

Vital motion as a mode of physical motion.

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VITAL MOTION

AS A MODE OF PHYSICAL MOTION.

BY

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NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC; &c.

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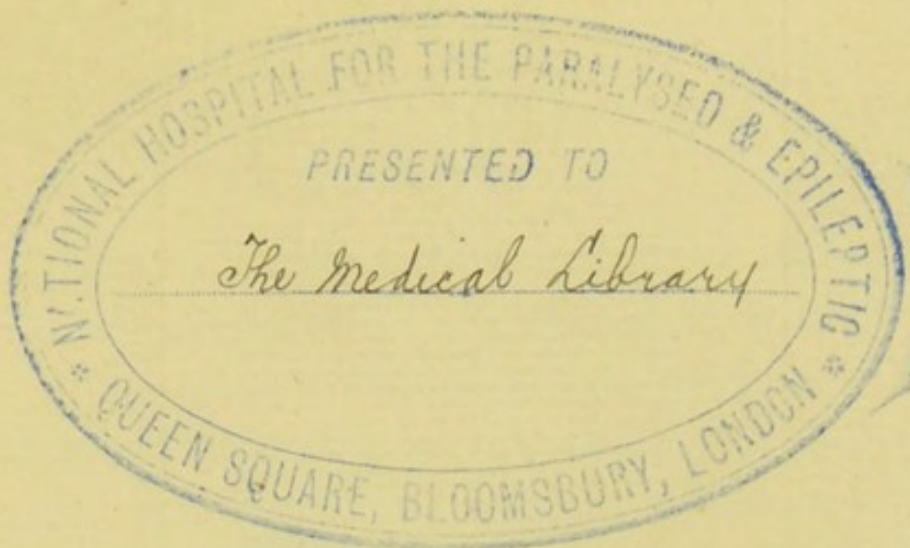
VITAL MOTION

AS A MODE OF PHYSICAL MOTION

LONDON:

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TO MY FRIEND

C. E. BROWN-SEQUARD, M.D.,

LAUREATE OF THE INSTITUTE OF FRANCE,

FELLOW OF THE ROYAL SOCIETY OF LONDON,

PROFESSOR OF MEDICINE IN THE COLLEGE DE FRANCE

&c., &c., &c.,

WHOSE DISCOVERIES

IN PHYSIOLOGY AND PATHOLOGY

ENTITLE HIM TO THE VERY FOREMOST PLACE AMONG THE

PHYSIOLOGISTS AND PHYSICIANS OF THE

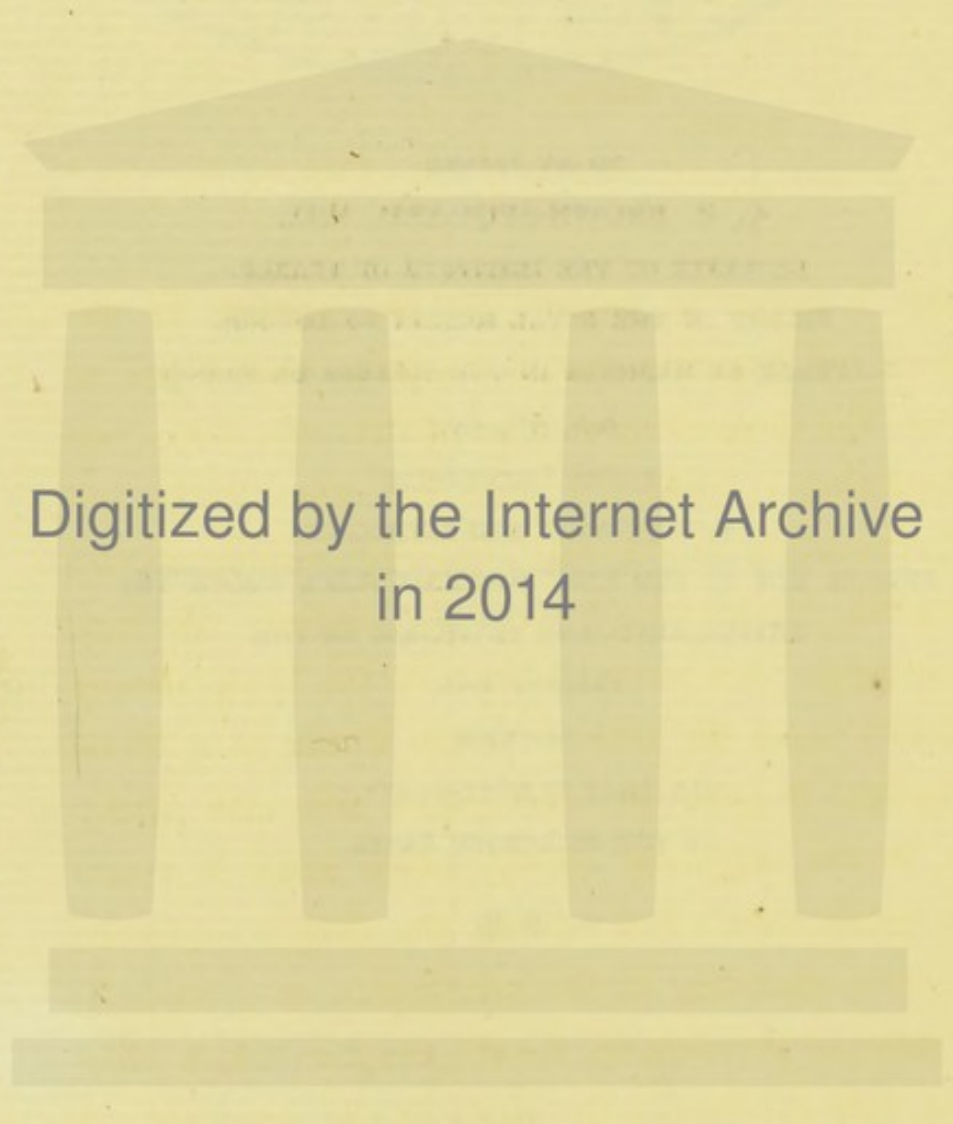
PRESENT DAY,

I INSCRIBE

ALL THAT IS NOTE-WORTHY

IN THE FOLLOWING PAGES.

C. B. R.



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Aliusque et idem
Nasceris.

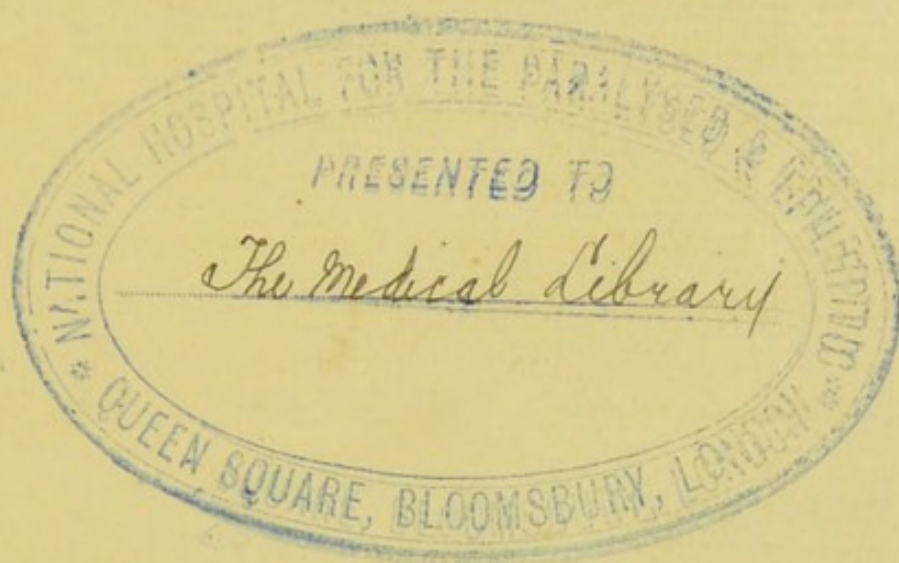
Hor. Carm. Sec. 10.

WHEN I began to prepare for the printers in this instance my intention was to compress what I had to say into the dimensions of a pamphlet. As I went on I found that I could not state my case clearly and comprehensively in this way, and that nothing shorter than a thin volume would serve my purpose. As it is, I have prepared a second edition of the book already published under the title of "Vital Motion as a Mode of Physical Motion," in fewer pages, and with alterations, in the argument chiefly, which make it a new book rather than a new edition, the most conspicuous alterations consisting in the substitution of a consecutive series of propositions for the chapters which often served only to conceal the steps of the argument. I might have said much more, and perhaps I ought to have done so: and if I have erred on the side of conciseness I must plead as an excuse the motive

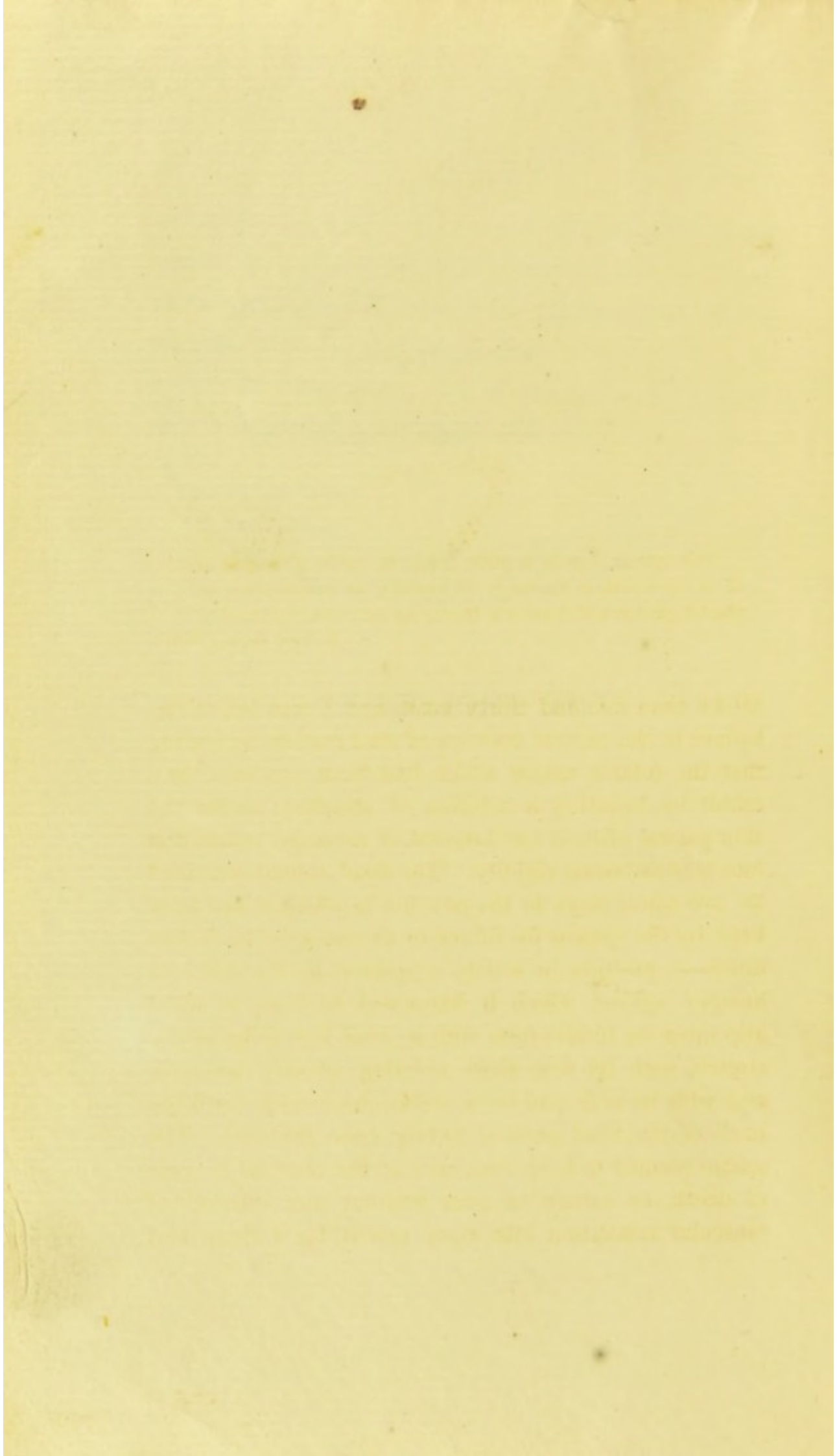
which led me first of all to think of a pamphlet as the vehicle for my thoughts, which was the wish to please those who—naturally enough in these days of books without end—are apt to shudder at the sight of a book with many pages, and, if they have ventured to take it up, to put it down un-read.

25, Cavendish Square,

June, 1882.



THE PROLOGUE.



“Le nom de *Galvani* ne périra point ; les siècles futurs profiteront de sa découverte, et comme le dit Brandes, ‘ils reconnaîtront que la physiologie doit à *Galvani* et à *Harvey* ses deux bases principales.’ ”

F. VON HUMBOLDT.

MORE than five and thirty years ago I was led to disbelieve in the current doctrine of vital motion by finding that the tetanic spasm which had been produced in a rabbit by injecting a solution of strychnia under the skin passed without any interval of muscular relaxation into true cadaveric rigidity. The dead animal remained for two whole days in the position in which it had been kept by the spasms for fifteen or twenty minutes before death—a position in which, supported by the side of a hamper against which it happened to lean, it stood atip upon its hinder toes, with its hind legs fully on the stretch, with its fore paws pointing directly upwards, and with its neck and body arched backwards until the back of the head pressed tightly upon the scut. The spasm seemed to keep firm hold of the muscles in spite of death, or rather to pass without any interval of muscular relaxation into rigor mortis, for if there had

been any such interval at death the body must then have collapsed and fallen down as it did do when the time came for cadaveric rigidity to give place to putrefactive limpness. At first I was much perplexed. Afterwards, by slow degrees, I became convinced—that a radical change was necessary in the doctrine of vital motion,—that the interpretation of spasm was to be sought, not on the side of life, but on that of death,—that spasm and rigor mortis were to be regarded, not as signs of vital action in certain vital properties of contractility, but as physical phenomena akin to, if not identical with, the return of an elastic body from a previous state of extension,—that muscular contraction in all its forms might be the simple consequence of the operation of the natural attractive force or forces inherent in the physical constitution of the muscular molecules,—that life is concerned in antagonizing contraction rather than in causing it,—that this antagonizing influence might be electrical in its nature,—that, in short, vital motion might have to be regarded as a mode of physical motion.

