it is difficult to understand how it is that the artificial current should act so differently according as it is passed up the nerve or down the nerve, but on referring to the particular reactions which must take place between the galvanic current and the nerve current under these circumstances, this difficulty is done away with. The irritability of the nerve is impaired or destroyed when the galvanic current through the nerve is *direct*—when it passes, that is, from the origin to the expansion of the nerve; and this is not to be wondered at if the irritability of the nerve is to be measured, as it assuredly is, by the activity of the "nerve current" in the nerve. For what is the case? The case is that the *direct* galvanic current has been passed in a direction which is contrary to the direction of the *current of the sciatic nerve* (*v.* p. 42), and that, having been so passed, it must especially tend to weaken or extinguish this current. On the other hand, the irritability of the nerve is augmented, or at any rate revived, when the direction of the galvanic current in the nerve is *inverse*, or when it passes from the expansion of the nerve to its origin. In other words, the irritability of the nerve is augmented or revived when the galvanic current is passed in the *same* direction as the *current of the sciatic nerve*. And thus it is in some degree intelligible that a property of the nerve, which is to some extent connected with the nerve current, should be impaired by what has been called (hastily perhaps) the *direct* current, and preserved by what has been called (not less hastily) the *inverse* current, for these results may after all be no more than the natural consequence of the way in which the galvanic and nerve currents clash in the one case and coincide in the other case.

On referring more particularly to the reactions which must take place between the artificial current and the nerve current, it is possible to understand why the muscles should contract only at the opening or closure of the circuit, and not during the passage of the current.

Now, it is very evident that certain difficulties would have to be removed before it can be held that the muscles contract because the galvanic current has acted upon the nerve in the sense of a "stimulus." It is indeed true that the muscles contract when the current begins to pass in the nerve and when it ceases to pass, but it is not less true that *the muscles do not contract during the actual passage of the current through the nerve.* It is also true that *the muscles will pass out of the tetanized state when a constant current is passed through the nerve.* Two facts are indeed certain—one long familiar to all physiologists, the other recently discovered by Dr. Eckardt[1]—which seem to show, if they show anything, that the *action* of the galvanic current upon the nerve is to produce elongation, and not contraction; for in the one case, a muscle *already elongated* is seen to remain *elongated* so long as the current continues to pass through the nerve; and in the other case, a *contracted muscle* is seen to *become elongated* when the nerve is acted upon by the current. And certainly there is no contrary conclusion to be drawn from the fact that the muscle contracts when the galvanic current begins to pass in the nerve, and when it ceases to pass.

When a nerve, or a portion of nerve, is included between the poles of a galvanic battery, it will be with the nerve current as it was with the muscular current when a portion of muscle was so included,—

[1] 'Grundzüge der Physiologie der Nervensystems,' Giessen, 1854.

for the nerve current and the muscular current are absolutely obedient to the same law. *When the circuit is closed,* the galvanic current will pass across the course of the nerve current, and so passing it will neutralize, and receive an equivalent amount of neutralization from, the nerve current. And thus an instant of neutralization, a moment of inaction, will precede the establishment of the galvanic current in the nerve. *When the circuit is opened,* the galvanic current will be suspended, and the nerve current will return to the nerve; but before the nerve current can thus return, it will have clashed for an instant with that *reverse* current which springs into momentary existence and traverses the nerve and other portions of the galvanic circuit upon the cessation of the primary galvanic current. In other words, the re-establishment of the nerve current after the cessation of the artificial current, will also be preceded by an interval of inaction, in which the first instalments of the nerve current and the momentary reverse current suffer that reciprocal neutralization which arises out of the differences of their direction. And hence it would appear that there is some neutralization of electrical action in the nerve at the moment of closing and at the moment of opening the circuit, and that the nerve is only under full electrical action either when the artificial current is passing through it, or when the nerve is left to the influence of its own current.

And if these neutralizing reactions between the galvanic current and the nerve current are connected with the contractions, it is possible to understand, in some measure, how it is that the contractions should vary as they are found to vary, at different stages of an experiment in which the nerve is acted upon by a galvanic current.

It is possible to explain why the contractions should gradually die out. In order to these contractions, the hypothesis assumes that the antagonism of the nerve-current is essential, and hence it follows that the contractions will die out *pari passu* with the current. Nor is it impossible to explain the particular and very singular mode in which the contractions die out.

When the *direct* current is passed through the sciatic nerve, *the contraction on breaking the circuit is the first to die out,* and so it should be according to the premises. Under these circumstances, indeed, the effect of the artificial current—which is *opposed* to the current of the sciatic nerve—is to weaken the nerve-current, and hence it follows that the nerve-current will be weaker when it returns to the nerve after the galvanic circuit is broken, than it was before the nerve was included in that circuit. Under these circumstances, indeed, the effect of the galvanic current will be to weaken the nerve current, and in that way to render that contraction less marked which happens when the galvanic current ceases to pass,—for this contraction, according to the premises, is proportionate to the amount of neutralization which takes place between the nerve current and the artificial current—in this particular case, the reverse current. And hence it is—as soon as this nerve current has become weakened by the action of the artificial current upon the nerve—that the apparent effect of the so-called *direct* galvanic current will be to weaken the contraction which happens when the circuit is broken more than to weaken the contraction which happens when the circuit is closed.

When, on the other hand, the so-called *inverse* current is passed through the sciatic nerve, *the contraction on closing the circuit is the first to die out*, and so it should be according to the premises. Under these circumstances, the artificial current is passed in the *same* direction as the current of the sciatic nerve, and its effect is to revive the nerve current rather than to exhaust it. Under these circumstances, indeed, the effect of the galvanic current is to revive the nerve current, and in that way to perpetuate the contraction which happens when the galvanic circuit is broken. And hence—as soon as the nerve current is really weakened by the action of the galvanic current upon the nerve—the apparent effect of the *inverse* current will be to weaken the contraction which happens when the circuit is closed more than the contraction which happens when the circuit is opened.

Nor is there anything unintelligible in the *voltaic alternatives* when the aid of the galvanometer is brought to their interpretation. For what is the simple fact? The fact is that the direction of the current in the sciatic nerve is altered by the prolonged passage of a galvanic current, and that *the nerve current has become reversed when the closure or opening of the galvanic circuit ceases to provoke contraction in the muscles supplied by the nerve.* This fact I have ascertained by repeated experiments upon the common frog. So long, indeed, as the muscles continue to contract when the galvanic circuit including the nerve is closed or broken, so long does the nerve current continue to pass in the same direction as at first; and when the contractions on opening or closing the circuit have come to an end, then the nerve current has become reversed. So long, moreover, as muscular contractions attend upon the opening or closure of the circuit after the galvanic current has been reversed, so long does the nerve current continue to be reversed also. In the common frog I have not been able to pursue the voltaic alternatives beyond the second stage, for it always happened that at the end of this stage the reversal of the galvanic current had no further effect in renewing the contractions. At this time, also, it always happened that the nerve current had become extremely faint or altogether undistinguishable. In a word, then, there is always the same antagonism of the nerve current and the artificial current at the moment of the contraction, and when the contraction has come to an end the change may be traced to the cessation of this antagonism. And thus, upon these principles, the voltaic alternatives cease to be altogether unintelligible.

— Looking at muscular action, then, in relation to the *electrical* changes of the nerves concerned in this action, it would seem that muscular elongation is coincident with the *action* of natural or artificial electricity upon the nerve, and that muscular contraction is connected in some way with the diminution or annihilation of this action; and no reason has yet arisen which can support the idea that any vital property of nerve has been called into active exercise during the time of contraction by the "stimulus" of electricity.

— And, certainly, there is little reason to believe that muscular contraction is produced by any stimulation derived from the nervous centres.

Indeed, there are certain facts which seem to be altogether fatal to such an opinion.

After destroying the spinal cord in the lumbar region of a pigeon, the muscles of the paralysed legs are soon found to become hard and contracted. At first these muscles are soft; in a few days they become somewhat hard; and, after a few weeks, they pass into an evident state of contraction—a state which serves to keep the legs continually extended and divergent. This fact was first noticed by Drs. Brown-Séquard[1] and Martin-Magron.

It has also been pointed out by the same physiologists that the facial muscles of a rabbit become contracted after the division of one of the facial nerves, and that not on the healthy side, as in man, but on the paralysed side. This deviation goes on gradually increasing for three or four weeks, and at last it may be very considerable. In one case, twenty-one months after the operation the bones even had become altered in form, and the whole face had a strong twist towards the paralysed side. In addition, also, to this permanent state of contraction, there is a marked disposition to tremor and convulsive movement in the paralysed muscles, particularly when the breathing of the animal is temporarily interfered with. In a dog, a cat, a

[1] 'Experimental Researches applied to Physiology and Pathology.' New York, 1853. By E. Brown-Séquard, M.D., p. 104.

guinea-pig, there was generally no permanent contraction on either side after division of the facial nerve, but convulsive movements were very common in the paralysed muscles. It is also evident that the nerve could have had no share in these movements, for its peripheric portion had entirely lost its vital properties upon the fifth day after the operation, or thereabouts.

There are also certain experiments by Dr. Brown-Séquard,[1] which show that the muscular power of the hind legs of a frog becomes greatly increased *after* the division of the spinal cord.

In these experiments weights of different sizes are suspended from a small hook that has been previously attached to one of the hind legs a little above the heel. The animal is then held up by its fore limbs, and a weight that is just sufficient to put the hind leg gently upon the stretch is placed upon the hook. In the next place the toe of the weighted leg is pinched, and the weight is changed for one heavier until the animal is no longer able to withdraw its leg from the torture to which it is subjected. This being done, the next thing is to divide the spinal cord immediately behind the second pair of nerves; and to go on testing the muscular power of the paralysed legs. The results are very strange. Immediately after the operation, the

[1] 'Comptes Rendus,' May 10th, 1847.

the muscular power that can be put forth by the weighted leg when the toe is pinched is sometimes nil, but generally it is no more than a third or fourth of what it was before the operation. Fifteen minutes later this power is evidently rallying. In twenty or five-and-twenty minutes it has recovered all it had lost. An hour after the operation it is greater than it was before the operation, perhaps doubled. An hour or two later still it is certainly doubled, and possibly trebled; and from this time up to the twenty-fourth hour, when the increase generally attains its maximum, it goes on slowly augmenting. The particulars of two experiments with very fine frogs (A and B) were as follows, the weights raised being expressed in grammes:

	Before the Operation.	Immediately afterwards.	5 minutes afterwards.	15 minutes afterwards.	25 minutes afterwards.	1 hour afterwards.	2 hours afterwards.	4 hours afterwards.	24 hours afterwards.	48 hours afterwards.
A	60 gram.	20	45	60	80	130	140	140	150	150
B	60 gram.	10	30	40	60	100	120	130	140	140

When the increase of the muscular power has attained its maximum, it may remain nearly stationary for six, ten, fifteen, or twenty days, and after this time it fails by slow degrees. In a month, if the animal lives, this power will have fallen to its original value before the operation; and at the expiration of six, seven, or eight months, it may have fallen still lower, until it is not more than a third or a half of this value. It is possible, however, that the increased muscular power would not have failed in this way if care had been taken to exercise the paralysed limbs by galvanism.

Nor is it easy to agree with Dr. Marshall Hall in thinking that this increase of muscular power is due to an increased stimulation of the muscles on the part of the spinal cord (which inordinate stimulation had come into play because the controlling influence of the brain had been withdrawn), for there are other experiments which show plainly enough that the muscular power is augmented, not only in a similar but even in a higher degree, after the muscle has been cut off altogether from the spinal cord.

In the paper on muscular irritability, to which reference has already been made, and which is deserving of study, as well for the facts as for the opinions contained in it, Professor Engel, of Zurich, has shown that muscles are more prone to enter into the state of contraction after the complete removal of the cerebro-spinal system. In this experiment he clips out the whole of this system, bones and all, and, after five or ten minutes, he finds that the muscles have become so irritable as to be thrown into a state of contraction by a blow on the table. He finds, indeed, that the muscles are as irritable as they are in narcotized frogs.

Some very conclusive evidence to the same effect is also furnished in the following experiment by Dr. Brown-Séquard.[1] In this experiment the spinal cord of a frog is divided immediately behind the roots of the brachial nerves, and then the nerves proceeding to *one* of the hind legs are cut through at the points where they leave the cord. *Two hours later both the hind limbs are separated from the body,* and the contractility of their muscles is tested by the prick of a needle and by galvanism. This is the experiment. The result is *that the "irritability is augmented" in both these limbs, and that this augmentation is most considerable in the limb whose nerves had been divided—in the limb, that is to say, which had been cut off from the spinal cord.*

This result is entirely opposed to the conclusion which had been previously arrived at by Dr. Marshall Hall, but, as Dr. Brown-Séquard shows, this conclusion is not at all warranted by the experiment upon which it is based. Dr. Hall paralysed one of the hind legs of a frog, by dividing its nerves, and then tested the "irritability" of this

[1] Op. cit. p. 68.

limb—first, by passing a galvanic current through the animal, and afterwards, by poisoning with strychnia. In this way he produced contractions in the different muscles, and, as might be expected, he found that these contractions were less marked in the paralysed limb. But this experiment does not show that the paralysed muscles had lost any of their irritability. On the contrary, it only shows that they had been put almost altogether out of the field of action—for strychnia has scarcely any action, and galvanism has *comparatively* little action, upon muscles which are not in connexion with the nervous centres—and that they did not contract, simply because they had been thus put out of this field of action.

Now it is very difficult to reconcile these several facts with the idea that muscular contraction is produced by any stimulation derived from the nervous centres, and if any conclusion may be drawn from them it is one, or it would seem to be one, which must harmonize with that which has been already drawn from the electrical aspect of nervous influence.

— But, it may be asked, is not a fatal objection to this view to be drawn from the mode in which the will acts in producing muscular contraction? Is it not certain that the will acts in this case by stimulating the muscle to contract? It is difficult, no doubt, to think differently. It is more than difficult to wean the mind from so old an idea. But, on the other hand, it must not be forgotten that the will may have *acted* in bringing about muscular contraction, not by imparting anything to the muscles, but by withholding something from the muscles; and this being the case, we may well refuse to allow a mere opinion respecting the action of the will, however sanctioned by time that opinion may be, to rank as an objection to a view of the action of "nervous influence" in muscular motion, which view appears to arise necessarily out of the general history of "nervous influence" as concerned in muscular motion.

— And if this last objection may be set aside, then there appears to be no difficulty in assenting to the proposition at the beginning of this section, which is, *that muscular contraction is not produced by the stimulation of "nervous influence."*

3. *That muscular contraction is not produced by the stimulation of the blood.*

Arguing from the comparative anatomy of muscle, it would seem as if a muscle were not most disposed to contract when it is most liberally supplied with blood. It would even seem as if the degree as well as the duration of contraction were inversely related to the supply of blood: thus, this degree and duration of contraction is greater in the voluntary muscles of fishes and reptiles than in the voluntary muscles of mammals and birds; greater in involuntary than in voluntary muscles; and greater in the muscles of any given animal during the syncope of hybernation than during the fever of summer life.

The fact, moreover, that *rigor mortis* may be "relaxed," and the lost "irritability" restored to the muscle, by the injection of blood into the vessels—a fact which has been abundantly demonstrated by Dr. Brown-Séquard[1] and Professor Stannius[2]—would appear to be in direct contradiction to the idea that the muscle is in any way stimulated to contract by the blood.

Dr. Brown-Séquard placed a ligature around the aorta of several rabbits, immediately behind the origin of the renal arteries, and in a short time he found that the hind limbs were deprived of feeling and power of motion. He then waited until *rigor mortis*, or a rigidity like *rigor mortis*, had seized upon the paralysed parts, and when this state had lasted for about twenty minutes he untied the ligature, and found that the sensibility and power of motion

[1] 'Comptes Rendus,' 9 et 25 Juin, 1851.

[2] 'Archiv für Physiologische Heilkunde' (Vierordt's), Heft i, Stuttgart, 1852.

returned as the blood again made its way to the parts from which it had been excluded.

A still more conclusive experiment was performed upon the arm of a criminal who had been guillotined at 8 a.m. on the 12th of July, 1851. This arm, which was severed from the body, was in a state of perfect *rigor mortis* at 11 p.m.—fourteen hours after decapitation—and at this time the experiment was commenced by injecting a pound of defibrinated dog's blood into the brachial artery. As the blood began to penetrate into the vessels, some reddish spots appeared in different parts of the skin of the forearm, of the arm, and more particularly of the wrist. Then these spots became larger and larger, and the skin acquired the appearance it has in rubeola. Soon afterwards, the whole surface had a reddish-violet hue. A little later still, and the skin had acquired its natural living colour, elasticity, and softness, and the veins stood out distinct and full as during life. Then the muscles relaxed, first the fingers and lastly the muscles of the shoulders, and on examination they were found to have recovered their lost irritability. At 11·45 p.m. the muscles were more irritable than they had been at 5 p.m., at which time the corpse was first examined; and this degree of irritability was kept up, without abatement, until 4 a.m., when fatigue compelled Dr. Brown-Séquard for the time to abandon the experiment. When the experiment commenced the temperature of the blood was 73° Fahr., and that of the room 66° Fahr.

The subject of another experiment was a full-grown rabbit, which had been killed by hæmorrhage. Dr. Brown-Séquard waited until *rigor mortis* had fully set in, and then injected the defibrinated blood of the same animal into one of the hind limbs, which limb had been previously removed from the body. Fifteen minutes afterwards the muscles had lost their stiffness, and responded readily to mechanical or galvanic irritation. From this time, through the night, until 3 p.m. on the day following, the blood was injected at intervals of from twenty to thirty minutes, and all this time the muscles were perfectly soft and irritable. All this time, also, the muscles of the other hind limb, and of the rest of the body, were in a perfect state of *rigor mortis*. From 3 p.m. to 4·30 p.m. the injections were discontinued, and when they were resumed the limb had again become rigid, with the exception of a few bundles of fibres here and there. The effect of the injections was precisely as at first, and when they were again abandoned, from the lateness of the evening, the muscles were as soft and irritable as before. On the following morning, the limb upon which the injections had been practised was in a perfect state of cadaveric rigidity, while the muscles of the rest of the body, which had been left to themselves, were already beginning to pass out of this state. On the third morning the *rigor mortis* of the left limb was undiminished, and the other muscles of the body were in an advanced stage of putrefaction.

About the time that Dr. Brown-Séquard was engaged with these interesting experiments, Professor Stannius, without any knowledge of what was being done in Paris, was carrying out an analogous series of inquiries in Rostock. Dr. Brown-Séquard published the account of his experiments in June, 1851; Professor Stannius published his account in the beginning of 1852; and therefore the first-named physiologist has the priority in so far as their results agree.

The experiments of Professor Stannius were performed upon young dogs, and two will serve as examples of the fifteen which are given.

On the 21st of July, 1851, at 7·30 a.m., Dr. Stannius tied the abdominal aorta and crural arteries of a young dog. About 10·15 a.m. the muscles began to stiffen in the parts from which the blood was excluded, and at 10·45 a.m. both hinder extremities were stretched out, and perfectly stiff and cool. At 11·40 a.m. the ligatures were loosened, and the blood was seen and felt to penetrate into the empty vessels. At 11·45 a.m. both hinder extremities were warmer, and the right appeared to be a little more flexible than the other. At noon these limbs had recovered their flexibility, and it appeared once *as if the left had moved spontaneously,* but no sign of pain was caused by pinching the toes. At 12·25 a.m. incisions were made into both paralysed limbs, and everywhere the muscles were seen to contract upon the application of the electrodes. At one point, also, *there was evidence of pain,* for the animal, which was before quiet, gave a sudden plunge forwards. Death happened unexpectedly at 12·28 p.m.

A similar operation was performed upon another young dog, on the 22d of July, 1851, at breakfast time. At noon there were no evidences of stiffness in either of the hind limbs, but the muscles below the knee had ceased to respond to the touch of the electrodes. At 2·15 p.m. both these limbs were stretched out and rigid, and all evidences of irritability were at an end. At 2·35 p.m. the ligature around the aorta was untied, and at 2·50 p.m. the ligatures around the crural arteries. At 3·35 p.m. the application of the electrodes caused strong contraction in the muscles of both thighs, and weaker contractions in the muscles of the left leg below the knee, while at the same time nearly all traces of rigidity had disappeared from both limbs. At 5·35 p.m. every trace of stiffness had disappeared, and the muscles responded perfectly to the prick of a knife, as well as to the touch of the electrodes. On the following morning the animal was found dead, and the rigidity of death was fully established everywhere.