And yet more did this conviction grow in strength on the food supplied by two other facts to which my attention was called at a later period.

Of these two facts the first was brought to light in an epileptic patient in whom it had been thought expedient to try and cut short a succession of very violent convulsions by taking blood from the temporal artery. The artery was divided when the fit was at his height, and the blood escaped by jets in the usual way, but not of the usual colour. Instead of being *red*, the blood

was *black*: instead of being *arterial*, that is to say, it was *venous*. The state during the convulsion was evidently that of suffocation; and, for this reason, black, unaërated blood had found its way into the arteries, and was being driven through them at the time. The case was intelligible enough as regards the suffocation, for in this state the simple fact is, that black blood does for a time penetrate into and pass along the arteries; but it was not intelligible as regards convulsion, if convulsion were, as it is assumed to be, a sign of exalted vital action. I could connect such exaltation with increased supply of *red* blood to certain nerve-centres, but not with the utterly contrary state of things involved in the actual circulation of *black* blood; and, do what I would, I could see no other conclusion than that which had been already forced upon me by the history of the poisoned rabbit, namely this, that the convulsion pointed to a state of things which had to do with death rather than with life,—that, in short, this state of muscular contraction was due, not to the *black* blood having acted as a stimulus, but to the withdrawal of an *inhibitory* influence which had served to keep up the state of muscular relaxation as long as certain nerve-centres were duly supplied with *red* blood.

And so likewise with the second of the two facts to which I have alluded. I had the good fortune to be present on one occasion when Matteucci was watching the action of strychnia upon the common electric ray of the Mediterranean. I saw very plainly that this action was marked by involuntary electric shocks as well as by involuntary spasms, and I was much struck by

what was said by this excellent physiologist in support of the notion that muscular contraction was attended by a discharge analogous to that of the torpedo, and that there was much in common between the action of the electric organ and the action of the muscles: and, seeing and hearing all this, I could not help wondering whether muscular relaxation might not be the consequence of the muscular molecules being kept in a state of mutual repulsion by the presence of an electrical charge, and whether the discharge of this charge might not bring about muscular contraction by allowing the attractive force or forces inherent in the physical constitution of the muscular molecules to come into play. I could, indeed, bring myself to adopt no other conclusion than this: and thus it was that this experiment upon the torpedo proved to be the means of adding not a little strength and definitiveness to the conviction at which I had already arrived respecting vital motion.

Looking back I can now see plainly enough that there are not a few faults and shortcomings in the argument by which hitherto I have striven to bring others to the same way of thinking with myself in this matter. About the first published statement* of this argument I may say, in the words of Dryden, that it was "only a confused mass of thoughts tumbling over one another in the dark, when the fancy was yet in its first work, moving the sleeping images of things towards the light, there to be distinguished, and then either chosen or rejected by the judgment:" and, most certainly,

* "Philosophy of Vital Motion." 8vo. Churchill, 1851.

no feeling of complacency is called up by the remembrance of any other statement published subsequently*,†,‡,§. I should, in fact, be very glad if much that I have written on this subject at different times could be cancelled. What has been done, however, has been done, and, after all, if it were still to do the argument in all essential particulars would be the same. Indeed, except in being clearer and far more comprehensive, the view now taken is substantially that I have always taken; and this is all that I would now say about it except this—that I have thought it expedient to prolong these introductory remarks so as to bring in certain points in the history of vital motion which can scarcely fail to be of service in preparing the way for the just appreciation of the more serious work with which I propose to occupy myself presently.

Very misty notions about vital motion prevailed in olden times, and most assuredly, some of this mistiness still clings to the notions which are now in the ascendant.

In the beginning, as it would seem, all motion was believed to be essentially vital.

Thales talked about the world as being animated by

* "Epileptic and other Convulsive Affections of the Nervous System." (Incorporating the Gulstonian Lectures for 1860.) 3rd edition. Post 8vo. Churchill, 1861.

† "Lectures on certain Diseases of the Nervous System." Delivered at the Royal College of Physicians. Post 8vo. Churchill, 1864.

‡ "Dynamics of Nerve and Muscle." Post 8vo. Macmillan & Co., 1871.

§ "Vital Motion as a mode of Physical Motion." 8vo. Macmillan & Co., 1876.

a soul and actuated by demons, and looked upon motion as being due to the working of one or other of these two causes.

Hippocrates believed in the universal presence of a living, intelligent, active principle, to which he gave the name of *nature* (φύσις), and to him, as to many in the present day, it was enough to refer motion to *nature*—to regard it as *natural*. The power of motion, indeed, was one of the faculties with which the principle of nature was endowed.

Plato says little to the point. With him all philosophy merged in theology: to him vital motion, and motion generally, when traced to its source, resolved itself into a display of divine power.

Aristotle, the great contemporary of Plato, recognized, not a divine being as Plato did, but a *First Moving Cause*, a *primum mobile*, one in essence, eternal, immaterial, at once immoveable, and the spring of all movement. According to him, this First Moving Cause worked in the living body (ζῶον) through the instrumentality of a principle which was distinctive of this body, and to which he gave the name of soul (ψυχή)—a principle possessing various energies or faculties of its own, distinct from the organs in which it was manifested, and yet requiring these organs for its manifestations. To this soul, when most developed, belonged several faculties (δυνάμεις)—the faculty of receiving nourishment (δύναμις θρεπτική), the faculty of sensation (δ. αἰσθητική), the faculty of motion in place (δ. κινητική), the faculty of impulse or desire (δ. ὁρετική), the faculty of intelligence (δ. διανοητική). Vegetables even, by having the lowest of

these faculties, the threptic, were supposed to have souls. Moreover, it is hinted that the seat of this kinetic faculty in animals is in the muscles, and that—a conjecture for which Praxagoras, who lived two hundred years previously, ought to have credit—there were nerves, some of which had to do with movement and others with sensation. Nay, it is scarcely just to speak of the localization of the kinetic faculty in the muscles as being only hinted at, for this was the definite conclusion at which Aristotle arrived after witnessing the working of the intercostal muscles of a living chameleon as displayed under the transparent pleura.

After this time, for a thousand years and more, when anything was done in this direction it was little beyond a servile copying of what had been said by Hippocrates and Aristotle. Even Galen had nothing to say that was really new; nor yet the schoolmen of the middle ages, with many of whom the notions chiefly in the ascendant were those of alchemy and magic and astrology. At the revival of letters, indeed, the only light of importance was that derived from the writings of the old Greek fathers in science; and what was done tended rather to deepen the darkness than to dissipate it. No new light, for instance, was shed by the doctrine of occult causes which found most favour in these times, for this doctrine was no more than a copy of the doctrine of Hippocrates or Aristotle, that various bodies had various powers by which they were able to act in the various ways *natural* to them. And still less did new light radiate from the notion, which in too many instances was associated more or less closely with that

of *occult causes*—that there were elementary spirits, intermediate between material and immaterial beings, in the four elements of air, water, fire, and earth—sylvans or fairies in the air, nymphs and undines in the water, salamanders in the fire, gnomes, trolls, pigmies, spirits of the mine, little folks, cobolds, in the earth,—that the body had its double or dæmon, called Archæus, whose primary function was to superintend the work of the stomach, and who managed the various functions of the body, that of motion included, through the instrumentality of a legion of underling deputies undignified by any distinctive names.

Indeed, it was not until Von Helmont, Stahl, and Hoffman appeared on the scene that the notions handed down from the ancients began to be materially modified, and to take the forms belonging to modern times.

With Paracelsus, Von Helmont held that the Archæus and its underlings were the agents in all vital manifestations, and he also thought for himself a little, for to him belongs the credit, if credit it be, of being the first to maintain that the living body had powers of a specific character altogether different from those belonging to inanimate nature.

Accepting the doctrine that there was one law for animate and another for inanimate nature, Stahl went further, and maintained that matter is essentially and necessarily passive and inert, and that all its active properties or powers are derived from a specific and immaterial animating principle imparted to it—a principle to which he gave the name of *anima*. The body, he held, as body, has no power to move itself. All vital

motion is the result of animation. The physical powers of matter, which have only free play after death, are in every way opposed to, and counteracted by, the *anima*, of which he further says, as the followers of Hippocrates said of *nature*, that "it does without teaching and without consideration what it ought to do;"—a remark which makes it evident that the *anima* of Stahl is not to be confounded with the conscious personal Archæus of Paracelsus and Von Helmont.

What Stahl explained in this way, Hoffman, who took the next noticeable step in advance, explained on the hypothesis of *nervous influence*, or *nerve-fluid*, whatever that may mean. By this influence or fluid, according to him, the moving fibres have a certain power of action, or tone, which may be increased or diminished. If increased unduly, spasm is the result: if decreased, atony.

Next in order have to be named Glisson, Haller, and *the* Brown, known as the author of the Brunonian system of medicine, men whose speculations form the basis of the doctrine of vital movement now in favour.

Glisson, an eminent professor at Cambridge in his day, was the first to advance the present doctrine of *muscular irritability*. He asserted that there was in muscle a specific vital property, to which he gave this name, and that contraction was due to this property being in some way put in action.

Haller expanded this idea, and drew for the first time a line of distinction between the special vital property of muscle and the special vital property of nerve. He retained the name of irritability for this property in muscle: he gave the name of sensibility to this property

in nerve. Each property was something vital, something departing at death, something in no wise akin to any power in inanimate nature. The property was a *life* of which muscular contraction and nervation were *acts*.

Brown, starting from this point, added another idea—that of *stimulation*. Everything acting upon the vital property of irritability or sensibility (to which he gave the common name of *excitability*), according to him, acted as an excitant or stimulus. Action is caused by a process of stirring-up, as it were, the capacity for acting being asleep, or at rest, until it is so stirred-up. The idea would seem to be none other than that all vital movement in its nature is identical with that which is produced by teasing a sleeping man until he wakes up and strikes about him in fun or in anger.

And this doctrine of vital motion, which thus took form in the speculations of Glisson, Haller, and Brown, is, with little change, the doctrine at present in favour.

In point of fact, the position taken at present has but little shifted since the days of the schoolmen, when *occult qualities* of one kind or another were thought to be a sufficient explanation for everything—when, for example, terreity, aqueity, and sulphureity, the occult qualities of the three elements, earth, water, and sulphur, of which, in varying proportions, according to Paracelsus, all bodies are composed, were supposed to account for all that was general in these bodies,—when Petreity was thought to be a sufficient explanation of the peculiarities distinguishing Peter from Paul or other men,—when the answer of Argan* to the question, “*quare opium facit*

* Molière, “*La Malade Imaginaire* :” 3ième intermède.

dormire," in the mock examination for the diploma of physician, would have been listened to without a smile if it had been given in sober earnest before the examiners of a real faculty of medicine :—

Mihi a docto doctore
 Demandatur causam et rationem *quare*
Opium facit dormire.
 Et ego respondeo
Quia est in eo
Virtus dormitiva
 Cujus est natura
 Sensus assoupire.

For in referring vital motion to a property of irritability what more is done than to say, that the moving body moves because there is in it an occult quality which is suspiciously akin to terreity, aqueity, or sulphureity, or to Petreity, or to the "virtus dormitiva" of opium in the comedy? "To tell us," as Newton said, "that every species of thing is endowed with an occult specific quality, is to tell us *nothing*." Even to say that the phenomenon is *vital*, is, as Whewell remarks, "very prejudicial to the progress of knowledge by stopping enquiry *by a mere word*." Moreover, the very assumption upon which the doctrine in question is based—that vital motion is altogether distinct from physical motion—is itself not altogether satisfactory. "At the best," as Coleridge says,* "it can only be regarded as a hasty

* "Hints towards the Formation of a more Comprehensive Theory of Life." By S. T. Coleridge. Ed. by Dr. Seth B. Watson. Churchill, 1848.

deduction from the first superficial notions of the objects that surround us, sufficient perhaps for the purpose of ordinary discrimination, but far too indeterminate and diffuent to be taken unexamined by the philosophic enquirer. * * * * By a comprisal of the *petitio principii* with the *argumentum in circulo*—in plain English, by an easy logic which begins by begging the question, and then, moving in a circle, comes round to the point where it begins—each of the two divisions has been made to define the other by a mere re-assertion of their assumed contrariety. The physiologist has luminously explained $y + x$ by informing us that it was a somewhat that is the antithesis of $y - x$, and if we ask what then is $y - x$, the answer is, the antithesis of $y + x$;—a reciprocation that may remind us of the three sisters in the fable of the Lamiaë, with one eye between them which each borrowed from the other as she happened to want it, but with this additional disadvantage, that in the present case it is, after all, but an eye of glass.”

But this glance at the history of vital motion is not yet ended. Up to the time of Von Helmont, the idea of a well-defined gulf between animate and inanimate nature was not clearly defined: nor yet after this time did this idea gain universal acceptance.