Now, the stiffness of which mention is here made is perfectly identical with *rigor mortis*, and this will appear from the following experiment. In this experiment, the aorta and crural arteries of a young whelp were all carefully tied, and the operation was over at 8·22 a.m. At noon all irritability had disappeared, and the muscles behind the ligature had become perfectly rigid. Seven and thirty hours after the operation the animal was still alive—at least in its anterior half—and upon the whole it was comparatively fresh and quiet. On the following morning it was found dead, with the parts *before* the ligature in a state of *rigor mortis*, and with the parts *behind* the ligature, not in a state of *rigor mortis*, but flaccid, moist, and partially putrescent. In other words, the parts *behind* the ligature were in the state which comes on after *rigor mortis*, and hence it follows that the stiffness which existed in these parts before the complete death of the animal must have been identical with *rigor mortis*.

Now, these experiments would only seem to be intelligible upon the supposition that the influence of the blood, be the *modus operandi* what it may, is exercised in counteracting, and not in causing, muscular contraction; and this conclusion, which I had drawn from the experiments of Dr. Brown-Séquard before my attention was directed to those of Dr. Stannius, is the same conclusion as that which (*v.* p. 6) Dr. Stannius himself has drawn from his own experiments.

— There are, however, certain facts[1] which seem to show that living, irritable, muscle is affected differently by arterial and by venous blood, and these facts have led Dr. Brown-Séquard to think that the office of arterial blood is to minister to the nutrition of muscular and other tissue, and to the storing up of contractile and other forms of power, and that the office of black blood—ordinary venous blood, or the blood of asphyxia—is to supply a stimulus by which the power derived from the red blood is called into action. Hence, according to this view, the function of venous blood is not a whit less important to the interests of the economy than that of arterial blood.

One argument in favour of the idea that muscle and other contractile tissues are stimulated to contract by venous blood, is based upon the well-known fact that these tissues are thrown into violent and general contraction when the whole mass of blood has become venous, as in asphyxia.

[1] Op. cit. and 'Comptes Rendus,' No. 16, 1857.

Another argument, also found among the phenomena of asphyxia, is derived from the fact that the left ventricle of the heart appears to pulsate more powerfully during the first moments of asphyxia, for at this time the pulse is fuller and firmer, and the mercury is raised to a higher point in the hæmadynometer.

Other arguments are based upon certain original experiments of Dr. Brown-Séquard.

In one of these experiments the abdomen of some mammiferous animal, generally a rabbit, was opened, and black or red blood was injected alternately into the aorta above the origin of the renal arteries. On injecting black blood convulsive movements were set up in all the parts to which the blood had penetrated; on injecting red blood these movements were suspended. The convulsions, moreover, were most violent when the blood was blackest, and most speedily brought to an end when the blood was richest in oxygen.

In another experiment the pregnant uterus of a bitch or doe-rabbit was separated from its connections with the cerebro-spinal centres, and blood was injected into the aorta. On injecting black blood the uterus was thrown into a state of contraction, and one or more fœtuses were expelled; on injecting red blood this contraction passed off.

Dr. Brown-Séquard says further, that muscles of animal life, paralysed by the division of their nerves, behave in the same manner under the influence of red and black blood, with this difference only, that the contractions caused by the black blood are less marked than in the two experiments just mentioned.

Now these are facts which show unequivocally that muscle is affected very differently by venous blood and by arterial blood, but they are very far from showing that muscle is stimulated to contract by venous blood.

It may, indeed, be questioned whether the convulsions of asphyxia are not rather due to the want of the stimulus of red blood than to any stimulus derived from the black blood with which the system has become charged; for it is certainly true that the muscles are similarly convulsed when an animal is bled to death. In other words, it is certainly true that the muscles are similarly convulsed, as well when the animal is left without blood as when it is left full of venous blood.

Nor can it be allowed that the (apparently) more powerful contraction of the left ventricle during the first moments of asphyxia are due to increased stimulation on the part of the venous blood; for the fuller and firmer pulse, and the rise of the mercury in the hæmadynometer may be owing, not to increased contraction in the ventricle, but simply (as is indeed allowed on all hands) to the fact that there is some impediment to the free flow of blood through the capillaries of the systemic circulation—an impediment, that is to say, by which the systole of the ventricle is made to expend itself with greater force upon the coats of the intermediate arteries.

Again, it may be questioned whether the muscular contractions which are produced in the two other experiments, when black blood is injected into the vessels, may not also be due to the want of some stimulus belonging to the red blood rather than to the action of any stimulus derived from the venous blood. At any rate it is well known that the uterus has often contracted and expelled its contents when a pregnant animal has been bleeding to death. And there are several facts on record in which the human uterus, even, has expelled its burden after the mother has yielded to the utter syncope of actual death.

As it seems, however, the grand difficulty in the way of accepting this idea—that muscle is stimulated to contract by venous blood—is a chemical difficulty. For what is the main difference between arterial blood and venous blood? It is that the oxygen of the former has in the latter become displaced by carbonic acid. Now carbonic acid has an action upon all parts of the nervous system which minister to intelligence or sensibility—upon all parts of the frame indeed, with the supposed exception of those which minister to motion—which action is so obviously opposed to that of stimulation, that it is extremely difficult to suppose that carbonic acid can be a stimulus in any case. Indeed, it is so difficult, as to make it wellnigh impossible to entertain such a supposition for a single moment.

— Nor is there any reason to believe that any kind of blood is ever a stimulus to muscular contraction. It is possible, perhaps, that such an idea might be gathered from the fact that the convulsions of hæmorrhage and asphyxia come to an end when the bleeding or choking animal is upon the point of death; for looking at this fact in one point of view it may seem as if the convulsions had come to an end because the stimulus of arterial blood was taken away. At the same time a conclusion such as this is neither natural nor necessary. Thus: if it be asked how it is that the loss of blood, or the want of arterial blood, is attended by convulsion, the answer must be that the muscles are affected, not directly, but indirectly. A certain change, be this what it may, is produced in the nervous centres, and this change issues in convulsion, if the nerves discharge their office of conductors between the nervous centres and the muscles. This is evident in this one fact, that the convulsed state of a muscle is immediately put an end to by the division of its nerve. And this being the case, the question is as to the condition of the nerves at the time when the convulsions of hæmorrhage or asphyxia come to an end. Have the nerves at this time ceased to be conductors between the nervous centres on the one hand and the muscles on the other? Now it is certain that a fair supply of red blood is necessary to preserve the conducting power of the nerves. Thus, when the principal vessel of a limb is tied, the sense of touch and the power of movement are paralysed, and this state of paralysis continues until the collateral circulation is established. And if it be certain that a fair supply of red blood is necessary to preserve the conducting power of the nerves, then it is reasonable to suppose that the nerves may have ceased to conduct all natural telegraphic messages at the time when the convulsions of hæmorrhage or asphyxia come to an end, for at this time the supply of such blood to the nerves must be defective in the extreme. And if the nerves have ceased to be conductors the convulsions must cease, for in muscles which are isolated from the nervous centres, and left to themselves, the "muscular current" and the "nerve-current" will be re-established in the muscle, and as a consequence of this the contraction will disappear. And not only must the muscles become elongated under these circumstances, but, according to the same process of reasoning, they must continue elongated until they are allowed to pass into the state of *rigor mortis* by the dying out of the "muscular current" and "nerve current."

In this way, then, it appears to be possible to set aside the apparent objection which has been mooted, and hence the fact that an animal is convulsed by loss of blood or by want of arterial blood, becomes an indirect argument against the idea that muscle is roused into a state of contraction by the stimulation of the blood, while at the same time it corroborates in some degree what has been already said upon the relation of nervous influence to muscular contraction, inasmuch as the loss of blood or the want of arterial blood must necessitate a less active condition of the nervous centres, and (as consequent upon this less active condition) a less liberal supply of "nervous influence" to the muscles during the convulsions.

— In a word, there is no sufficient reason for supposing that muscle is ever excited to contraction by the stimulation of the blood, and there is some reason for believing that muscular elongation and not muscular contraction is coincident with this action.

4. *That muscular contraction is not produced by the stimulation of any mechanical agent.*

When a muscle is touched by a needle or any other mechanical agent the contraction which follows is referred to the stimulation of some vital power of contraction inherent in the muscle; but this conclusion may well be called in question after what has been said already. After what has been said, indeed, it would rather seem that the foreign body had served to *discharge* something of which electricity was one of the signs, and that the contraction was the result of this discharge. At the same time this idea is not without its difficulties.

The first difficulty is to be found in the fact that the muscle will contract with equal readiness and power whether it be insulated or not insulated, or whether it be touched with a conductor or a non-conductor. This is a difficulty, but it is one which may perhaps be overcome by interpreting the electrical history of the muscle by some points in the history of the electrical organs of the torpedo. Nor is this mode of interpretation at all illegitimate, for the analogy between the electrical organ and muscle has been abundantly proved by Professor Matteucci and others. Thus : the nerves of the electric organs arise from the anterior tract of the spinal cord, and terminate in loops or loop-like plexuses; and in this they agree with the nerves of the muscles. The electric organs are paralysed by the division of their nerves; and in this they agree with the muscles. The electric organs are made to discharge their fire by irritating the ends of the divided nerves which remain in connexion with them, and the discharge is limited to the part to which the irritated portion of the nerve is distributed; the muscles also contract under these circumstances, and the contraction is equally localized. The electric organs are exhausted by exercise and recruited by repose; so are the muscles. And, lastly, the action of strychnia upon the two organs is analogous, in that a state of tetanus is produced in the muscle, and a succession of involuntary discharges from the electric organs. There are, indeed, many and obvious points of resemblance between the electric organs and the muscles, and, therefore, any interpretation which may be furnished by these organs may be supposed to apply directly, rather than indirectly, to the question under consideration.