At the time of Paracelsus the facts of chemistry began to occupy a large share of the attention of philosophers, and soon afterwards a school, called the iatro-chemical school, propounded various physiological doctrines founded upon chemistry. The opposition of acid and alkali, and the workings of ferments of one kind or another, were supposed to supply the solution

of many problems in vitality. Then came the hope, kindled naturally by the splendid discoveries of Galileo and Newton in physical science, that the mechanical principles of the macrocosm would supply the key to all requiring interpretation in the microcosm—a hope which called into existence the so-called iatro-mathematical or mechanical physiologists. The question was of the cohesion, the attraction, the resistance, the gravity, which operate in inert matter, and of mechanical impulse and elasticity, not of powers of a higher order. It was believed that all the various bodily functions were problems to be solved, as, in the main, so many hydraulic or hydrostatic problems, partly by gravitation and the laws of motion, and partly by chemistry, which itself, as far as its theory was concerned, was but a branch of mechanics, working exclusively by imaginary wedges, angles, and spheres. The restoration of ancient geometry, aided by the modern invention of algebra, had placed the science of mechanism on the philosophical throne. It was thus, for example, that Borelli dealt with the problem of muscular motion, and after him Bellini.

As far back also as the time of the great Bacon, Gilbert had struck out a new path in the same direction, the following out of which has led to more satisfactory results than any of those arrived at by the iatro-mathematical school in their own particular lines of inquiry. He had investigated the phenomena of magnetism with much success, and by continually poring over this subject had come to believe that magnetism supplied the key to vital movement, and to vital and physical pro-

blems in general ; but his speculations bore little or no fruit, and are chiefly of interest as being the first step towards a discovery which marks the birth of a new epoch in the philosophy of vital motion, and on which it may be well to dwell for a moment or two. This discovery originated in a house—to which I have made more than one pilgrimage—in the Via Ugo Bassi, già Strada Felice, at Bologna, and is commemorated on a marble slab over the doorway in these words :—

LUIGI GALVANI
 in questa casa
 di sua temporaria dimora
 al primi di settembre
 dell' anno MDCCLXXXVI
 scoperse dalle morte rane
 LA ELETTRICITA ANIMALE.
*Fonte di maraviglie
 a tutti secoli.*

Experimenting with an ordinary electrical machine at no great distance from a plate on which lay several pairs of frogs' legs prepared for cooking, and noticing that these legs jumped whenever a spark was drawn from the prime conductor, it occurred to Galvani that the parts which had been intended simply as a dish for dinner might be made to do good service as electrosopes in some experiments on atmospheric electricity in which he was then engaged. He expected that the limbs which had jumped in obedience to discharges of franklinic electricity would also jump in obedience to discharges

of atmospheric electricity; and in order to see whether they would do so or not, he, and his nephew Camillo Galvani, who was then acting as his assistant, lost no time in transferring them from the kitchen, where they had been seen to jump in this way, to a belvidere on the roof of the house, which served the purpose of an electrical observatory, and in suspending them by means of pieces of iron wire bent hookwise—one of which was made to transfix each pair at the spinal juncture—upon an iron stay which, at a short distance above the parapet, still passes horizontally across each of the arched openings with which three out of the four sides of the belvidere are pierced. Describing what happened, Galvani says, “Ranas itaque consueto more paratas uncino ferreo earum spinali medulla perforata atque appensa septembris initio (1786) die vesperscente supra parapetto horizontaliter collocavimus. Uncinus ferream laminam tangebatur: en motus in rana spontanei, varii, haud infrequentes. Si digito uncinulum adversus ferream superficiem premeretur, quiescentes excitabantur et toties ferme quoties hujusmodi pressio adhiberetur.”* In this place, and in this way, was the discovery made which is commemorated on the slab in the front of the house as the wellspring of wonders for all ages, “fonte di maraviglie a tutti secoli,” and of which, a short time before the close of the last century, the illustrious author of *Cosmos*, then in his prime, wrote, “le nom de Galvani ne périra point; les siècles futurs profiteront de sa découverte, et comme le dit Brandes, ‘ils reconnaîtront que la physiologie doit à Galvani et à

* “De Viribus Electricitatis in motu musculari Commentarius,” 1791.

Harvey ses deux bases principales.'"* At this time, then, and in this place, Galvani saw the contractions he describes, and discovered, or rather divined, in them the existence of animal electricity. How, he asked himself were these contractions to be accounted for? They could not be due to discharges of atmospheric electricity, for the sky at the time presented no indications of electric disturbance: they could not be due to the discharges which gave rise to them within the house, for the electric machine, which remained behind, was not then in action: they could not be due, that is to say, to discharges of either of the two kinds of electricity then known: and having arrived at this point, he jumped from it to the conclusion, that the limbs themselves must have an electricity of their own, and that the contractions were brought about by discharges of this electricity. It never occurred to him to doubt that electricity was the agent at work in causing these contractions: and, in short, he did not hesitate to conclude, not only that the contractions were in themselves abundant proof of the existence of animal electricity, but also that the muscular fibres are charged during rest as Leyden jars are charged, and that muscular contraction is the sign and effect of the discharge of this charge, the discharge, in one way or another, being brought about by an electrical action of the nerves upon the muscles.

From this time until the day of his death, Galvani went on performing experiment after experiment, sacri-

* *Expériences sur le galvanisme, et en général sur l'irritation des fibres musculaires et nerveuses*: F. A. Humboldt. Traduit par J. F. N. Jadelot. 8vo. Paris, 1799, p. 361.

ficing hecatombs of frogs, and never wavering in his belief in the existence of animal electricity, or in the conclusion he had come to respecting the action of this electricity in vital motion: but during his lifetime his hopes to bring others to the same mind with himself were in the main foiled, and that too by a weapon which lay hid in one of his own experiments. The experiment in question was one in which a galvanoscopic frog* was thrown into a state of momentary contraction by placing a conducting arc, of which one-half was silver and the other half copper, between the lumbar nerves and the crural muscles. Galvani, as was his wont, explained these contractions by supposing that the conducting arc had served to discharge animal electricity, and that the contractions were the result of the discharge. His colleague Volta, on the other hand, was of opinion that the electricity producing these contractions originated in certain reactions between the silver and copper portions of the conducting arc; and he was not shaken in this view by what he did afterwards, for, wishing to confirm it, he began a series of investigations which ended in the discovery of the voltaic pile and battery—a discovery which filled all minds with wonder, and for a long time afterwards diverted attention altogether from the consideration of the claims of animal electricity. In the meantime, however, while Volta was demonstrating the existence of that electricity which originates in the reaction of heterogeneous bodies, and

* The *galvanoscopic frog* is prepared from the hinder half of the animal, by stripping off the skin, and snipping away all the parts between the thighs and the fragment of the spine except the principal nerves.

which is now known as voltaic electricity, Galvani continued his search after animal electricity, and made many important discoveries as he went along. He discovered, among other things, that a galvanoscopic frog would contract without the help of a conducting arc composed of heterogeneous metals. He discovered, not only that these contractions would happen when this arc was composed of a single metal, but also that an arc composed of muscle or nerve would answer the same purpose as the metallic arc. He also discovered that the limb of a galvanoscopic frog, of which the nerve had been divided high up in the loins, would contract at the moment when the end of the nerve below the line of section was brought down and made to touch a part of the trunk of the same nerve. At last, indeed, he hit upon an experiment in which he seemed to have to do with an electricity other than that arising from the reaction of heterogeneous bodies—an electricity which must belong to the animal tissues themselves. He did much, but he did not do enough to win the battle in which he was engaged, for Volta still kept his position, denying the existence of animal electricity, and maintaining that the electricity which produced the contractions in the galvanoscopic frog was always due to electricity arising in the reaction of heterogeneous bodies of one kind or other—silver and copper, metal and organic tissue, muscle and nerve, nerve in one state with nerve in another, as the case might be.*

In 1799, Humboldt took up the question at issue

* *Ann. de Chim.*, T. xxiii, pp. 276 and 301.

between Galvani and Volta, and published a work* in which he shows by many new and curious experiments that there was error on both sides—that Volta was wrong in ignoring altogether the influence of animal electricity in Galvani's experiments, and that Galvani was not less wrong in recognising nothing but this influence. He, himself, as is proved in the extract already given, was a firm believer in animal electricity; but he failed to supply reasons for this belief which can be regarded as thoroughly satisfactory at the present day. Still, he did something in this direction by making out—first, that the agent assumed to exist, and to be animal electricity, has this in common with electricity, that its action is permitted by conductors and prevented by non-conductors; and, secondly, that it is not to be confounded with voltaic electricity, because the action, which is permitted by conductors, is possible across a gap in the circuit which would allow the passage of franklinic electricity, but which would altogether prevent that of voltaic electricity—would allow, that is to say, electricity of high tension to pass, but not electricity of low tension. What Humboldt did, in fact, was to increase the probabilities of the existence of animal electricity not a little, and at the same time to make it appear that this electricity would prove to be of higher tension than voltaic electricity under ordinary circumstances.

In 1803, Aldini, Galvani's nephew,† published an

* *Op. cit.*

† "Account of the late Improvements in Galvanism, with a series of curious and interesting experiments performed before the Commissioners of

account of certain experiments which furnish further evidence in favour of the existence of animal electricity, by showing that living animal tissues are capable of giving rise to attractions and repulsions which seem to be no other than electrical attractions and repulsions. "I held," he says, "the muscles of a prepared frog in one of my hands, moistened with salt and water, and brought a finger of the other hand, well moistened in the same way, near to the crural nerves. When the frog possessed a great deal of vitality, the crural nerves gradually approached my hand, and strong contractions took place at the moment of contact." And again:—"Being desirous to render this phenomenon more evident, I formed the arc by applying one of my hands to the spinal marrow of a warm-blooded animal, while I held the frog in such a manner that its crural nerves were brought very near to the abdominal muscles. By this arrangement the attraction of the nerves of the frog became very evident."

About this time, however, the discovery of the voltaic battery had given the victory to the opinions of Volta—a victory so complete that nothing more was heard about animal electricity for the next thirty years.

In 1827, Nobili* brought back the subject of animal electricity to the thoughts of physiologists by discovering an electric current in the frog. He made this discovery by means of the very sensitive galvanometer

the French National Institute, and repeated in the Anatomical Theatres of London, &c." 4to. London, 1803.

* Bibl. Univ., 1828, T. xxxvii, p. 10.

which he himself had invented a short time previously—an instrument which, as perfected by Professor Du Bois-Reymond and others, by Sir William Thomson more especially, ought to be as prominent an object as the microscope in every physiological laboratory. Immersing each end of the coil of the instrument in a vessel containing either simple water or brine, and completing the circuit between the two vessels with a galvanoscopic frog—the fragment of the spine being immersed in one vessel, and the paws in the other—he found that there was a current in the frog from the feet upwards, which current would cause a considerable permanent deflection of the needle—to 30° or more, if brine were used, to 10° , or thereabouts, if water were put in place of the brine. Nobili supposed that this current was peculiar to the frog, and in this he erred; but he did, nevertheless, a great thing, for, by this experiment, he furnished the first unequivocal proof of the real existence of animal electricity.

Twelve or thirteen years later, Matteucci published an essay* which, as M. De la Rive says,† “restored to animal electricity the place which it ought to occupy in electrical and physiological phenomena.” This essay, moreover, had a great indirect influence upon the fortunes of animal electricity, for M. Du Bois-Reymond, as he himself tells us, was led to undertake the investigations which have made his name famous in this depart-

* *Traité des Phénomènes Electro-physiologiques des Animaux.* Paris. 1844.

† *A Treatise on Electricity, in Theory and Practice.* Translated by C. V. Walker. 8vo. Longman. 1853-1858.

ment of physiology by the inspiration arising from its perusal.

The joint labours of MM. Matteucci and Du Bois-Reymond have left no room for entertaining any doubt as to the reality of animal electricity. Matteucci has demonstrated in the most unequivocal manner that a current is generated in living muscle which is capable of decomposing iodide of potassium, of giving "*signes de tension avec un condensateur délicat*,"* and of producing movement in the needle of the galvanometer; and not only so, but also—a fact, the discovery of which will always give Matteucci a place in the very foremost rank of physiological discoverers—that muscular contraction is accompanied by an electrical discharge analogous to that of the torpedo. And as for M. Du Bois-Reymond,† it may be said that he has demonstrated most conclusively that there are electrical currents in nerve—in brain, spinal cord, and other great nerve-centres, in sensory, motor, and mixed nerves, in the minutest fragment as well as in masses of considerable size,—that the electrical current of muscle, which had been already discovered by Matteucci, may be traced from the entire muscle to the single primitive fasciculus,—that Nobili's "frog-current," instead of being peculiar to the frog, is nothing more than the out-flowing of the currents from the muscles and nerves,—that the law of the current of the muscle in the frog is the same as that of the current of the muscle in man, rabbits, guinea-pigs, and mice, in pigeons and sparrows, in

* "*Cours d'Electro-Physiologie*." Paris. 1858.

† "*Untersuchungen über thierische Electricität*." Berlin. 1849, 1853.

tortoises, lizards, adders, toads, tadpoles, and salamanders, in tench, in freshwater crabs, in glow-worms, in earth-worms—in creatures belonging to almost every department of the animal kingdom,—that the law of the current in muscle agrees in every particular with the law of the current in nerve, and also with that of the feeble currents which are met with in tendon and some other living tissues,—and that there are sundry changes in the current of muscle and nerve under certain circumstances, as during muscular contraction, during nervous action, under the influence of continuous and interrupted voltaic currents, and so on, which changes, as I shall hope to show in the sequel, are of fundamental importance in clearing up much that would otherwise be impenetrable darkness in the physiology of muscular action and sensation.