What, then, it may be asked, is the process which takes place in the electric organ when it is touched by a foreign body? It is evidently the discharge of electricity previously present. If the fish be touched in a particular manner, a severe shock is the sign of this discharge; but the discharge is not less real when the shock is not felt in this manner. The discharge will indeed take place when the animal is touched by a piece of glass or other non-conductor; or it will take place when the animal is not touched at all, as under the influence of strychnia. This is evident, as well from the continuance of the convulsive movements which accompany every discharge, and particularly from the singular retraction of the eyeballs, as from the exhausted state in which the animal is left after the experiment. In a word, it is not necessary to the discharge that the electricity discharged should extend *beyond* the animal discharging. If fit channels have been provided, the current may extend beyond the animal, and in that case the discharge, with its attendent shock, may be felt accordingly; but if these channels have not been provided the discharge will take place within the animal itself.

And if this be so, the necessary inference is that the electricity of a muscle *may* be discharged indifferently by a conductor or by a non-conductor, for all that is necessary is to suppose that the discharge does not extend beyond the muscle when the non-conductor is used.

Another difficulty is to be found in the fact that muscle contracts with very different degrees of force when it is touched lightly and when it is tapped briskly. When tapped briskly a strong contraction is the result; when touched lightly there may be no contraction at all. In a word, the degree of contraction would seem to be proportionate to the stimulation of the touching or tapping body. It would seem, also, as if these phenomena could not easily be explained on the supposition that some agent like electricity had been discharged, for, according to this view, it may be said that the discharge ought to be equally complete, and the consequent contraction equally marked, when the muscle was touched in the lightest way possible, in that any touch must be sufficient to bring about this discharge. This may be said, but at the same time it is very possible that the tension of the electricity in the muscular fibres may be so low, and the insulation of these fibres so complete, as to require very perfect contact—something more than a light touch—before any discharge can be produced; and it is also possible that the effect of the firmer tap may be to bring more fibres within what may be called discharging distance, and in that way the more marked contraction may be the natural sign of the increased range of the discharge. At any rate, it is very plain that more fibres are made to contract by a sharp tap than by a light touch; for ocular demonstration of this may be had at any time by experimenting upon the surface of any muscle.

A third difficulty may also be disposed of by a little care and consideration. There is no doubt that a muscle will gradually cease to respond to the touch of a foreign body, and that the contractions will not recommence until after an interval of rest; but it does not follow from this fact that there is in the muscle a property of contractility which is exhausted by exercise and recruited by rest. At any rate it is quite as easy to explain the phenomenon upon the hypothesis under consideration. It is easy to suppose that a recently separated muscle will be more quickly charged with electricity than the muscle which has been removed from the body for a longer time, seeing that the molecular changes (respiratory, nutritive, and other), in which the electricity originates or consists, must be more active at first than they are afterwards; and, if so, then it follows that the recently separated muscle will be able to afford a greater number of those *discharges* of which (upon the provisional hypothesis at present under consideration) contraction is the sign. It follows, also, that an interval of time must be necessary to the production of the *charges*—an interval which for many obvious reasons must become continually longer and longer from the time when the muscle is removed from the body to the time when the molecular changes of the muscle come to an end; and hence the result will be precisely as if a vital power of contraction had been fatigued and required to be recruited by rest.

— Nor is the ordinary history of muscular contraction as seen in the vesiculæ seminales, or bladder, or bowel, or uterus, at variance with this mode of interpretation.

If there is one instance in which, more obviously than in any other, mechanical irritation would seem to have acted upon the muscles in the sense of a stimulus, it is in the contractions which take place in the vesiculæ seminales and elsewhere during the sexual orgasm, but even here a very different conclusion may be necessary. In one point of view, indeed, it is even probable that the irritation may have acted by diminishing, and not by increasing, the stimulus derived from the nerves. For what is the case with respect to sensation but this—that each sensation involves a corresponding expenditure of nervous influence and nervous substance? A given amount of nervous influence or nervous substance, so to speak, is used up in each sensation, and after a longer or shorter time the nerve is exhausted and enervated. And so also with the nerve-current. It is not, as might be expected, that this current is intensified at the instant of a sensation. It is a contrary change, for Dr. du Bois-Reymond has found that the current falls whenever the nerve is subjected to a treatment which would give rise to sensation if it remained in connexion with the brain. Thus, the current of the sciatic nerve of a frog is found to fall when the foot is dipped into hot water. Now in the case under consideration, it is not at all difficult to realise this idea and believe that sensation involves a corresponding expenditure of nervous influence, or nerve-current, or nerve-substance; for if there is any case in which exhaustion is the price which has to be paid for sensation, it is this. And if this is the case with respect to sensation, what is it with respect to motion? Is it to be supposed that the irritation which has acted in this manner upon the sensory nerves has acted similarly upon the motor nerves, and that contraction is set up in the vesiculæ seminales, and in other parts of the muscular system, because these motor nerves do not supply the usual amount of nervous influence to the muscles. At any rate, such a supposition cannot be regarded as improbable. On the contrary, there is so intimate a connexion between all parts of the nervous system, and particularly between the sensory and motor nerves belonging to the same part of this system, that it is scarcely possible to suppose that any change of state can be strictly limited to any one part. In pain or pleasure, without any figure, there is indeed no part of the nervous system which does not sympathize with every part, and hence (reasoning from the state of things which is evidently present in the sensory nerves) it is not at all improbable that the explanation of the muscular contractions occurring during the sexual orgasm may be altogether in accordance with the premises.

A similar mode of explanation may also be applied to the contractions by which the bladder or bowel is emptied, for the irritation of the accumulating urine or fæces may be supposed to have brought about the requisite state of enervation in the nerves concerned. At any rate, the uncomfortable feeling of fulness which precedes the contraction may be appealed to as an argument in favour of the idea that the energy of the different afferent nerves is being used up by being converted into sensation.

And, certainly, the doctrine of stimulation is not wanted to explain the parturient contractions of the uterus. At the time of labour this organ returns from the state of progressive expansion in which it had been during the period of pregnancy; and as *one* cause of the previous state of expansion would seem to be found in the increasing vital activity of the fœtus, so now *one* cause of the return from this state would seem to be found in the failure of this activity—a failure brought about, first in the mother, and afterwards in the fœtus, in consequence of the growth of the fœtus having then passed the limit beyond which it cannot pass without trenching upon the supplies necessary for the proper nourishment of the mother. It would seem, also, that this return of the uterus from the expanded state, or, in other words, this contraction of the uterine walls, must compress the vessels going to the placenta,—that the vital activity of the fœtus must suffer a corresponding depression from this interference with the sufficiency of the placental respiration—and that this depression must again lead to contraction in the uterus—for if this organ contracted in the first instance in consequence of a depression of this kind, there is no reason why it should not do so again. And, further, it would seem that this second contraction must lead to a third, and the third to a fourth; and that thus, the uterus acting upon the fœtus, and the fœtus reacting upon the uterus, contraction must follow contraction, until the completion of birth. Nor does it follow from this hypothesis that the uterine contraction should be unintermitting, for it is quite possible (this among other reasons) that the blood which is displaced from the uterus during contraction may temporarily "stimulate" the system of the mother to a degree which is inconsistent with an unintermitting continuance of contraction in any of the muscles belonging to the involuntary system. At any rate, it is quite impossible, upon any rational view of parturition, to refer the contraction of the uterus to any "stimulation" on the part of the fœtus, without ignoring the whole of the previous history of pregnancy.

— Such, then, are the considerations which appear to belong more or less intimately to the present section. They are imperfect, no doubt, and by themselves unsatisfactory, but taken in connection with what has been said in the three preceding sections, they serve to show, if they do no more, that many important difficulties must be done away with before we can conclude that muscle is stimulated to contract by any mechanical agent, natural or artificial.

5. *That muscular contraction is not produced by the stimulation of light.*

Regarding the question generally, contraction would seem to be favoured by darkness rather than by light. It is in the darkness, and not in the light, that contraction takes place in the irritable cushions of the untouched sensitive plant; and it does not seem to be far otherwise with the iris, for it is as easy to suppose that the radiating fibres of this organ elongate under the influence of light, and in that way close the pupil, as to suppose that this curtain is closed by sphincter fibres, which have a very doubtful existence. At all events, this explanation is supported by the authority of Bichât; it equally accounts for the phenomena: and it harmonises with the known influence of light upon the cushions of the sensitive plant.

6. *That muscular contraction is not produced by the stimulation of heat or cold.*

Muscle, it is found, will bear considerable variation of temperature without contracting, but if the temperature be higher or lower than a given point, it is immediately thrown into a state of contraction.

Now, there is reason to believe that the "muscular current" is more or less suspended by a temperature which is higher or lower than a given point, and not by intermediate degrees of temperature, and that the presence or absence of contraction in these experiments may be accounted for by the suspension or non-suspension of the "muscular current." Professor Matteucci has shown that the "muscular current" is suspended by a low temperature, and hence, according to the premises, it is intelligible that the muscle may contract under a sufficient degree of cold. On the other hand, I find that the muscular current is weakened to the last degree, or altogether extinguished by the amount of heat which is sufficient to produce contraction in the muscle. In this experiment I placed the gastrocnemius of a frog across the cushions of the galvanometer, and having laid the end of a small straight band of watch-spring upon it, I raised the temperature of the band by applying a spirit-lamp to the other end. It is intelligible, therefore, that muscle should contract under a temperature which is higher or lower than a given point, and that it should not contract under those intermediate degrees of heat which are not sufficient to weaken the "muscular current" to the point which allows of contraction.

And, certainly, it cannot be said that muscular contraction has been produced by any stimulative action of heat upon the motor nerves, for Dr. Eckardt has recently shown[1] that the effect of the heat is to impair the "irritability" of the nerve, and that the contraction which is caused by heat is coincident with the temporary loss of this "irritability." In this experiment, the leg of a frog is prepared so as to have a long portion of its nerve attached to it, and the irritability of the nerve is tested at different degrees of heat by immersing the nerve in water, the temperature of which may be raised by additions of hot water, and by observing the readiness with which the muscles may be made to contract by irritating the nerve with a needle. The experiment is

[1] Op. cit., p. 81.

simple, and the results are unmistakeable. Thus, in water about the natural temperature of the frog—about 70° Fahr.—the irritability of the nerve is not appreciably affected; in water of a higher temperature the irritability is sensibly impaired by every additional quantity of hot water; at 144° Fahr., or thereabouts, the muscles refuse to respond any longer to the action of the needle upon the nerve; in water at a still higher temperature *the muscles contract in obedience to the action of the hot water upon the nerve.* In other words, the muscle is exhibited as contracting under the influence of heat when the degree of heat is sufficient to have destroyed the irritability of the nerve. Or, as Dr. Eckardt expresses it, "das Zustandekommen der Zuckung durch eine momentane Zerstörung der Structur des Nerven bedingt sei."