Before the discovery of the galvanometer the attention of those who cared to meddle in these matters was directed exclusively to the static phenomena of animal electricity. Then the only definite electrical ideas were, charge on the one hand, and discharge on the other. After the discovery of the galvanometer, the original point of view was abandoned altogether, or nearly so, and the attention diverted from the static to the current phenomena of electricity. And herein, as I believe, was an unmixed misfortune. In making out the electrical history of living creatures there is work to be done which, as will be seen in due time, can best be done with the electrometer; and, for my own part, I am disposed to assign to the new quadrant electrometer of Sir William Thomson a position in these investigations

which is not less important than that which must be assigned to the galvanometer, and to think that the apparatus of any physiological laboratory would, to say the least, be far from complete in which this instrument is wanting.

And thus, by the fact of the existence of animal electricity being now established beyond question, the way is more prepared than it was in the days of Galvani for the adoption of any view of vital motion in which animal electricity has to serve as the basis.

There are also others who must be named as taking what is substantially the same view as that taken by Galvani, and who have a just claim to be commemorated in these introductory remarks, about whose views I would say what I would also say about the views of Galvani, that I was in complete ignorance respecting them for long after the time when my own thoughts on the subject had taken definite shape.

The name to be mentioned first in order here is that of the late Dr. West, of Alford, in Lincolnshire. As early as 1832,* in some remarks upon the influence of the nerves upon muscular contractility, this writer maintains, "that the nervous influence which is present in relaxed muscular fibre is the only influence which the nerves of volition possess over that tissue; that its office there is to restrain or control the tendency to contract which is inherent in the muscle; and that contraction can only take place when by an act of the will this influence is

* "On the Influence of the Nerves over Muscular Contractility," "London Medical and Surgical Journal," edited by Michael Ryan, M.D. Vol. i. 1832.

suspended, the muscle being then left to act according to its own innate properties ;” . . . and again, “that nervous influence is imparted to muscular fibre for the purpose of restraining its contraction, and that the action of the will, and of all other disposers to contraction, is simply to withdraw for a while this influence, so as to allow the peculiar property of muscular fibre to show itself.” The co-existence of spasmodic action with nervous debility, the efficacy of stimulants as antispasmodics, and the postponement of rigor mortis until all traces of nervous action have disappeared, are the principal facts which are advanced in support of the probability of this theory.

A similar idea appears to have been also hinted at by Sir Charles Bell in a lecture at the Royal College of Surgeons of England, for, after premising that the question could never be settled, the lecturer said, “that *relaxation* might be the act, and not contraction, and that physiologists, in studying the subject, had too much neglected the consideration of the mode by which relaxation is effected.” This remark is preserved by Dr. West in the essay to which reference has just been made.

Six years later, in a chapter of his classical work on comparative anatomy,* Professor Dugès, of Montpellier argues with much clearness that all organic tissues are the seat of two opposite movements—expansion and contraction—and that “la contraction musculaire ne consiste que dans l’annihilation de l’expansion.” The muscle is supposed to contract in virtue of its elasticity, just as a

* “Traité de Physiologie comparée de l’Homme et des Animaux.” 8vo. Montpellier and Paris. 1838.

piece of caoutchouc is found to do when set free from a previous state of extension ; and it is hinted that between the expanded state of the muscle and the fluid state of the fibrine of the blood, and between rigor mortis and the coagulated state of this fibrine, there is a very close connexion. Analogous in its effects to electricity, the vital agent is supposed to accumulate in the muscles, and to produce expansion by causing the muscular molecules to repel each other ; and contraction is supposed to be brought about either by the sudden discharge (as in ordinary contraction) or by the gradual dying out (as in rigor mortis) of the vital agent. And, further, it is supposed that the rhythmical movements of muscle are caused by successive discharges of the vital agent, which discharges are brought about whenever this agent acquires a certain degree of tension ; and that the cramps of cholera, or the spasms of tetanus or hysteria, are consequent upon the development of the vital agent being for the time suspended.

More recently still, namely in 1847, Professor Matteucci communicated a paper to the Parisian Academy of Sciences* upon the influence of the nervous *fluid* in muscular action, in which he writes:—"Ce fluide développé principalement dans les muscles, s'y repand, 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 électrisés. Quand ce fluide nerveux cesse d'être libre dans le muscle, les éléments de la fibre musculaire s'attirent entre eux, comme on le voit arriver

* "Comptes Rendus." March 17, 1847.

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 importance to the 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."

Next in order, and almost contemporaneously with the date of my own first publication on the subject, Professor Engel, of Vienna, wrote :*—"So hat der Nerve die Aufgabe, nicht die Zusammenziehungen des Muskels zu veranlassen, sondern den Zusammenziehungen bis auf einen geringen Grad entgegenzuwirken. Im le-

* "Ueber Muskelreizbarkeit," "Zeitschrift der Kais. Kön. Gesellsch. der Aertze zu Wien," Erster Band, pp. 205-219, and pp. 252-270. 1849.

benden 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 entgegengesetzten Eigenschaften des Nerven und des Muskels ist das, was man gemeinhin Zustand der Ruhe, Zustand des Gleichgewichtes, oder an Muskeln auch Tonicität nennt. Das Verlassen dieses Gleichgewichtes ist die Bewegung einerseits, die Lähmung andererseits. Die Bewegung wird aber erzeugt, indem entweder der Einfluss des Nerven auf den Muskel herabgesetzt 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 Einwirkung des motorischen Nerven auf den Muskel. Sollen daher abwechselnde Muskelcontractionen zu Stande kommen, so ist die Gegenwart des lebendigen Nerven im Muskel unerlässlich, und auch bei unmittelbaren Muskelreizen können abwechselnde Zusammenziehungen nur erfolgen, so lange noch die Nerven lebensfähig sind; hört letzteres auf, so zieht sich der Muskel ohne Hinderniss zusammen. Diesen Zustand nennen wir die Todtenstarre." The chief grounds for this opinion are:—first, certain original experiments, some of them very remarkable, which afford additional proof that the muscles of frogs are more prone to con-

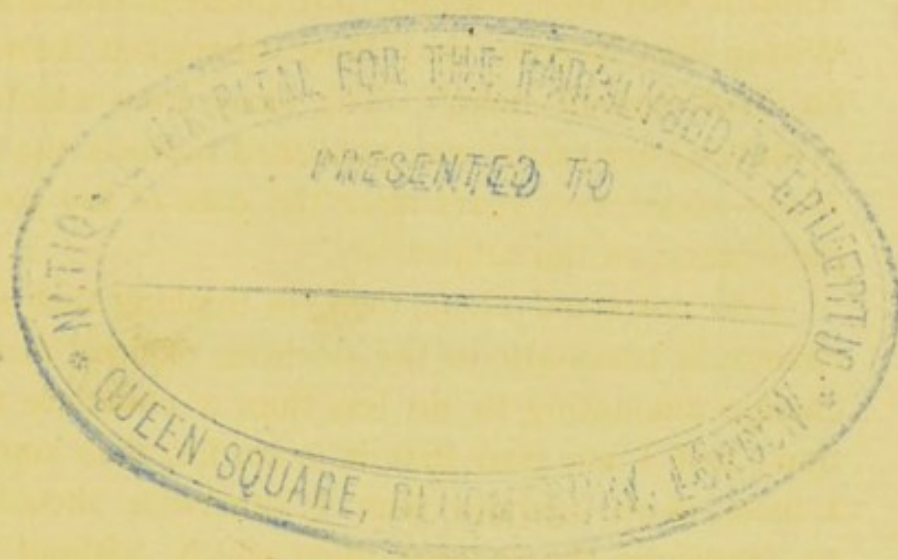
tract when they are cut off from the influence of the great nervous centres; secondly, the frequent spontaneous occurrence of cramps and other forms of excessive spasmodic contraction in paralysed parts; and, thirdly, the supervention of the permanent contraction of rigor mortis when all signs of nervous irritability are completely extinguished.

And, last of all, I find Professor Stannius, of Rostock,* arriving at this conclusion:—"dass es eine wesentliche Aufgabe der sogenannten motorischen oder Muskelnerven sei, die natürliche Elasticitätsgrösse 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 wieder 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." M. Stannius was led to this conclusion by certain original experiments, in which he found blood to have the power of relaxing rigor mortis and restoring muscular motility, and these experiments are advanced in evidence. Reference is also made to arguments to be brought forward on another occasion, which will prove—"dass diese Anschauungsweise, so paradox sie immer auf den ersten

* "Untersuchungen über Leistungsfähigkeit der Muskeln und Todtenstarre," "Vierordt's Archiv für Physiol. Heilkunde." Stuttgart, 1 Heft, p. 22, 1852.

Anblick sich anlassen mag, mit unserem thatsächlichen Wissen über Nerven- und Muskelthätigkeit keineswegs im Widerspruch steht." The essay from which these quotations are taken was published towards the end of 1852—about two years after the date of my own first publication on the subject.

I do not stand alone, then, in thinking that a great change is necessary in the doctrine of *vital motion*—a change amounting to no less than a complete revolution; and I am glad that it is so, for, thus supported, I have more courage than I otherwise should have to prosecute the inquiry upon which, without further preamble, I now propose to re-enter.



THE ARGUMENT.

THE ARGUMENT

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I.

That the fibres of living nerve or muscle, while at rest, are charged more or less positively at any point of their sides, and more or less negatively at any point of either of their two ends.

THE exhaustive investigations of Professor Du Bois-Reymond have made it certain that in nerve and muscle alike the fibres, while alive and at rest, are the source of a current of electricity—the so-called *nerve-current* in the case of the nerve, the so-called *muscle-current* in the case of the muscle—which is obedient to exactly the same law in both cases. This current is detected when the electrodes of a galvanometer, of which the coil is made up of a very great length of very fine wire, are applied so as to connect a point on the sides of the fibres with a point on either of the two ends of the fibres, and also when the two points thus connected are on the sides, or on either of the two ends, provided they are not equidistant from the edge between the sides and the ends, the current in this latter case being always considerably more feeble than in the former case. In nerve and muscle alike this current

pursues the same course, and, unless it be reversed, as it may be now and then, this course is one which plainly shows that the surface made up of the sides of the fibres is positive in relation to that which is made up by either one of the two ends of the fibres, and also that the former surface is more positive and the latter surface less positive, or more negative, in direct proportion to the distance from the edge between the two surfaces.

In nerve and muscle alike, as long as they are alive and at rest, it is also found that the fibres are *charged* more or less positively at their sides and more or less negatively at each of their two ends,—that the surface charged positively is most positive and the surface charged negatively most negative at the point which is most distant from the line of separation between these surfaces,—and that the charge, positive or negative, at its maximum is equal to about a third of the charge, positive or negative, of a Daniell's cell in the state of open circuit. By using the new-quadrant-electrometer of Sir William Thomson, or a Lipmann's electrometer, I have again and again satisfied myself that it is so, and, in point of fact, these statical phenomena are in every way as real as the current phenomena already mentioned.

In seeking to explain these facts I have come to the conclusion that the fibres of living nerve and muscle, while at rest, are *charged* as leyden-jars are charged, and that the nerve-current, or muscle-current, is in reality a *discharge* bridled down to the quieter pace of the current by the resistance met with in the coil of the galvanometer. Taking my stand upon the fact that the coatings

of the fibres agree closely in chemical composition with elastic tissue, which is a very bad conductor of electricity, I am led to think, not only that these coatings may be far worse conductors of electricity than the comparatively watery contents, but also that they may conduct electricity so very badly as to be capable of acting as *dielectrics*. And, so thinking, I can easily see that the main facts with which I have here to do are not altogether inexplicable. I can easily see why it is that while the sides of the fibres are positive each of the two ends is negative, for in order to do this all that is necessary is to suppose that a positive charge is developed on the exterior of the coatings—by oxygenation in all probability—and that an equivalent negative charge is *induced* on the interior, which negative charge is carried to each of the two ends, where the contents are exposed by the fibres being cut across there, simply because the contents conduct electricity more readily than the coatings. I can easily see how it is that the *insulation* is not wanting which is necessary to *retain* the charge, for the case of the charged coating is simply that of the charged leyden-jar. And I can also account for the nerve-current or muscle-current which is met with when differently *charged* points on the nerve or muscle are connected by the coil of a suitable galvanometer. The nerve-current or muscle-current is met with when the coil is made up of a very great length of very fine wire: the nerve-current or muscle-current is *not* met with when this coil is made up of a short length of comparatively thick wire. In order to detect either of these currents, that is to say, it is necessary to have a coil in which a high degree of

resistance is opposed to the passage of electricity: and, therefore, it may be that the current in question is, as I have said, no more than a *discharge* bridled down to the quieter pace of the current by the resistance met with in the coil. So it may be: and that it is so is, I think, not improbable, for at the moment when a nerve or muscle is included in the circuit of the coil it is thrown into a state of action, as it is by a discharge, if the resistance of the coil be insufficient to transform a discharge into a current, as it is when the coil is made up of a short length of comparatively thick wire, but not so if this resistance be sufficient to do this, as it is when the coil consists of a very great length of very fine wire. Thus far then I may go without any great danger of being bewildered, and this is further than I need go at present, for all I want to do now is to assume provisionally that the fibres of living nerve and muscle, while at rest, are *charged* in exactly the same way as leyden-jars are charged.

II.

That the charge which is present in living nerve and muscle during the state of rest disappears in great measure when this state changes into that of action.

WHEN living nerve or muscle passes from the state of rest into that of action there is, as is proved by many beautiful experiments with the galvanometer, a change

in the nerve-current and muscle-current to which its discoverer Du Bois-Reymond has given the name of "negative variation"—a change in which the current is, not reversed as some suppose, but simply weakened to a great degree. Nor is it otherwise when a Sir William Thomson's new-quadrant-electrometer, or a Lipmann's electrometer, is made to take the place of the galvanometer, for in this case it is always found—that the charge which is met with in living nerve and muscle during the state of rest is *not* to be met with when the state of rest has changed into that of action. I have often repeated this experiment with the electrometer and always with the same result. Indeed, so far as my own experience goes, the teaching of the electrometer in this matter is quite as clear and conclusive as that of the galvanometer.

III.

That the state of action in nerve and muscle is accompanied by a discharge analogous to that of the Torpedo.

A *rheoscopic limb** is thrown into a state of contraction when the free end of its nerve is brought into contact

* A *rheoscopic limb* is the hind leg of a frog, detached at the hip, and with all the parts above the knee snipped away except the sciatic nerve. It is placed in a dry test tube, with the nerve brought out over the lip, and when in use the tube is held so as to allow the free end of the dangling nerve to rest lightly upon the body whose electrical condition has to be examined.

with a muscle or nerve which at the time is in a state of action. It behaves, in fact, in exactly the same way as that in which it behaves when the end of the nerve is brought into contact with any part of a Torpedo which at the time is in a state of action; and, therefore, it is possible to find in this fact a reason for believing that the state of action in the nerve and muscle is attended by a discharge analogous to that which attends upon the state of action in the Torpedo.

Another reason to the same effect may also be found in the fact that there is a disappearance or discharge of electricity whenever a nerve or muscle passes from the state of rest into that of action, for in this disappearance or discharge there is a *movement* of electricity which must lead to the development of instantaneous faradaic currents of high tension in and around the fibres of nerve and muscle. Indeed, it is quite conceivable that the discharge of the Torpedo may in great measure consist of these very faradaic currents, intensified, it may be, by the interaction of the different cells and fibres concerned in the generation of the electricity.

And besides these reasons for arriving at this conclusion there are also those which were sufficient to satisfy Matteucci that muscular contraction is accompanied by a discharge analogous to that of the Torpedo, namely these:—that the nerves of the electric organs and of the muscles agree in originating in the same tract of the nervous system, in terminating in the same loop-like plexuses, and in responding in the same curious way to the action of the so-called *inverse* and *direct* voltaic currents, if only contraction be taken as the

equivalent of discharge,—that the electric organs and the muscles are alike paralysed by the division of their nerves, and thrown into action, after being so paralysed, by faradizing the nerve below the line of section,—that the electric organs and the muscles alike are thrown into a state of involuntary action by strychnia,—and that in both cases intervals of rest are needed in order to keep up the power of acting to a proper pitch of intensity.

In a word, there is, as it seems to me, sufficient reason to agree with Matteucci in believing that the state of action in nerve and muscle is accompanied by a discharge analogous to that of a Torpedo.

IV.

That there are in dead nerve and muscle no traces of the electrical phenomena which are peculiar to living nerve and muscle during the state of rest.

IN really dead nerve and muscle the nerve-current and the muscle-current are no longer to be detected by the galvanometer, and the signs of electricity which are brought to light by the new quadrant electrometer are, not those which are peculiar to living nerve and muscle, but those which are common to all lifeless bodies, and of which more will have to be said presently.

V.

That the natural electricity of a nerve is affected very differently by anelectrotonus and by cathelectrotonus, anelectrotonus augmenting, preserving, and even re-calling it, cathelectrotonus diminishing and speedily destroying it.

ONE of the ways in which the nerve-current of a rheoscopic limb is shown to be affected differently by anelectrotonus and cathelectrotonus is indicated in the two following figures. The voltaic electricity which is used

FIG. 1.

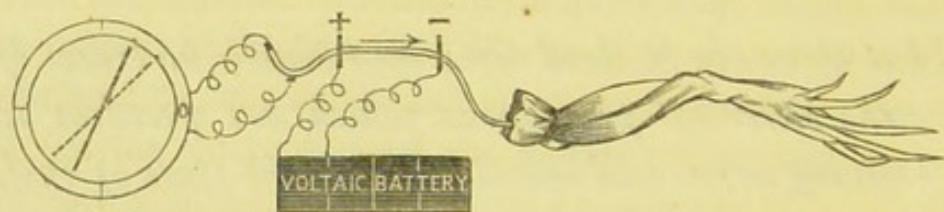
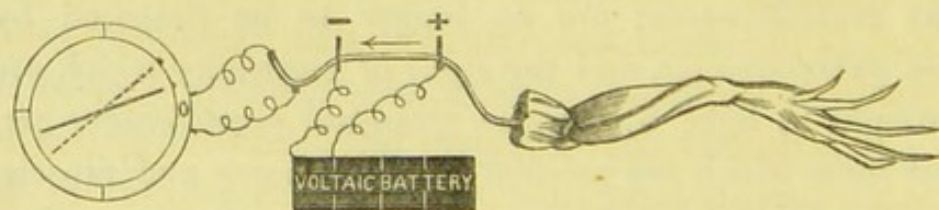


FIG. 2.



in producing the state of electrotonus is brought to bear upon the nerve midway between the free end from which the nerve-current passes into the galvanometer

and the end which loses itself in the muscles. The first step in the experiment, which is taken when the voltaic circuit is open, is to allow the nerve-current to enter the galvanometer, and to wait until the needle has come to rest at the point to which it has diverged under the action of the nerve-current. The next step is to set up the state of electrotonus by closing the voltaic circuit, and to see how the needle behaves, first in the case of *an*-electrotonus—which is that in which, as in fig. 1, the positive voltaic pole, or *anode*, is next the galvanometer, and afterwards in the case of *cath*-electrotonus—which is that in which, as in fig. 2, the negative voltaic pole, or *cathode*, is in this position. And this is what has to be noticed. Under the action of the nerve-current, before the state of electrotonus is set up, the needle diverges from zero and takes up the position which is indicated in the two figures by the dotted lines. Under the action of electrotonus the needle, which in the figures is represented by the continuous black line, is found to be more divergent in anelectrotonus and less divergent in cathelectrotonus—to be, in fact, where it is seen to be in figs. 1 and 2. The movements of the needle, that is to say, go to show that the nerve-current is strengthened in the use of anelectrotonus and weakened in that of cathelectrotonus.

In addition to these familiar facts there are also other facts which are less familiar but not less important. In the case of anelectrotonus the needle remains stationary at the same point for a long time, but not so in the case of cathelectrotonus. In the latter case, indeed, instead of remaining stationary, the needle presently passes to

the other side of zero, and takes up a position which is almost as far from zero as is the point on the other side of zero at which the needle remains stationary under the action of anelectrotonus. At the cessation of anelectrotonus the nerve-current is found to be little or not at all impaired in vigour, but not so at the cessation of cathelectrotonus. Indeed, at the cessation of cathelectrotonus, if only this state has been kept up for twenty minutes, or thereabouts, no trace of the nerve-current is discoverable. And, lastly, the nerve-current which has been driven away by the action of cathelectrotonus may be recalled by reversing the position of the voltaic poles so as to make the states of anelectrotonus and cathelectrotonus change places. In a word it is found, not only that the action of anelectrotonus upon the nerve-current is preservative and restorative, and that the action of cathelectrotonus is diametrically opposed to this, but also that, in the case of cathelectrotonus, the needle may move as it moves in cathelectrotonus *when the nerve has lost its nerve-current*.

And here arises a question the answer to which makes it evident that the electrotonic movements of the needle which are referred to modifications in the nerve-current are not sufficiently accounted for in this way. Does the needle move in anelectrotonus as it moves in cathelectrotonus when the nerve has lost its nerve-current? This is the question. The answer is—yes, and also when any other sufficiently bad conductor is substituted for the currentless nerve. It moves if any sufficiently bad conductor is used ; it does not move when a wire or other good conductor is used.

The presence or absence of the nerve-current is a matter of indifference. It is not even necessary to make use of nerve, living or dead, for the same results are arrived at if a piece of common string slightly moistened with saliva, or any other bad conductor, be put in place of the nerve. In each of these cases, the needle, now starting from zero, moves as it moves in anelectrotonus if the positive voltaic pole, or anode, be next the galvanometer, and as it moves in cathelectrotonus if the negative voltaic pole, or cathode, be in this position. After making these observations I found on looking about me recent footsteps which showed that Matteucci had already gone over the same ground and ascertained, first, that electrotonic movements of the needle are obtainable, not only from currentless nerve, but also from strips of other kinds of currentless organic tissue, and, secondly, that they are not obtainable when the galvanometer and the voltaic apparatus are connected by a wire of amalgamated zinc, coated with cotton thread, and wetted with a saturated solution of sulphate of zinc. And thus there is no reasonable room for doubt as to the correctness of the facts to which I am now wishful to direct attention.

Why it should be so is a question which must stand over for the present. Indeed, all I have to do now is to show that the nerve-current is, as I have pointed out, affected very differently by anelectrotonus and by cathelectrotonus.

VI.

That the natural activity of a motor nerve is affected very differently by anelectrotonus and by cathelectrotonus, anelectrotonus suspending, preserving, and even recalling it, cathelectrotonus, after exaggerating it for a short time, speedily destroying it.

A COUPLE of rheoscopic limbs, one or at most two voltaic pairs, and a Du Bois-Reymond's inductorium are wanted to establish the truth of this proposition. The nerve of the rheoscopic limb is arranged across the poles

FIG. 3.

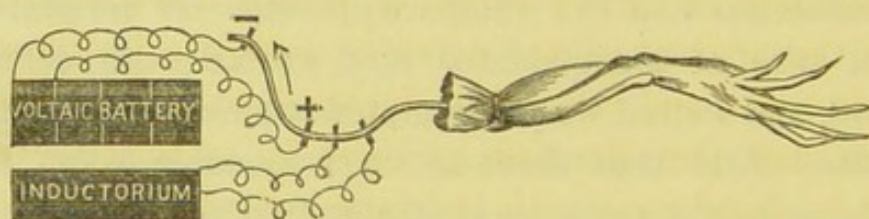
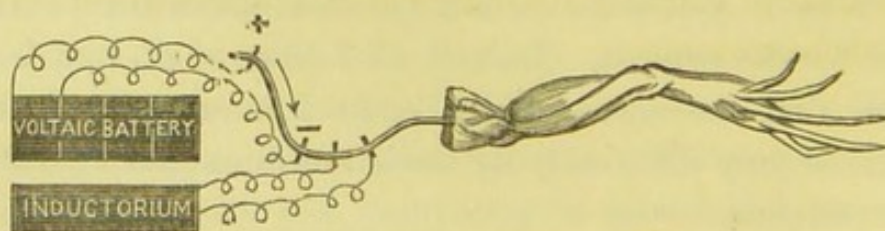


FIG. 4.



of the voltaic apparatus and across the electrodes of the inductorium, with the positive pole, or anode, next the

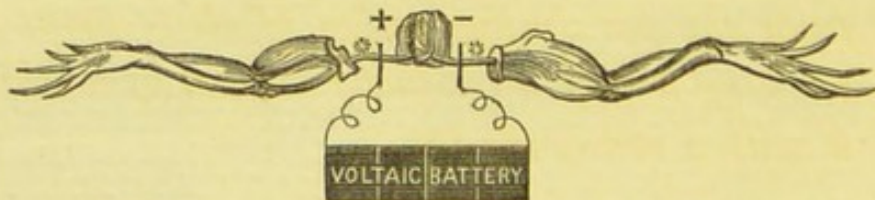
inductorium, as in fig. 3, if the object be to produce *an*-electrotonus on that side, with the negative pole, or cathode, in this position, as in fig. 4, if the object be to produce *cath*-electrotonus. Before setting up the state of electrotonus by closing the voltaic circuit, the inductorium is put in action, and, by drawing away the secondary coil from the primary, the nerve is at last exposed to shocks which are only just strong enough to produce very feeble contractions in the muscles. And then, while the muscles are still contracting in this very feeble manner, the state of electrotonus is set up by closing the voltaic circuit, with this result. If the part of the nerve acted upon by the inductorium be in a state of anelectrotonus the contractions are at once brought to an end. If the part of the nerve acted upon by the inductorium be in the state of cathelectrotonus these contractions are immediately exaggerated. And if the experiment be carried on a little further it is also found that the activity of the nerve is unimpaired after anelectrotonus, and very much impaired after cathelectrotonus, and that the damage done by cathelectrotonus may be done away with, not once only, but again and again, by shifting the position of the poles so as to make the state of cathelectrotonus change places with the state of anelectrotonus.

VII.

That a motor nerve is paralysed by cathelectrotonus as well as by anelectrotonus if the voltaic apparatus used in producing the electrotonic state consist of three or more elements.

THE truth of this proposition is easily demonstrated by repeating the last two experiments with a voltaic apparatus in which there are *three or more elements*, for if this be done the constant result is found to be, not suspension of contraction in the case of anelectrotonus and exaggeration of contraction in the case of cathelectrotonus, but *suspension of contraction in both cases*. And if other evidence to this effect be wanted it is to be found in a simple experiment upon a galvanoscopic frog, which I have often repeated with the same result. After arranging the frog across the voltaic poles as in the accompanying figure, after placing a drop of

FIG. 5.



strong solution of common salt upon each of the two nerves at the points indicated by the asterisks, and

after waiting until both the limbs are convulsed by the action of the salt-water upon the nerves, I set up the state of electrotonus by closing the voltaic circuit, and find—that the convulsive movements are at once brought to an end in both limbs alike—that, in fact, the contraction at once ceases alike in the limb beyond the positive pole, of which the part of the nerve acted upon by the salt is in the state of anelectrotonus, and in the limb beyond the negative pole, of which the part of the nerve acted upon by the salt is in the state of cathelectrotonus. If one or at most two voltaic elements be used the contraction, as I have already shown, is suspended by anelectrotonus and exaggerated by cathelectrotonus; but if, as in the case under consideration, the voltaic apparatus consist of three or more elements, the result is constantly what I have just stated it to be, that is, suspension of contraction by cathelectrotonus and by anelectrotonus equally.