— In a word, there is little reason for saying that muscular contraction is produced by the stimulation of heat, and there is scarcely any more reason for thinking that the muscles are ever made to contract under the stimulation of cold.

7. *That muscular contraction is not produced by the stimulation of any chemical or analogous agency.*

The recent investigations of Dr. Harley[1] upon the physiological action of strychnia and brucia

[1] 'Lancet,' June 7th and 14th, and July 12th, 1856.

are calculated to shed much light upon the mode in which muscle is affected by chemical and analogous agencies.

These investigations, which are of extreme importance in a therapeutical as well as in a physiological point of view, show very clearly that these poisons do not cause death by exhaustion, or by suffocation arising either from closure of the glottis, or from spasm in the walls of the chest, but "by destroying the powers of the tissues and fluids of the body to absorb oxygen and give off carbonic acid." It is argued that death is not caused by exhaustion, because it cannot be supposed that the system can be fatally exhausted in less than two minutes. It is proved, that death is not caused by closure of the glottis, because the animal dies as speedily when its windpipe has been freely opened before the administration of the poison. It is proved, moreover, that spasm in the walls of the chest is not the cause of death, because artificial respiration can be performed without averting or even deferring the fatal issue. At the same time, the animal seems to "feel a want of oxygen," and that this is one cause of death Dr. Harley shows very plainly by the examination of its blood.

In this examination Dr. Harley uses the fresh blood of the calf. Of this blood he takes two portions, and mixing a small quantity (0·005 grammes) of strychnia with one, he ascertains the amount of oxygen absorbed and carbonic acid given off by examining the composition of air that has been left in contact with each. In each case the blood is thoroughly saturated with oxygen by shaking it with fresh quantities of air; and after this it is corked up in a graduated tube with 100 per cent. of ordinary air, and frequently agitated for the next twenty-four hours. At the end of this time, the air contained in the tubes is analysed by Bunsen's method, and the following is the result arrived at:

	Composition of common Air.	Composition of Air after having been in contact with *simple blood* for 24 hours.	Composition of Air after having been in contact with *blood containing strychnine* for 24 hours.
Oxygen . . .	20·96	11·33	17·82
Carbonic Acid .	·002	5·96	2·73
Nitrogen . .	79·038	82·71	79·45
	100·000	100·00	100 00

The air, that is to say, which has been in contact with the blood containing strychnia, has more oxygen and less carbonic acid than the air which had been left in contact with simple blood; and thus it would appear, that less oxygen has been absorbed, and less carbonic acid given off by the blood containing strychnine. When brucine is used instead of strychnine, the only difference in the result is one of degree:

	Composition of common Air.	Composition of Air after having been in contact with *simple blood* for 24 hours.	Composition of Air after having been in contact with *blood containing brucine* for 24 hours.
Oxygen . . .	20·96	6·64	11·63
Carbonic Acid .	·002	3·47	2·34
Nitrogen . .	79·038	89·89	86·03
	100·000	100·00	100·00

As with strychnine, therefore, so with brucine, the air which had been left in contact with the poisoned blood, in that it contains more oxygen and less carbonic acid than the air which had been left in contact with the pure blood, has absorbed less oxygen, and given off less carbonic acid than the pure blood.

Dr. Harley has also shown very conclusively that strychnine has, in addition, a direct power of destroying muscular irritability.

In one of these experiments, in which the hearts of two frogs are cut out and placed, one in distilled water, the other in a solution of acetate of strychnine, the result is, that the heart placed in distilled water goes on pulsating regularly for twenty-four hours, and that the heart which had been placed in the poisoned solution, not only ceases to beat in a few minutes (from one to five according to the strength of the solution), but even passes into a state of rigor mortis before the other heart has lost its irritability.

In the other experiment, the hind legs of a frog are prepared after Galvani's method, and placed, one in a vessel containing distilled water, the other in a vessel containing a strong solution of acetate of strychnine. The muscles and nerves of these limbs are separately tested by galvanism, and the result is, that the muscles of the limb immersed in simple water, are seen to contract freely after the muscles of the limb immersed in the poisoned solution have passed into the state of rigor mortis.

The action of the strychnine upon the muscles, indeed, may be supposed to be in some degree analogous to the action upon the blood, for, as Dr. Harley points out, the destruction of the "irritability of the muscle may be supposed to imply the suspension of that process of absorbing oxygen and giving off carbonic acid—the so-called respiration of the muscle—which is certainly most energetic when the irritability is most marked."

At any rate, these very important facts go to show that the action of strychnia, in producing muscular contraction, is not an action of stimulation, for they show that the poison acts first of all by rendering the blood less apt to appropriate its stimulating element, oxygen, and in the second place by diminishing the irritability of the muscles.

In another place, moreover, Dr. Harley says—

"many other poisons, I doubt not, exert their influence in a similar manner; for I have found that hydrocyanic acid, chloroform, nicotine, alcohol, ether, morphine, and several other narcotics, have the same power of destroying the property possessed by the organic constituents of the blood of absorbing oxygen and exhaling carbonic acid."

— An inference as to what really takes place in muscle under the influence of chemical or analogous agencies may also be drawn from Dr. Eckardt's recent experiments upon the "irritability" of the nerves connected with muscle. Experimenting with an acid, for example, it was found that the "irritability" of the nerve was damaged by the application of the acid, and that the muscle was not made to contract *by the acid* unless the concentration of the acid was such as to destroy the "irritability" of the nerve. The experiment, in fact, is the precise counterpart of the one related in the preceding section, the only difference being that an acid of continually increasing strength was employed in this case, and heat of continually increasing intensity in that case. It was the same also in the experiments with other agents. The agents themselves were found to act very differently—some by attracting water from the nerve, some by altering the normal albuminous constituents of the nerve, and some in a more recondite manner—but all agreed in this that they acted by destroying the "irritability" of the nerve, and that they did not cause contraction until they had destroyed this property. The experiments, indeed, appear to show that these agents produce contraction by suspending, and not by exciting, the "irritability."

Nor is any evidence of a contrary character to be met with elsewhere. There is little or no "irritability" in the muscles of animals which have been killed by immersion in carbonic acid, carbonic oxide, hydrogen, sulphurous acid or chlorine—and as little in muscles to which narcotic substances have been applied. The "irritability," moreover, is completely destroyed by the application of concentrated acids or alkalies. On the other hand, the muscles retain their "irritability" for a long time in atmospheric air or oxygen. And this is as might be expected. It has been said, for example, that a muscle *may* contract under the touch of a foreign body, not because it was stimulated to do so, but because the electricity of the muscle was discharged at the time; and that the muscle might cease to contract under these circumstances when the *charge* of electricity necessary to the discharge had ceased to be produced. Hence, loss of "irritability," according to this view, is nothing more than the want of the requisite charge of electricity in the muscle —a view, it may be remarked in passing, which is quite consistent with the supposition that sufficient electricity may still be present in the muscle to counteract the rigidity of death. Now it is quite intelligible that oxygen should be necessary to the development of the full electrical charge of muscle, and hence there is no difficulty in understanding that the muscles should retain their "irritability" for a longer time in atmospheric air or oxygen than in carbonic acid, carbonic oxide, hydrogen, sulphurous acid, or chlorine. It is also intelligible that oxidization should not be the sole source of electrization. The mere contact of dissimilar metals, as zinc and silver in a vacuum or in hydrogen, for example, will give rise to a current. And, therefore, it is possible that there may be such dissimilarities in muscle as may react for a time and give rise to an electrical charge, of feeble tension perhaps, and such as may not be discharged by the touch of a foreign body under ordinary circumstances, but still sufficient to counteract the state of contraction so long as it continues. It is also possible that these dissimilarities may cease after a time—from coagulation of fluids, from desiccation of solids, or from some other cause—and, so ceasing, that contraction may supervene. And, finally, it is possible that a muscle may immediately and effectually lose its power of contracting under the action of strong acids or alkalies, for the physical integrity of the fibre is necessary to contraction upon any hypothesis.

— In a word, it is not necessary to have recourse to the doctrine of stimulation to account for the facts which present themselves for consideration under this section, and there is much reason for believing that muscular contraction is not produced by the stimulation of any chemical or analogous agency.

— Reviewing the whole evidence, then, there appears to be no reason why *rigor mortis* may not be taken as the type of muscular contraction in general. For what is the case with respect to this form of muscular contraction? The case is simply this. As long as there is any trace of that action of which the "muscular current" is a sign, so long is there no *rigor mortis*. As long as there is any trace of that action of which the "nerve current" is a sign, so long is there no *rigor mortis*. If this action dies out speedily, as in persons in whom the vitality of the frame has been exhausted by long life, or by chronic disease, such as consumption, the muscles become speedily rigid; if this action dies out slowly, as in persons who have been cut down suddenly in the full glow of life, the muscles are equally slow in becoming rigid. Once contracted, moreover, the muscles remain contracted until they break up in the ruin of final decay,—an event which happens most speedily in the case where the muscle retains its physical integrity least perfectly. And this is precisely as it should be according to the premises, for according to the premises all that is necessary to the commencement of *rigor mortis* is the cessation of that action of which electricity is a sign, and all that is necessary to its continuance is the absence of this action and the physical integrity of the muscular fibre. In a word, it is possible to explain those unexplained and seemingly contradictory facts which constitute the distinctive features of that contraction into which the muscles pass after death; and hence *rigor mortis* may be accepted, not only as a type of muscular contraction in general, but as an *experimentum crucis* in favour of the proposition —that *muscular contraction is not produced by the stimulation of any contractile power belonging to muscle.*

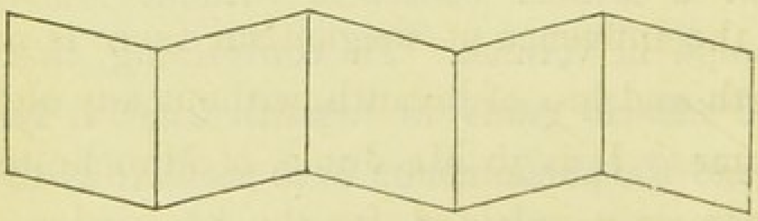
II. The Second Proposition.