VIII.

That a motor nerve is delivered for the moment from the paralysing action of electrotonus by touching it with a conductor but not by touching it with a non-conductor.

AS in the last proposition so in this I have to do with a new fact. The new fact with which I had to do in the

last proposition was this—that a motor nerve is paralysed by cathelectrotonus as well as by anelectrotonus if the voltaic apparatus used in producing the state of electrotonus contain three or more elements. The new fact with which I have now to do is this—that a motor nerve is for the moment delivered from this paralysing action of electrotonus by touching the nerve or muscle with a conductor but not by touching it with a non-conductor. Nothing can be more simple than the experiment, or more constant than the result. The contraction which had been banished by either form of electrotonus is at once recalled by using the conductor but not by using the non-conductor.

IX.

That a motor nerve is prevented from acting in electrotonus by the artificial charge which is communicated to it from one or other of the voltaic poles, and that the state of action is recalled when this charge is discharged.

THE condition of the voltaic circuit when the poles are connected by a nerve or other bad conductor is one of charge as well as one of current. When the circuit is open, or imperfectly closed as in this case, free electricity accumulates at the poles—free positive electricity at the positive pole, free negative electricity at the negative pole—and overflows to the neighbouring parts. When

the nerve is kept in a state of enforced rest by anelectrotonus or cathelectrotonus it is so charged, positively in the former case, negatively in the latter. All this is made out very clearly by using a suitable electrometer so as to test the electrical condition of the parts. The case, indeed, is one in which it is easy to see why a motor nerve which is kept in a state of enforced rest in electrotonus should respond differently to the touch of a conductor and to the touch of a non-conductor. There is contraction when the conductor is used: there is no contraction when the non-conductor is used. There is a charge which may be discharged by the conductor, and which cannot be discharged by the non-conductor. And, therefore, it is no unwarrantable stretch of fancy to suppose, as I do, that the motor nerve is prevented from acting in electrotonus by the artificial charge which is communicated to it from one or other of the voltaic poles, and that the state of action is brought back by the discharge of this charge. Indeed there is, as it seems to me, no room for any other supposition.

X.

That a motor nerve or muscle may be prevented from acting by its natural charge of electricity, and that the state of action may be brought about by the discharge of this charge.

AFTER what has just been said it is not difficult to entertain this proposition favourably. It is not likely

that artificial electricity should act upon motor nerve and muscle in one way and natural electricity in another: and certainly the fact that a motor nerve or muscle is charged naturally during the state of rest, and that this charge disappears wholly or in great measure when the state of rest changes into that of action may be regarded as a reason for believing that the nerve and muscle are affected by natural electricity and by artificial electricity in the same manner.

XI.

That the charge which prevents the state of muscular contraction may act by keeping the muscular molecules in a state of mutual repulsion, and that the discharge which permits the state of muscular contraction may act by leaving the muscular molecules free to yield to the attractive force which is inherent in their physical constitution.

THERE is little or nothing in this proposition which may not be accepted as self-evident. The molecules of a body which is charged by either positive or negative electricity are kept by the charge in a state of mutual repulsion; and therefore it is easy to believe that muscle may be relaxed because the molecules of the contents of the fibres are kept in a state of mutual repulsion by participating in the natural or artificial charge induced

upon the interior of the coating (v. Prop. I.). Nor can it be in any way wonderful that muscle should contract when the charge which inhibited the state of contraction is discharged, for in this case the muscular molecules must be more or less given over to the working of the attractive force which is inherent in their physical constitution. This physical attractive force must play a part in the production of muscular contraction. It may play an important part for anything that yet appears to the contrary; it may even have to do all the work which has to be done, for, in fact, the law of contraction in muscle is none other than the very same law which regulates the return of an elastic body from the state of extension to the state of contraction which is natural to it. But this question is one with which I must not concern myself seriously at this stage of my argument; and I therefore merely throw out these hints in passing.

XII.

That the chief if not only difference between ordinary muscular contraction and rigor mortis may be in this—that the charge which prevents contraction departs suddenly and returns immediately in the former case, and that it departs slowly and stays away permanently in the latter case.

THE facts to which reference is here made are not to be called in question, and the inference to which they lead

is almost inevitable. One cause of the difference between ordinary muscular contraction and rigor mortis may certainly be found by seeking here, but it does not follow that this is the only cause. On the contrary it may be necessary to suppose that the mobile material of muscular fibre is altered in rigor mortis in a way in which it is not altered in ordinary muscular contraction. It may be necessary to suppose that in rigor mortis there is a change in this material analogous to that which has taken place in the granular amœba or amœboid body in which amœboid movements no longer happen—that in rigor mortis this material has, in fact, become granular and coagulated. But all this may be allowed and more without making it necessary to shift the stand-point which is taken in the proposition under consideration, for it is quite conceivable that in living muscle the material in question may be kept in a hyaline and mobile state by the charge, and that it may become granular and coagulated and immobile when it is not so kept. In a word, there is, as it seems to me, no valid objection to the notion that the chief if not only difference between ordinary muscular contraction and rigor mortis may be in this—that the charge which prevents contraction departs suddenly and returns immediately in the former case, and that it departs slowly and stays away permanently in the latter case.

XIII.

That the movements of the needle of the galvanometer which are characteristic of electrotonus may be due to the discharge through the coil of the charge which proceeds from one or other of the voltaic poles, the discharge being bridled down into the quieter pace of the constant current by the resistance which it encounters in the coil.

ACCORDING to Matteucci polarization is at the bottom of these movements. The nerve, or other bad conductor, is polarized by the voltaic electricity acting upon it, and hence the movements. The zinc wire, coated with cotton thread and wetted with a saturated solution of sulphate of zinc is not polarized, and hence the absence of these movements. For my own part, however, I prefer to account for the presence and absence of these movements in these experiments by supposing that a charge, the discharge of which is virtually transformed into a constant current by the resistance opposed to its passage in the coil, is admitted into the coil in the former case and shut out from the coil in the latter. It is more than probable that the electrotonic charge, of the presence of which unequivocal proof is supplied by the new quadrant electrometer, may act upon the needle, for the needle moves as it moves in anelectrotonus and cathelectrotonus when a + or - charge of franklinic electricity is passed

through the coil in the direction in which it is supposed to pass in anelectronus and cathelectrotonus. It is certain that this charge will prefer to pass by the route in which it encounters least resistance—that it will pass through the coil when the resistance of the coil is less than that of the body which is being electrotonized—that it will be shut out from the coil when on the contrary the resistance of the coil is greater than that of this body ; and therefore the case with which I have to do is in reality sufficiently simple. For what is the case when the needle moves as it moves when a bad conductor, like living or dead nerve, is experimented upon ? It is one in which the charge from the nearest voltaic pole must pass through the coil and put the needle in motion in this way or that according as it happens to be positive or negative, because the resistance of the *bad* conductor which lies across the electrodes of the galvanometer, and which connects these electrodes with the voltaic poles, is *greater* than that of the coil. And what is the case when a good conductor, like wire, is put in place of a bad conductor, like nerve ? It is one in which the charge is shut out from the coil, and the needle remains at rest for that reason, because the resistance of the *good* conductor which lies across the electrodes of the galvanometer, and which connects these electrodes with the voltaic poles, is *less* than that of the coil. In the one case the needle must move ; in the other case it cannot. There is, as it seems to me, no occasion to go further in search of an explanation. And certainly it is better to stay here, as I am content to do, than to go further and fare worse, as I am likely to do, if I try

to make all the facts fit in with Matteucci's notion of polarization, or to enlist the services of constant currents derived from the voltaic circuit. There must be polarization in the case where a simple wire is experimented on, and yet the needle does not move in this case. And as to the derived currents, all that need be said is that they are shut out from the coil by effectual barriers of insulation. Indeed, the only result of trying to go further in search of an explanation of the problem under consideration is to make me more ready to go back, and be content with the explanation which was available before starting off on this excursion.

XIV.

That electricity supplies a key to the electrotonic alternations in the natural activity of nerve which are brought about by transposing the voltaic poles.

IF the voltaic poles are transposed, the states of an-electrotonus and cathelectrotonus at once shift places ; if there be no such transposition there is no such shifting of places. The cause at work in producing these alternations is plainly the transposition of the poles, and no other. The changes in activity are in no way to be disassociated from the changes in electricity. And, therefore, it is surely not too much to say that the electrotonic

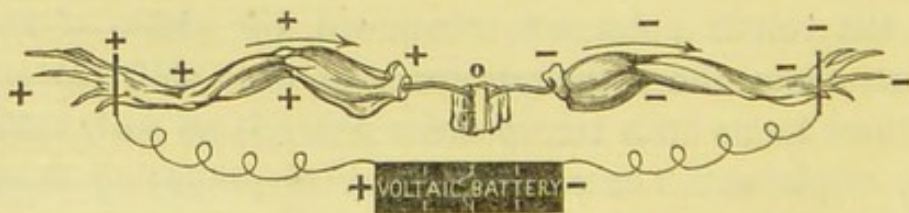
alternations in the natural activity of nerve which are brought about by transposing the voltaic poles are the necessary consequence of this transposition, or, in other words, that the natural activity of the nerve has an electrical basis.

XV.

That there is a strict parallelism between the changes brought about in the natural activity of motor nerve by the action of anelectrotonus and cathelectrotonus and the changes brought about in the same activity by the action of the so-called inverse and direct voltaic currents.

WHEN, as in the accompanying figure (fig. 6), a voltaic current is passed for some time along a galvanoscopic frog from foot to foot in the usual way it is found, not

FIG. 6.



only that one limb loses its activity much more speedily than the other, but also that this loss may be repaired again and again by reversing the position of the voltaic

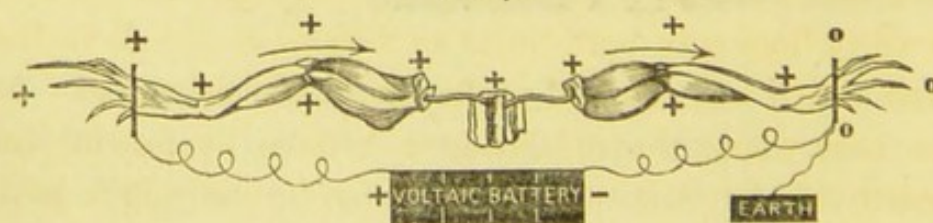
poles. It is found, indeed, that the limb which loses its activity speedily is the one in which the voltaic current is centrifugal, or *direct*, and that the limb which retains its activity is the one in which the current is centripetal, or *inverse*. What happened in the case of anelectrotonus is repeated in the case of the *inverse* current; what happened in the case of cathelectrotonus is repeated in the case of the *direct* current; and the parallelism between the action of anelectrotonus and that of the *inverse* current, and between the action of cathelectrotonus and that of the *direct* current does not fail when the effect of reversing the position of the voltaic poles upon the nerve is inquired into. What happens in the two limbs of a galvanoscopic frog under the action of the *inverse* and *direct* currents is indeed very much in keeping—and it is well to bear this fact in mind—with what the experimenter upon the frog experiences in himself whenever he passes through himself a voltaic current of sufficient strength from hand to hand or from foot to foot, for on doing so he finds before long, not only that the hand or foot belonging to the limb in which the current is *direct* is stiff and clumsy when the hand or foot belonging to the limb in which the current is *inverse* is as supple and clever as ever, but also that this state of things is transposable by simply reversing the position of the poles and keeping the circuit closed for a short time.

XVI.

That the changes in the motility of a galvanoscopic frog which are commonly referred to the voltaic current being inverse in one limb and direct in the other are really due, not to this cause, but, as in electrotonus, to the charge associated with the current being positive in one case and negative in the other.

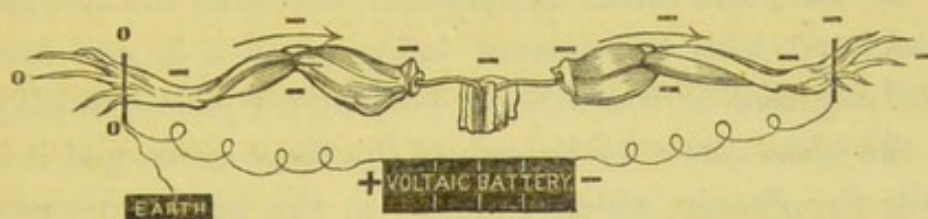
IN the case where a voltaic current is passed along a galvanoscopic frog from foot to foot in the usual way it is found that the limb in which the current is *inverse* is charged positively and that the limb in which the current is *direct* is charged negatively, *if the frog be insulated*. The case, in fact, is that which is indicated in the last figure (fig. 6) by the arrangement of the signs + or -. In the case where the insulation is broken by putting an "earth-wire" to the negative pole the direction of currents remains the same, but not so the arrangement of the charges, for now the - charge is run off to earth by the "earth-wire" and both limbs are, as is shown in the accompanying figure (fig. 7), charged positively.

FIG. 7.



In the case where the insulation is broken by putting an "earth-wire" to the positive pole, the direction of the currents remains the same, but not so the arrangement of the charges, for here the positive charge is run off to earth by the "earth-wire," and both limbs are, as is shown in the accompanying figure (fig. 8) charged negatively.

FIG. 8.



That it is so is easily made manifest by testing the electrical condition of the two limbs in these three cases by a Thompson or Lipmann's electrometer. Indeed, it could not well be otherwise.

Along with these electrical changes are also changes in the motility of the galvanoscopic frog which are full of significance. In the first case, where the insulation is unbroken, and where one limb is charged positively and the other negatively, as in fig. 6, the limb which is charged positively is found to retain its natural motility very much longer than the limb which is charged negatively. In the case where the insulation is broken by putting an "earth-wire" to the negative pole and where both limbs are charged positively, as in fig. 7, the natural motility lingers in both limbs as long as it did do in the limb which was charged positively in the first case. In the case where the insulation is broken by putting an "earth-wire" to the + pole, as in fig. 8, and where both

limbs are charged negatively, the natural motility departs from both limbs as speedily as it did do from the limb which was charged negatively in the first case. So it is most assuredly.

Here, then, are facts which make it evident that the changes in motility about which I have been speaking are dependent, not upon the voltaic current being inverse in one case and direct in the other, but upon the charges associated with the current being positive in one case and negative in the other. The current passes steadily in the same direction in each of the three cases, and it is only the charge which changes in the last two cases. As it was in the case of electrotonus so it is here. As the charge changes the duration of motility changes with it, and it is difficult to avoid the conclusion that here, as in electrotonus, the motility has a particular electrical basis. At all events, there is no room left for the supposition that the changes in motility are dependent upon the voltaic current being inverse in one case and direct in the other.

Nor is it necessary to change this point of view in order to see why the natural activity of nerve and muscle is affected favourably by the positive charge associated with the inverse current and anelectrotonus, and unfavourably by the negative charge associated with the direct current and cathelectrotonus. Indeed, if the natural activity of the nerve and muscle be based upon the natural electricity of the nerve and muscle, and if, as is suggested in the first proposition, the outsides and insides of the dielectric sheaths of the fibres are in opposite conditions as to charge, the outsides being positive,

- it follows as a matter of course that the communication of a positive artificial charge to the outsides of these dielectric sheaths will affect the natural activity of the fibres favourably by sustaining their natural charge, and that the communication of a negative artificial charge will have an opposite effect.

XVII.

That in the case where a state of action is set up in a galvanoscopic frog by voltaic electricity the explanation has to be sought, not in the working of the constant current, but in that of the instantaneous faradaic currents which traverse the voltaic circuit at the moments of closing and opening it.

WHAT is seen in the case of the galvanoscopic frog is seen in all cases where motor nerve or muscle is acted upon by voltaic electricity. At the moments of closing and opening the circuit, or at one of these moments, the state of the muscles is that of action : while the circuit remains closed this state is that of rest. In other words, the state of action is connected, not with the constant current, but with one or both of the instantaneous faradaic currents which traverse the voltaic circuit at the moments of closing and opening it. And, therefore, it may be taken for granted that the explanation of this state of action is to be sought, not in the working of the constant voltaic current, but in that of one or both of these instantaneous currents.

XVIII.

That the apparently eccentric behaviour of the muscles of a galvanoscopic frog under the action of the inverse and direct voltaic currents is found to be strictly in order, when due heed is paid to the differences in the direction and strength of the faradaic currents which attend upon the closing and opening of the voltaic circuit, to the gradual loss of muscular activity, to the differences between nerve and muscle as to electric conductibility, and to the loss of the capacity for reflex action which takes place in course of time.

IN order to deal satisfactorily with the present problem it is necessary to remember more than one fact. It is necessary to remember that the instantaneous faradaic current agrees in direction with the constant voltaic current at the closing of the circuit, and disagrees at the opening. It is necessary to remember that the faradaic current at the closing of the circuit is more powerful than that at the opening. It is necessary to remember that muscle is a far better conductor of electricity than nerve. It is necessary to remember that in any case the galvanoscopic frog presently loses in great measure

its capacity for *reflex* action. If these facts are not remembered the peculiar behaviour of a galvanoscopic frog in the matter of contraction under the action of the inverse and direct voltaic currents is altogether unintelligible; if they are remembered what seemed to be so very eccentric is soon found to be strictly in order.

Two distinct cases which are often confounded, and which for the sake of clearness may be called *the case of the nerves* and *the case of the muscles* have to be considered here, *the case of the nerves* being that in which, as in figures 6, 7, and 8, the exposed nerves of the galvanoscopic frog are kept on the stretch in the usual way, *the case of the muscles* being that in which either the muscular parts of the two thighs are made to touch, or else the space between them is bridged over by a pair of compasses or any other good conductor.

Up to a certain point, in both these cases the contraction, in both limbs equally, is at the moment of closing the circuit and also at the moment of opening it: beyond a certain point, the order of contraction changes in each case and differs in the two cases, the contraction in *the case of the nerves* taking place at the moment of closing the circuit and not at the moment of opening it in the limb in which the constant voltaic current is *direct*, and at the moment of opening the circuit and not at the moment of closing it in the limb in which this current is *inverse*, the contraction in *the case of the muscles* being in both limbs equally at the moment of closing the circuit and not at the moment of opening it. The problem which has to be dealt with is not a little difficult, but the difficulty is not insuperable. Indeed all real difficulty

is at an end when the facts to which attention has been directed are made use of at the proper time and in the proper way, and when it is clearly realized that in *the case of the nerves* the exposed nerves are acted upon by the faradaic currents which attend upon the closing and opening of the voltaic circuit in a way in which they are not acted upon in *the case of the muscles*—that, in fact, the exposed nerves are acted upon in *the case of the nerves* because they form part of the voltaic circuit, and that, in fact, the muscles rather than the exposed nerves are acted upon in *the case of the muscles*, because the faradaic currents are diverted from the exposed nerves, which are comparatively bad conductors, to the muscles, which are comparatively good conductors, by the muscles of the two thighs being made to touch, or by the space between the two thighs being bridged over by a pair of compasses or some other good conductor.

How is it, then, that in *the case of the nerves* the contraction in time comes to be at the closing of the circuit, and not at the opening, in the limb in which the voltaic current is *direct*, and *vice-versâ* in the limb in which this current is *inverse*? Is it that at this time the muscles respond only to the faradaic current which passes along the exposed nerves towards them? Is it that at this time the bit of spinal cord belonging to the galvanoscopic frog has become so far inactive as to be incapable of giving rise to *reflex* contraction under the action of the faradaic current which passes along the exposed nerves towards it? I see no reason why it may not be so; and if it be so then the order of contraction which has to be accounted for ceases to be inexplicable. In

the limb in which the constant voltaic current is *direct* the faradaic current is towards the muscles at the closing of the circuit, and not at the opening, and therefore the contraction is at the closing of the circuit, and not at the opening. It is the *reflex* contraction which is absent. In the limb in which the constant voltaic current is *inverse*, the faradaic current is towards the muscles at the opening of the circuit, and not at the closing, and therefore the contraction is at the opening of the circuit, and not at the closing. It is still the *reflex* contraction which is absent. And that it is really the *reflex* contraction which is absent in these two cases is evident, for when the contractions which are supposed to be reflex are present, as they are at first, they are at once banished by snipping away the bit of spinal cord, or by the operation of pithing.

And how is it that in *the case of the muscles* the contraction in time, in both limbs alike, comes to be at the closing of the circuit and not at the opening? Is it that the muscles differ from the nerves in being indifferent to the direction of the faradaic currents along or across their fibres? Is it that the muscles at this time have become so far inactive as to be capable of responding only to the stronger of the two faradaic currents, which is that which attends upon the closing of the circuit? If it be so—and there seems to me to be no reason why it may not be so—then it is easy to see why it is that, in both limbs, the contraction should be, as it is, at the closing of the voltaic circuit, and not at the opening.

And thus there is nothing remarkable in the fact that, under the action of the *direct* and *inverse* voltaic

currents, the muscles of a galvanoscopic frog should contract at one time and not at another as in reality they are found to do.

XIX.

That the last proposition is not contradicted by what happens when the undivided sciatic nerve of a rabbit is exposed to the action of the inverse and direct voltaic current, for here it is found that the cry, which betokens sensation, like the contraction in the muscles supplied by the sciatic nerve, must be referred, not to the working of the constant current, but to the working of the faradaic currents, one or both, which attend upon the beginning and ending of the constant current, and that the key to sensation and motion in this case is one and the same.

WHEN the undivided sciatic nerve of a rabbit is exposed to the action of the inverse and direct voltaic current this is what happens. All along there is neither sensation nor contraction while the constant current is passing steadily. All along the sensation and contraction are at the beginning or ending of the constant current, one or both. At first the sensation and contraction go hand in hand at the beginning of the constant current and also

at the ending ; afterwards they separate and the sensation is only at the beginning of the constant current if the contraction is only at the ending, and *vice-versâ*. Until the moment arrives in which the sensation is only at the beginning of the constant current and contraction only at the ending, or *vice-versâ*, the order of sensation and the order of contraction is the same in this experiment. Where sensation is absent contraction is also absent ; where sensation is present contraction is also present : and therefore, so long as this is the case, it fairly follows that the sensation and the contraction have to be accounted for in the same way. Nor is it necessary to change the point of view in order to see how it is that afterwards the sensation may separate from the contraction as it is found to do, for all that appears to be necessary is to suppose that now, by reason of some impairment in their natural activity, the sensory fibres of the nerves respond only to the faradaic currents which pass towards the sensorium and the motor fibres respond only to the faradaic currents which pass towards the muscles. It is not to be doubted that the faradaic currents which in this case produce sensation are centripetal in direction. It is not to be doubted that the faradaic currents which in this case produce contraction are centrifugal in direction. And, in fact, the history of sensation, as exhibited in this experiment, is in perfect keeping with the history of contraction as made out here and elsewhere.

XX.

That faradaic currents may bring about the state of action in nerve and muscle by discharging a charge which kept the nerve and muscle in a state of rest.

THE effect of subjecting a muscle to the shocks of a Rumkorff coil for a few minutes is to put an end to the charge which is present in living muscle during the state of rest, and to bring about the state of rigor mortis at once. The case is one in which it is easy to believe that faradaic currents do their work by driving away charge. Nor does it appear to be otherwise in any case where ordinary muscular contraction is brought about by faradaic currents. The state of ordinary contraction, however brought about, is accompanied by the disappearance of the charge which is present in nerve and muscle during the state of rest; and as very strong faradaic currents act as they do in producing rigor mortis there is no difficulty in supposing that faradaic currents of ordinary strength may act in the same way in producing the state of action in nerve and muscle, that is, by discharging for the moment the charge which is present in nerve and muscle during the state of rest. Nay there is no difficulty in supposing that these faradaic currents do not act differently when they are as feeble as the natural faradaic currents which

are induced in and around nerve and muscle when the state of rest changes into that of action. These currents, as is seen in the experiments for exhibiting the phenomenon of *secondary* contraction, are strong enough to act across a narrow gap, and, in fact, for anything that appears to the contrary, they may be strong enough for the purpose, as strong even as the discharge of the Torpedo, for in the body it is quite conceivable that their strength may be in great measure masked by their being short-circuited. In a word, there may be no occasion to go beyond the domain of physics in order to account for the state of action in nerve and muscle which is certainly brought about by the workings of natural or artificial faradaic currents.

XXI.

That a nerve or muscle which is thrown into a state of action by franklinic electricity is so thrown, not by the charge, but by the discharge.

THERE is no occasion to waste time in showing that a living body may be charged, and kept charged, with positive or negative *franklinic* electricity—if only care be taken to manage the charging so as to avoid a spark—without giving rise either to sensation or muscular contraction, and that the sudden discharge of this charge

is attended by sensation and contraction, for all this is familiar to every one who has paid the least attention to the subject. Indeed, all that I have to do now is to take this fact for granted, and to mention another fact, not so well known, which is easily verified by placing a galvanoscopic frog astride upon the end of a glass rod, by charging it at one time with positive franklinic electricity and at another time with negative, and by then bringing a knuckle into contact with one of the toes, for if this be done it is found that contraction is brought about from fifteen to twenty times before the discharge is completed if the charge be positive, and for only four or five times if the charge be negative. The contraction is repeated in this case, as it would seem, because the toe is twitched away from the knuckle by the contraction before there has been time for the charge in the limbs to be wholly discharged; and the repetitions are more numerous in the case of the positive charge than in the case of the negative charge, because the former charge is more friendly to the continuance of the natural activity of the parts than the latter. This is the conclusion which arises naturally out of the premisses: and thus there is nothing in the action of franklinic electricity in vital motion which does not chime in harmoniously with the previous conclusion respecting the action of voltaic electricity and faradaic electricity and animal electricity in vital motion.

XXII.

That the state of action may be marked by contraction in muscle and unmarked in nerve, not because the muscle and nerve are endowed with two different sorts of vital irritability, but simply because the material of muscle and nerve may differ in very much the same way as that in which an amœba which is capable of moving is found to differ from an amœba which is incapable.

BETWEEN the mobile material of the muscle and the semi-hyaline amœba in which amœboid movements are manifested there is a tolerably close bond of kinship: and between the semi-hyaline amœba in which amœboid movements are present, and the granular amœba in which such movements are absent, the relationship is still closer. The same amœba which is hyaline and mobile at first is granular and immobile afterwards: and it is difficult to avoid the conclusion that amœboid movements may come to an end when an amœba becomes granular for the very same reasons that ordinary muscular motility comes to an end when muscle becomes granular and stiffened in rigor mortis. And if so then the way is open along which it is only necessary to take a few steps in order to see how it may be that the state of action is marked by contraction in muscle and unmarked in nerve

—how there may be no occasion to call in the help of two different sorts of vital irritability in order to account for the differences in question.