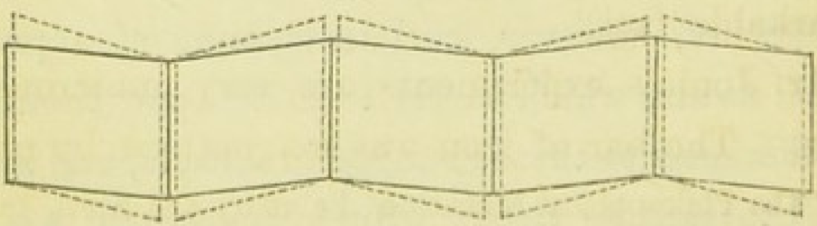
That muscular elongation is produced by the simple physical action of certain agents, electricity and others, and that muscular contraction is the simple physical consequence of the cessation of this action.

In seeking to establish the first proposition we are led, not only to believe that muscular contraction is not produced by the stimulation of any property of contractility belonging to muscle, but we are prepared to go a step further, and accept as probable the proposition which stands at the head of this section. For what is that view of muscular action which arises out of the previous consideration? It is that muscle and the parts more immediately related to muscle are acted upon by certain agents, electricity and others, and that the action is one which antagonizes contraction. It is a view, indeed, in which muscular contraction may be said to change from an active to a passive, from a vital to a physical phenomenon,—for there is nothing which does not seem to be capable of being reducible to a physical explanation. At the same time there are sundry difficulties of a very weighty character, which must be removed before such a view can be accepted.

A grave difficulty arises at the very outset in the fact that a muscle contracts without undergoing any change in volume. In contracting, that is to say, the muscle gains in breadth what it loses in length, and the phenomenon is so peculiar as to seem beyond the scope of any physical explanation.

There are, however, certain changes of a purely physical nature which would seem to approximate in their character to these changes in muscle, and it will be well to consider these. Such, apparently, are the changes which many crystalline bodies undergo under the influence of heat. Under this influence non-crystalline bodies dilate in every direction, becoming both longer and broader in so doing, and this is the case likewise with those crystalline bodies which are singly refractive and of equal axes; but *crystalline bodies (or at least some of them) which are doubly refractive and of unequal axes dilate only in particular directions.* If, for example, a primitive rhomb of calc-spar be heated, it *expands* in the direction of its shortest axis, and *contracts* in the direction perpendicular to the axis. At the same time the rhomb tends to become a cube by a change in its angles in which the obtuse angles become more acute and the acute angles more obtuse. Heated from 32° Fahr. to 212°, according to Mitscherlich and Dulong, the *expansion* of the shorter axis of this crystal of calc-spar is 0·00286, and the *contraction* in the directions perpendicular to this axis





0·00056, so that the relative expansion of the shorter axis is 0·00342 (0·00286 + 0·00056). These changes, which are very curious, may be roughly illustrated by two diagrams, in one of which a series of unexpanded rhombs is arranged longitudinally, and in the other a similar series of rhombs as altered by heat, only with the degree of alteration exaggerated for the sake of distinctness.

— But there is a more direct illustration of the changes in muscular fibre than that which may be found in the changes of calc-spar crystals under the influence of heat, and this is to be found in the changes of a bar of iron under the influence of magnetism. Under the influence of heat the series of crystals increases in length and loses in breadth, and there is little change of volume, but there is some change of volume, and, therefore, the illustration of the changes of muscular fibre is not perfect; but in the bar of iron the illustration is exact, for under the influence of magnetism there is increase of length and loss of breadth, without any alteration of volume. It is to Mr. Joule, of Manchester, that we are mainly indebted for the knowledge of this remarkable fact.[1]

Mr. Joule's experiments are very numerous and exact. The bar of iron was magnetized by placing

[1] 'The Philosophical Magazine,' February and April, 1857.

it in the axis of a coil of insulated copper wire, and by then passing a current of electricity through the coil. One end of the bar was fixed; the other end was attached to a system of levers by which any change in length was multiplied 3000 times. In one experiment a bar of rectangular iron wire, two feet long, one fourth of an inch broad, and one eighth of an inch thick, was placed in a coil of twenty-two inches in length and one third of an inch in diameter, and the result of the experiment was, that when the coil was traversed by the current of a battery capable of magnetizing the bar to saturation, or nearly so, the index of the multiplying apparatus sprang from its position and vibrated about a point one tenth of an inch in advance—a distance which gave $\frac{1}{30000}$th of an inch as the actual elongation of the bar. After a short interval the index ceased to vibrate, and began to advance very gradually in consequence of the expansion of the bar under the heat radiating from the coil; and this it did until the circuit was broken, when it immediately began to vibrate about a point exactly one tenth of an inch lower than that to which it had attained. On examining the end of the bar with a microscope the elongation or shortening of the bar was seen to take place with extreme suddenness. In other experiments it was found that the elongation was in the duplicate ratio of the magnetic intensity of the bar, and (for the same intensity of magnetism) that it was in direct proportion to the softness of the metal, being greatest in well-annealed iron and least in hardened steel.

In order to show that the bar of iron underwent no change of volume under these circumstances, Mr. Joule placed a bar of annealed iron, one yard long and half an inch square, in a glass tube, forty inches long and an inch and a half in diameter, around which an insulated conductor, consisting of ten copper wires, each 110 yards long and one twentieth of an inch in diameter, had been previously coiled. One extremity of this tube was sealed hermetically; the other was fitted with a stopper, which had been perforated so as to allow the insertion of a graduated capillary tube. The bulk of the iron bar was 4,500,000 times the capacity of each division of the graduated capillary tube. In performing an experiment, the tube was filled with water, the stopper adjusted, the capillary tube inserted so as to force the water to a convenient height within it, and the coil connected with a Daniell's battery of five or six cells—an apparatus of sufficient power to magnetize the iron bar to the full—and the invariable result was that no perceptible change of level was produced either on making or on breaking contact with the battery, and that equally whether the water was stationary in the capillary tube, or whether it was rising or falling from any change of temperature in the bar. The experiment, indeed, afforded most conclusive proof that the bar underwent no change of *bulk* on being magnetized, for if the elongation of the bar under these circumstances had not been accompanied by a corresponding decrease in breadth, the water would have been forced through twenty divisions of the capillary tube whenever the circuit of the battery was completed.

By other experiments Mr. Joule found that these results were greatly modified under certain circumstances. He found, indeed, that under a certain degree of *tension*, an iron wire, instead of becoming longer, actually became shorter, when magnetized; but under ordinary circumstances the results were as have been stated.

— The changes which take place in a bar of iron under the influence of magnetism are paralleled, therefore, by the changes which take place in muscular fibre, for while the fibre increases in length, and loses in breadth, under the influence of electricity and its associate agencies, and loses in length and gains in breadth when this influence is withdrawn, so likewise does the bar of iron increase in length and lose in breadth under the influence of electricity, and lose in length and gain in breadth when this influence is withdrawn. And not only is this parallelism preserved in the changes of shape and in the agencies concerned, but it is preserved in that point which is so characteristic of muscle, namely, the *suddenness* with which the contracted and elongated states may alternate upon each other,—for in Mr. Joule's experiments the bar was seen and heard and felt to *jump* into the longer form, and then to jump back again into the shorter form, as the electricity was communicated to or withdrawn from the coil.

— There are, however, certain facts which, at first sight, do not appear to be altogether consistent with this physical mode of regarding the phenomena of muscular contraction. How, for instance, can we explain that diminished degree of shortening which is noticed when a muscle contracts after death, except upon the supposition that the contractile power is a vital endowment? How is that loss of contractile power which is consequent upon death to be otherwise accounted for? How is it that the muscle loses in power as it contracts upon itself? How is it that the muscle wastes in proportion to the number of its contractions, without supposing that the contraction is the sign of functional activity in the muscle?

There is no doubt that a muscle contracts most perfectly during life, but this does not prove that the diminished degree of contraction which is noticed after death is owing to the loss of some vital power of contraction. On the contrary, there is no good reason why this diminished degree of shortening after death may not be the natural result of the peculiar circumstances in which the muscle is then placed. When a muscle contracts during life, the antagonist muscle either relaxes, or opposes no resistance to the contraction. The blood, also, is fluid, and the intra-muscular vessels are easily emptied when pressed upon by the contracting fibres. But after death the spasm is universal, and the contraction of any set of muscles is not favoured by the relaxation of the antagonist set. After death, moreover, the full degree of muscular contraction may be prevented by the coagulated contents of some of the intra-muscular vessels. At the same time it must not be forgotten, that muscle may contract to a very considerable extent after death, and that this is the case in any muscle when the antagonist muscle has been divided.

Nor is the loss of muscular strength after death a necessary proof that the contractile power of muscle is a vital endowment. On the contrary, loss of strength may, or rather must be, the natural consequence of the peculiar circumstances in which the muscle is then placed. In the first place, the fibre may be acted upon by those solvent juices which are present in muscle, and which are more or less analogous in their properties to gastric juice; and, if so, then the fibre may be partially dissolved after death, and to that extent weakened. In the second place, the dead muscle is yielded up to the processes of decomposition, and the affinities of the *muscular* molecules may be weakened by resolution of these molecules into their constituent elements; and hence another reason why the dead muscular fibre may have suffered some loss of strength,—and this, not because the contractile power of muscle is a vital endowment, but simply because this power requires for its full manifestation a physical integrity of the muscular fibre, which no longer exists.

It is true, also, that the muscle contracts with diminished power as it contracts upon itself, but this is no objection to the idea that muscular contraction is a physical phenomenon. On measuring the force with which the muscles of a frog's leg contracted at different degrees of shortening, M. Schwann found that the force decreases as the fibres become shortened; and finding this, and supposing that the force of the contraction should increase after a definite law as the fibre contracts if the contraction were due to any known physical attractive force, he inferred that muscular contraction could not be due to any such force; but he overlooked the fact, that the non-contracting or imperfectly contracting cellular substance of the muscle, and the comparatively inelastic fluids contained in the muscle, may oppose such *resistance* to the contraction of the proper muscular fibres as to mask the pure law of that contraction, and, overlooking this, his inference has not even a shadow of foundation. This experiment may indeed show the *degree of resistance* which is opposed to muscular contraction; but it is altogether worthless as an argument in support of the idea that the law of muscular contraction is essentially different from the law of physical attractive forces,—indeed elastic bodies, in shrinking after elongation, behave in every respect as muscle behaves in this experiment, and here, unquestionably, this shrinking must be a physical process.

And, lastly, it is more than doubtful whether the inference to be drawn from the fact that the waste of a muscle is proportionate to the amount of muscular action is—that muscular contraction is the sign of functional activity in the muscle. It is more than doubtful, indeed, whether this waste can be directly related to the contraction even by a vicious process of reasoning. On the other hand, it is not at all improbable that this waste may have been incurred, not in producing but in counteracting contraction, for it is certain that that electrical state which, according to the premises, is concerned in counteracting contraction, cannot be kept up without a corresponding change,—that is, waste, in the tissues concerned.

— And if these objections may be overruled, then there is no longer any sufficient reason for continuing to believe that a vital property of contractility—whether of irritability or of tonicity—has anything to do with muscular contraction. On the contrary there is every reason for discarding such an idea. For what has been the unvarying drift of the argument? It has led us to regard the elongated rather than the contracted state as the chief peculiarity of muscle, and it has seemed to show that any vital property of muscle, if such there be, must be exercised in counteracting rather than in favouring contraction. It has seemed to show that living muscle is acted upon by certain agents, electricity and others; that this action antagonizes contraction; that the transitory contractions which are said to belong to that form of contractility which is called *irritability* occur in transitory lulls of this action; and that the persistent contraction of *rigor mortis*, which is referred to that form of contractility which is called *tonicity*, is persistent because the action which antagonizes contraction has then died out. The unvarying drift of the argument, indeed, has seemed to show that a property like contractility would hinder rather than help. In a word, the only conclusion to which we have been able to arrive is one which excludes altogether from the phenomena of muscular action the idea of a vital property of contractility and the doctrine of stimulation which is founded upon it,—for this doctrine implies the existence of this property.

It is, no doubt, a difficult matter to abandon an idea which has been so long fixed in the mind as the idea that a vital property of contractility belongs to muscle, and that that property is stimulated into action when the muscle contracts. It is difficult to believe that the final purpose of a muscle—its contraction, and particularly that form of contraction which is obedient to the mandates of the will, instead of being brought about by the infusion of more life into the muscle, should be brought about by a change which is realised to its fullest extent in *rigor mortis*. At the same time the difficulty, such as it is, diminishes when it is steadily looked in the face. So far as the will is concerned, it does not follow that this principle should be ever other than an active vital power; and the only change required is to suppose that the will should bring about muscular contraction, not by imparting something to the muscle, but by suspending something which had previously antagonized contraction. The idea changes with reference to the muscle, but not with reference to the will, for in either case the will must *act*. And as to the rest, it is surely more easy to suppose that the will acts through the instrumentality of a force which belongs to muscle as a physical structure than it is to suppose that it can only act through the instrumentality of a superadded property of contractility, and a special apparatus for stimulation.

— Instead of being vital and peculiar, therefore, muscular elongation would not seem to differ essentially from that elongation which takes place in a bar of iron under the action of the electric current; instead of being vital and peculiar, muscular contraction would not seem to differ essentially from the contraction which takes place in the same bar when the action of the electric current is withdrawn; and, if so, then there is no reason why we may not accept the second proposition, which is—*that muscular elongation is due to the simple physical action of certain agents, electricity and others, and that muscular contraction is the simple physical consequence of the cessation of this action.*[1]

[1] In these remarks the attention has been confined to the movements of muscular tissue, but there is no reason why the movements of all irritable tissues, the simplest as well as the most complex, should not be obedient to the same law. There is no reason, for example, why such movements as are seen in

III. The Third Proposition.

That the special muscular movements which are concerned in carrying on the circulation—the rhythm of the heart and those movements of the vessels which are independent of the heart—are susceptible of a physical explanation when they are interpreted upon the previous view of muscular action.

1. It is difficult to read the riddle of the heart's action, but the task is not a little simplified when

Protozoa, like the Amœba, or in a common colourless corpuscle of the blood, should not be produced in the same manner as the movements of the muscles in the hand of man. Dr. du Bois-Reymond has shown that all organized structures, so far as they have been examined, are the seat of electrical action, and therefore there is no difficulty in assuming that the coats of the Amœba or of the colourless corpuscle of the blood are under the influence of electrical action, or rather are under the influence of a force which in one of its manifestations is called electricity, that elongation of certain parts may be the effect of this action, and that contraction may follow when this action is suspended. At any rate, it is as easy to make this assumption as to suppose that elongation is produced by a process of which we know nothing, and that contraction is brought about by the aid of a property of contractility through the aid of a process of stimulation of which we know less than nothing. A fact, moreover, has just been brought to light by Dr. Macdonnell, of Dublin, which seems to show that there is actually a discharge of electricity when the tentacles of an Actinia are touched by a foreign body, and in this the previous view of muscular motion is made to serve as a key.

Upon any existing theory of muscular action it is more than difficult to understand why the *ventricles* remain distended with blood during the full half of the rhythmic period, if the *ventricular systole* is in anywise called into existence by the stimulation of the blood; but this fact is not altogether unintelligible if, on the contrary, it be supposed (as must be supposed upon the previous view of muscular action) that the office of the blood will rather be to antagonise the systole and induce the diastole. Indeed upon this view the difficulty appears to be at an end, for according to it the

way we are brought to a point from which we can, as it were, look directly into those changes of form which are exhibited in the processes put out from the Amœba, or colourless corpuscle of the blood. Indeed, every Actinia passes through a rudimentary condition which differs in no essential particular from that in which the protozoon, or corpuscle is permanently fixed. The fact which has been brought to light is this—that the muscles of a frog's limb, prepared in the manner which is called the rheoscopic limb, are thrown into a state of contraction when the end of the nerve is brought into contact with one of the tentacles of the Actinia, or even when a metallic conductor is interposed in a particular manner between the tentacle and the nerve. The fact is one, indeed, which seems to show that the tentacles were charged with electrical force, and that a discharge of this force took place when they were touched, for, after what has been said elsewhere, it may be assumed that such a discharge is indicated by the contractions in the frog's limb.

ventricles are thrown into the state of diastole by the stimulation of the blood which has been injected into the coronary system of vessels, and they remain in this state until this blood has given up its arterial properties and so ceased to be stimulating. And certain it is that the different action of the ventricles in anæmia and plethora is calculated to strengthen this idea. Thus: in plethora the pulse (which is the direct test of the action of the ventricles) is full and slow; in anæmia it is small and quick. In the one case, that is to say, the ventricle fills to distension with rich blood and the systole is deferred—in the other case, the ventricle takes in a small quantity of poor unstimulating blood, and the systole follows with scarcely any delay. The facts, indeed, are the very opposites of what they would be found to be if the blood stimulated the ventricle to contract, for in that case the pulse must be small and quick in plethora, and full and slow in anæmia. But if, on the other hand, the blood provokes the ventricle to the diastole by causing elongation in the muscular fibres composing this chamber, then it is intelligible that the ventricle should dilate more fully and the dilatation continue for a longer time, when the blood is rich and warm as in plethora, than when it is poor and watery, as in anæmia.

It may also be presumed that the ventricle is not stimulated to contract by "nervous influence." At any rate, this would appear to be the inference which may be drawn from the effects of fear upon the pulse. Thus: when the nervous influence is more or less depressed from this cause the heart beats quickly and yet little blood is propelled into the vessels. The beats are perhaps doubled, and yet the skin is cold and pale. Now, under ordinary circumstances, the double number of beats would propel a double quantity of blood into the vessels, and the skin would be hot and red instead of cold and pale; and hence the inference arising out of this anomalous state of the rapid pulse and cold skin attending upon fear is that the ventricular diastole is less complete, and that on that account, a less amount of blood is pumped out of the heart than usual. In other words, the ventricle would seem to have contracted coincidentally with a withdrawal of nervous influence, for some of this influence may be supposed to be withdrawn from the system during fear.

On realising the phenomena of the heart's action more distinctly it becomes even still more improbable that the systole of the venticle is caused by any kind of stimulation, and of the blood more particularly. For what are the facts? At the systole the blood rushes through the coronary arteries into the coats of the heart, and the diastole of the ventricles is attendant upon this rush. And after the blood has remained in these coats until it may be supposed to have lost some of it arterial properties, then the systole returns. These are the simple facts; and thus if stimulation has to do with the phenomena at all it is with the diastole and not with the systole.

It appears, indeed, as if the *ventricular diastole* were due, partly to the force with which the blood is injected into the coronary arteries at the ventricular systole, and partly to the elongating, electro-motive effects of the arterial blood upon the cardiac fibres. It appears, also, as if the diastole of the ventricles were made to continue as long as the blood retained its arterial properties, and that the systole returned when the oxygen was exhausted and the arterial converted into venous blood. And thus, it appears as if the rhythm of the ventricles had a *part* of its explanation, for according to this view, so long as the proper blood continues to be supplied, and so long as the ventricle continues to be capable of responding to it, so long must the systole give rise to the diastole, and the diastole be followed by the systole.

A little further examination will also serve to show that the *systole of the auricles* must be contemporaneous with the *diastole of the ventricle,* for this *systole of the auricles,* there is reason to believe, is, in great measure, the mere *falling in* of the auricular walls upon the sudden withdrawal of blood from the auricles by the diastole of the ventricles. There is reason for this opinion in the absence of valves at the mouths of the veins opening into the auricles, and the reason is obvious. For if the auricles had to contract primarily like the ventricles, is it not fair to assume that there would have been valves to prevent the reflux of the blood from the auricles into the great veins? And if so, then there is no difficulty in accounting for the rhythm of the auricles, for the *auricular diastole,* which is virtually coincident with the ventricular diastole, will be partly due to the same cause as the ventricular diastole, namely, the rush of blood into the coronary system of arteries, and partly to the onward current of blood which is continually setting in from the veins; and the *auricular systole* will be *mainly* due to the collapse of the auricular walls upon the sudden passage of blood into the ventricles at the ventricular diastole.

— But how, it may be asked, will this explanation accord with the known fact that the heart will go on beating after it is removed from the body, and that a mere fragment of the heart will often continue to beat for some time under the same circumstances?

It is a well known fact that the heart of many animals will beat for some time after removal from the body, and that a fragment even may continue to beat regularly under these circumstances; but it is not every fragment that has this power. If, for example, as Mr. Paget reminds us in the admirable and philosophical Croonian Lecture[1] recently delivered before the Royal Society, the cut-out heart of a tortoise be divided into two pieces, the one comprising the auricles and the base of the ventricle, the other comprising the rest of the ventricles, the former piece will go on acting rhythmically, but not the latter piece. Not that the latter piece has lost its capacity for contraction, for it contracts vigorously when touched with any foreign body, but when touched in this manner it contracts once and no more, like any ordinary muscle. Or, if the ventricle of a frog's heart be separated, and all traces of the auricles removed, so that its cavity is perfectly simple; and if this ventricle, so separated, be set upright upon a board with some blood in its cavity and around it, it will be found to pulsate less and less frequently as pieces are snipped away from its upper border; and after a zone of a certain depth (nearly one third of the length of the ventricle) has been snipped away,

[1] 'On the Cause of the Rhythmic Motion of the Heart,' Proc. of R. Society, May 28th, 1857.

it will cease to pulsate altogether. It appears, indeed, as if the rhythmically acting heart may be reduced to the region which intervenes between the auricle and the ventricle, and that every part of this region had this power, for every fragment, be it ever so small, will beat regularly for some time. Again: if, in a tortoise or frog, a ligature be tied tightly around the great veins at their line of insertion into the auricle, the rhythmic action of the heart ceases either immediately or presently, and then returns in the ventricles alone: but, if the veins be tied at some distance from the auricles, the rhythm continues. In the experiment, also, where the ligature embraces the veins at their line of insertion into the auricles, it is found that there is a rhythmic motion in the veins behind the ligature, but not the same motion as that which is exhibited in the ventricles.

It is evident, then, as Mr. Paget argues, that the origin of the rhythmic motion of the heart is not in the muscular structure alone, and the inference is that certain nerves and nerve-centres are concerned in it, for the dissections of Bidder and Rosenberger have shown that the region between the auricles and ventricles, and the neighbourhood of the mouths of the great veins are especially rich in nerves. And that nerves are concerned in it would also appear in the fact that the rhythmic actions of respiration (as many experiments show) are connected, not only with the muscular apparatus, but also with the system of nerves whose centre is the medulla oblongata.

Such, then, being the anatomical conditions of the rhythm when the heart is removed from the body, or when a mere fragment is concerned, the question returns which was asked before,—how is this rhythm to be explained?

Now, according to Mr. Paget, this rhythm is due to "time-regulated discharges of nerve force in certain of the ganglia in and near the substance of the heart, by which discharges the muscular walls are excited to contraction," and these time-regulated discharges are themselves due to the *nutrition* of the ganglia and contractile tissues being rhythmic, that is, to these ganglia and contractile tissues "being, in certain periods, by nutritive changes of composition, raised, with regulated progress, to a state of instability of composition, in their decline from which they discharge nerve-force, or change their shape in contracting." Now it is more than probable that certain periodical changes in *nutrition* are concerned in producing the action of the heart, and that, without these, this action must soon come to an end; but at the same time we incline to think that changes which must be referred to *respiration* rather than to nutrition, are those which are directly concerned in the production of the rhythm. Indeed, there appears to be no manner of reason why the same principles of explanation which have been applied to the ordinary movement of the heart should not be applicable also to the movement of the heart or a fragment of the heart out of the body. In the explanation already given, the idea was that the oxygen of the arterial blood, injected by the systole of the left ventricle, and acting upon the muscular and nervous elements of the coats of the heart, produced the diastole by rousing (among other things) the muscular and nerve currents, or, in other words, by rousing the polar condition of the muscular and nervous fibres; and that the systole followed the diastole, in consequence of the failure of these currents, when the arterial blood had parted with its oxygen, and so ceased to be sufficiently stimulating. And if, under ordinary circumstances, the blood acts in this manner, then there appears to be no great difficulty in understanding how it is that the heart, or a portion of the heart, may go on pulsating after removal from the body. For why should oxygen dissolved in the blood act differently from oxygen diffused in the air? Why, for instance, should not the air, which bathes the surface and permeates every interstice, provoke a diastolic state in the separate heart or a fragment of the same, by rousing that polar condition which in the nerves and muscles is designated under the name of the nerve and muscular currents? Why may not the systolic state supervene upon this diastolic state when the polar condition fails, in consequence, as it were, of the arterial air having become converted into venous air? And why, again, should not the diastole return after every systole, so long, that is, as the muscle is capable of responding to the action of the oxygen, for it may well be supposed that the commotion of the systole will displace the venous air and bring the muscular and nervous tissues into relation with fresh quantities of arterial air? Assuredly there is no evident reason to the contrary, and there is one reason why this view should be received, and this is to be found in the fact that the rhythm is rendered more rapid, and even revived for some time after its actual cessation, by placing the heart in oxygen instead of atmospheric air, and that it is brought to a stop by placing the heart in a vacuum or by immersing it in hydrogen or nitrogen or carbonic acid.

It may be assumed, also, that the same view is no less applicable to the explanation of other kinds of rhythmic motion, for it is easy to perceive that these different forms may depend, on the one hand, on differences of structure—differences in the kind of irritable fibre or substance, in the presence or absence of nerves, in the number of blood-vessels, or in the amount of exposed surface,—and, on the other hand, upon the different rates and degrees of oxidization which are consequent upon these differences of structure.

— According to this view, therefore, there need be no difficulty in explaining the rhythm of the heart in accordance with the previous view of muscular action, and the fact that the heart, or a portion of the heart, will go on pulsating for some time after removal from the body, instead of being an objection to this view, may even be an additional argument in its favour.

2. Nor is it easy to understand how it is that the circulation is carried on, if the vessels contract under the influence of certain stimuli acting upon them. If the blood stimulates the vessels to contract, there seems to be only one conclusion, and this is, that the contraction must impede the entrance of blood into the vessels; and so likewise with every other stimulus. But if the blood antagonizes contraction, this difficulty is at an end, and a key may be obtained to the interpretation of those mysterious movements of the blood which are independent of the action of the heart. And this view, which is deducible from the premises, is also warranted by the facts which have still to be mentioned. When the blood is rich and stimulating, as in plethora,

the vessels are red and full; when the blood is poor and watery, as in anæmia, the vessels are shrunk and empty. When the "nervous energy" is exuberant, as in joyous excitement, the skin is flushed; when the nervous energy is depressed, as during fear, the skin is pale. When the hand is held to the fire it flushes with blood; when exposed to cold it becomes blanched and bloodless. The facts, indeed, are utterly inconsistent with the idea that the muscular coats of the vessels are stimulated to contract by blood, or nervous influence, or heat,—and these facts are only examples of many others which might be adduced; but they are not inconsistent with the idea that the contracted state of the vessels is antagonized by the action of these so-called "stimuli."

Now, it follows from the premises that elongation of the vascular muscular fibres and consequent expansion of the vessel itself, may result from the admission of arterial blood into a vessel, and that in this way the admission of blood into a vessel may lead to the admission of more blood, until the vascular fibres have elongated to the full extent required by the amount of oxygen contained in the blood; and, if so, then it may be supposed that the blood will have the power of making for itself a way into and through the vessels—a power which will be in direct proportion to its arterial properties.

And certainly this view is in accordance with the facts which have been mentioned as well as with the facts which remain to be noticed. Indeed, it will afford the only explanation of such purely vascular movements as are exemplified in *"determination of blood"* and in those *ever-shifting-to-and-fro-movements* which are seen in the vascular area of animals before the formation of a heart, or in the laticiferous vessels of plants.

When the hand is held to the fire the heat will produce expansion of the vessels, partly by rousing the polar action of the vascular fibres, and partly by its direct expansive influence upon the areolar and other simple textures; and this expansion will necessitate *"determination of blood"* to the part. It is supposable, also, that the additional quantity of blood which is thus received into the expanded vessels will rouse the nerves of the part into fuller action, and that this fuller action, reacting upon the nervous centres, will lead to further expansion of the vessels, and further admission of blood into them, by augmenting, so to speak, the polar tension of the vascular coats. And hence, reasoning in this manner, "determination of blood" to the part is the necessary consequence of holding the hand to the fire.

Nor does the same principle of interpretation fail when applied to the *ever-shifting-to-and-fro move-*

ments of the "vascular area" of animals before the formation of the heart, or of the laticiferous vessels of plants. Now these fugitive centres of fluctuating movement occur in a web of vessels of unequal sizes; and it is this fact which appears to be concerned in the explanation of the difficulty. Assuming, indeed, that the reaction between the vessels and their contents will be proportionate to the extent of the coats and the quantity of the contents, and that for this reason the larger vessels will experience a greater degree of expansion than the smaller,—it follows that the largest vessels will tend to become still larger, and that they will go on expanding and filling until a limit is reached in which the elongating fibres of the vessels are elongated to the full extent of which they are physically capable under the "stimulus" supplied; and that after this the vessels next in size, because next in size, will expand and fill (partly at the expense of the fluid contained in the vessels which have already filled to their full extent, and partly at the expense of the fluid contained in the emptier vessels) until they can expand and fill no more. It follows, indeed, that the scene of expansion and filling will continually change to the vessels next in size, when the vessels already expanding are full, and that this transference of action will continue as long as the field of action consists of vessels of unequal sizes. As long as the field of action consists of vessels of unequal sizes,—for it is manifest that there could be none of those inequalities of action which, according to the hypothesis, are concerned in the production of these fugitive hearts if the vessels of the web were of the same size in every part.

— In this way, then, it appears to be possible to explain those special and peculiar muscular actions which are concerned in carrying on the circulation—the rhythm of the heart, and those movements of the vessels which are independent of the heart—and as this way is that which naturally arises out of the premises, this very fact may be taken as an additional argument in favour of the correctness of that view of muscular action which is set forth in the two previous sections of this inquiry.