XXIII.

That muscle may lengthen and shorten without undergoing any change of volume, not because it is endowed with a peculiar vital property of contractility, but simply because it is an elastic body.

IT is not necessary to suppose that muscle is endowed with a special vital property of contractility in order to understand why it is that muscle shortens and lengthens without any change of volume, for, in point of fact, a piece of india-rubber, or any other elastic body, behaves in exactly the same way. Indeed, all that may be necessary in order to arrive at a sufficient explanation of the fact in question may be to remember that muscle is an elastic body, and to suppose that it behaves like an elastic body in this matter. Moreover, in the fact that iron under the action of magnetism, and certain crystals under the action of heat, lengthen and shorten without any change of volume, may be found additional reasons for believing that the corresponding movements in muscle is a purely physical phenomenon.

Two experiments by the late Mr. Joule* show very

* "Philosoph. Magazine," Feb. and Apr. 1847.

plainly that a bar of iron lengthens and shortens without any change of volume when it is magnetized and demagnetized successively. In one of these experiments a bar of iron, enclosed within a coil of which the ends are in communication with a voltaic battery, is connected with a system of levers by which any changes in its length are multiplied exceedingly. In the other experiment, a similar bar is immersed in water by placing it in a glass tube, provided with a cork through which passes a sufficient length of glass capillary tubing, and surrounded by another coil of which the ends are connected with the same battery, and then, by pressing down the cork, the water is made to rise to a convenient height in the capillary tubing for showing easily any change of level. On magnetizing the bar by connecting the coil with the battery, there is, in the one experiment, a movement of the levers showing lengthening of the bar, and in the other experiment, an absence of any change of level of the water in the capillary tube. On de-magnetizing the bar by breaking the connection between the coil and the battery, there is, in the first experiment, a movement of the levers showing shortening of the bar, and, in the second experiment, the same absence of any change in the level of the water in the capillary tube. In point of fact, the bar lengthens and shortens under these circumstances without any change of volume, for if the lengthening and shortening to which the movement of the levers bears witness had been attended by any change of volume there must of necessity have been unequivocal risings and fallings in the level of the water contained in the capillary tube.

Under the action of varying degrees of heat there are also changes more or less parallel to these in rhombs of calc-spar, and in certain other crystals—changes of angles, that is to say, which show that what is gained in one direction is precisely compensated by what is lost in another. But it is not necessary to lay stress upon these or any other facts of which the precise significance is doubtful, for what has been said about elastic bodies is sufficient to show that muscle *may* shorten and lengthen without any change of volume, simply because it happens to be an elastic body.

XXIV.

That the force of the contraction may lessen as the muscle shortens simply because the muscle is an elastic body.

IN passing from the out-stretched to the un-stretched state the force of attraction displayed in a band of india-rubber is found to be inversely related to the degree of the shortening. In this respect, indeed, the case of the band is precisely that of the muscle; and, therefore, the fact that the contraction lessens in force as the muscle shortens may be nothing more than the necessary consequence of the muscle being an elastic body. At all events, it is possible to go far enough in this direction to be able to see clearly that there is nothing in the fact in question which points necessarily to the working of a vital property of contractility in muscle.

XXV.

That it is not necessary to entertain the hypothesis that a living power of contractility has been destroyed by death in order to account for the diminished cohesiveness of dead muscle.

DEAD muscle may be torn more easily than living muscle because the "juice of flesh," more or less analogous to gastric juice, which is contained in muscular tissue, is beginning to dissolve the muscle, or because the muscular molecules themselves have begun to be resolved into their constituent elements. Dead muscle may also be torn more easily than living muscle because the act of tearing may set up in the latter a state of contraction which it does not set up in the former—a state in which, by the constituent molecules being brought more closely together, molecular attraction is enabled to tell more forcibly. In a word, there is more than one way of accounting for the fact in question without being driven to the conclusion that dead muscle has lost a power which is peculiar to *living* muscle.

XXVI.

That a muscle may in time become incapable of contraction, not because a vital property of contractility is too much fatigued to allow of further contraction until it has been revived by rest, but simply because the electromotive apparatus of the nerve and muscle has got out of gear by being kept in action too long.

THE case of the muscle in which the capacity for acting is temporarily suspended from over-work may be not improperly likened to that of any ordinary battery in which the electromotive action has run down. Nor does it follow as a consequence of this view that the muscles should be contracted when they are in this case. It is the capacity for nervous action rather than the capacity for muscular action which is suspended by over-work: and, in point of fact, until rigor mortis sets in there is always enough electricity in the muscle to counteract contraction. Indeed, if living muscle be left to itself its electrical condition is naturally one which necessitates, not contraction, but relaxation—which, in short, prevents or inhibits contraction.

XXVII.

That there is no reason to look upon the contractile force of muscle as a product of the transformation of the natural electricity of muscle.

THE view that the contractile force of muscle is a product of the transformation of the electricity of muscle may be supposed to find no little support in the doctrine which suggests it—the doctrine of the correlation of the physical forces, but the view which resolves the contractile force of muscle into elasticity is, to my mind, more simple in itself, and more in accordance with the facts. Elasticity must operate very extensively in muscular contraction: and, this being the case, it is surely more philosophical to prefer a cause, about the existence of which there can be no doubt, to a cause which at the most can only be regarded as problematical, for, after all, there is no certain proof that the natural electricity of muscle is ever actually transformed into contractile force.

XXVIII.

That the action of so-called "stimuli" in causing vital motion does not necessarily imply the "irritation" of a vital property of irritability.

THERE is no occasion to think that the prick of a pin or the pinch of a pair of forceps provokes a state of action in nerve or muscle by "stimulating" a vital property of irritability, for, after what has been said, it is easy to see that the pin or forceps may derange the electrical balance of the nerve or muscle, and so bring about the electrical *movement* of which the necessary result is the development of the instantaneous currents of high tension which have been seen to have so much to do in setting up the state of action in nerve or muscle. The broad fact is that in every case in which nerve or muscle is made to act by any so-called "stimulus," be this mechanical, chemical, galenical, electrical, thermal, or other, there is always the same "negative variation" of the nerve-current or muscle-current—and, therefore, the notion that the "stimuli" may act thus electrically cannot be said to be merely fanciful.

And certainly it may be doubted whether the contraction of a hollow viscus is ever rightly ascribable to "irritation" on the part of the contents of the viscus. In the case of the bladder, for example, the urine ac-

cumulates, and the contraction is deferred until distention of the viscus is an occasion of discomfort. Up to this point the "stimulus" of the urine has seemed to favour dilatation rather than contraction in the bladder: and at this point it is not more necessary to suppose that the contraction is due to the urine having acted as a stimulus is supposed to act. When the bladder is full there is a feeling of uneasiness which shows—the act of sensation is accompanied by "negative variation" in the nerve-current of the sensory nerve—that the distention is acting upon the afferent nerves and producing a change in them, which, reflected through the efferent nerves belonging to the same reflex arc, may act upon the bladder *electrically* in the way which has been pointed out again and again. And if this be so with respect to the bladder it is easy to see that it may also be so with respect to any other hollow viscus—that, in fact, the hypothesis of "irritation" may be dispensed with in every case without any difficulty.

XXIX

That voluntary muscular action has nothing to distinguish it from other forms of muscular action so far as its electrical history is concerned.

IN voluntary muscular action, as in every other case of muscular action, there is the same "negative variation" of the muscle-current and nerve-current. As to the fact

there can be no doubt. There is an electrical basis of operation for the work of the will which cannot be ignored. And certainly the dignity of the will as a vital power is enhanced rather than compromised by finding this basis, for one practical consequence of the adoption of this view must be this—that instead of the will having to do its own work in muscular action by being, as it were, infused into the life of the nerve and muscle, electricity becomes the slave, and is made to do the work of the will.

XXX.

That there are many cogent physiological reasons for believing that the disposition to vital motion in nerve and muscle is inversely related to the supply of blood to the parts concerned in the movement, and that the blood acts, not as a stimulus to a vital property of irritability, but through the instrumentality of natural electricity.

THE facts with which I have here to do have nothing enigmatical about them, and it matters little how they are dealt with. Indeed, however placed, they may be made to serve as stepping-stones by which it is easy to get across any boggy ground in the argument.

The fact that death by bleeding is attended by violent convulsion in almost every muscle in the body is, to

my mind, altogether unintelligible if it be taken for granted that the work of the blood in the system is that of a stimulus to vital motion. Privation of blood and convulsion stand together here in the relation of cause and effect, and, so far as I can see, there is no escape from the conclusion that, in this case at least, the action of the blood, be this what it may, must have told upon the muscles *before* the convulsion rather than *in* it—that this action may have helped to keep off the convulsion rather than to bring it about.

The same story is also told, though not quite so plainly, by the fact that death by strangling is in like manner attended by general convulsion. For, as in the last case so in this, it is surely more easy to suppose that the convulsion has to do with want of arterial blood simply, than to agree with Dr. Brown-Séquard in believing that it is due to the stimulating action of the black blood with which the arteries are then filled to the full. Indeed, while one cause will serve equally for both cases, I cannot bring myself to think that two causes are really at work; and for this reason I shall, until I am obliged to shift my position, continue to believe that, in death by strangling no less than in death by bleeding, want of arterial blood is the common cause of the convulsion, and that the possibility of finding such common cause is in itself a sufficient ground for rejecting the notion that in death by strangling the neuro-muscular system is stimulated into convulsion by the carbonic acid with which all the blood in the system is then loaded.

In perfect keeping with these familiar facts are

also three experiments—one by Sir Astley Cooper,* two by Drs. Kussmaul and Tenner,†—in each of which it appears very plainly, not only that convulsion is connected with want of arterial blood, but also that this want tells directly upon certain great cranio-cervical nerve-centres, while in one of them it is further shown that convulsion has no connection with the opposite state of things.

“I tied,” says Sir Astley Cooper, “the carotid arteries of a rabbit. Respiration was somewhat quickened, and the heart’s action increased, but no other effect was produced. In five minutes, the vertebral arteries were compressed by the thumbs, the trachea being carefully excluded. Respiration stopped almost directly, convulsive struggles succeeded, the animal lost consciousness, and appeared dead. The pressure was removed, and it recovered with a convulsive inspiration. It then lay upon its side, making violent convulsive efforts, breathing laboriously, and with its heart beating rapidly. In two hours it had recovered, but the breathing was still laborious. The vertebrals were compressed a second time; respiration stopped; then succeeded convulsive struggles, loss of motion, and apparent death. When let loose, its natural functions returned with a loud inspiration, and with breathing excessively laboured. In four hours it moved about, and ate some greens. In five hours the vertebral arteries were compressed for the third time, with the

* Guy’s Hospital Report, No. III, 1836.

† Untersuchungen z. Naturlehre der Menschen, u. d. Thiers: Moleschott, vol. ii.—Frankfort, 1859.

same effect. In seven hours it was cleaning its face with its paws. In nine hours, the vertebrae were compressed for the fourth time, and the result was the same, viz., suspended respiration, convulsion, and loss of consciousness. On removal of the pressure, violent and laborious respiration ensued, and afterwards the breathing became very quick. After forty-eight hours, for the fifth time, the compression was applied, with the same effect."

In the first of the two experiments by Drs. Kussmaul and Tenner, ligatures, with slip-knots, placed around the common innominate and left subclavian arteries of a rabbit,—the only two great vessels proceeding from the arch of the aorta in this animal—are closed for a couple of minutes or thereabouts, and then unclosed. On closing them convulsion is at once set up everywhere. On unclosing them the convulsion, which is then raging at its height, ceases so suddenly and completely as to give rise to the impression that the animal has been struck down by a stroke of paralysis. This is what happens not once only, but again and again. At the very moment when the blood is shut off from the great cranio-cervical nerve-centres convulsion comes on. At the very moment when the blood is allowed to return to these centres the muscles become relaxed. There can be no doubt that here the convulsion is connected with a state of anæmia in certain great cranio-cervical nerve-centres, and not with a contrary state of things.

In the second of the two experiments by Drs. Kussmaul and Tenner, the supply of blood is cut off from the trunk and limbs, and diverted to the head, by tying

the descending aorta and the two subclavian arteries of a rabbit, and then the supply of blood to the head and neck is successively shut off by compressing the carotids between the fingers, and turned on by ceasing to do so. And this is what happens. When the blood is shut off from the parts below the neck by tying the vessels, all these parts, so far as they have to do with movement, are at once paralysed. When the blood is shut off from the head, by compressing the carotids between the fingers, general convulsion follows instantly. When the blood is allowed to return to the head by removing the fingers from the carotids, the former state of paralysis returns as instantly. As in the two last cases, so in this, convulsion comes and goes concurrently with the moments when the flow of blood to the head is prevented and permitted. The more plentiful supply of blood to the head in this case makes no difference in this respect: it only serves to give point to the same story, by making it evident that the effect of an unusually plentiful supply of blood to the great cranio-cervical nerve-centres—a state which may be supposed to be not unlike that to which the name of “active determination of blood to the head” is given—is, not convulsion, but paralysis.

Want of arterial blood would also seem to have to do with the spasms arising from the poisonous action of strychnia or brucia, for one way in which these poisons work mischief is, as is seen in the experiments of Dr. Harley,* by making the blood less capable of aëra-

* “Lancet,” June and July, 1856.

tion—by bringing about, that is to say, a change which is virtually equivalent to loss of arterial blood.

In one of the experiments in which this important fact is brought to light, air which had been over simple blood for twenty-four hours, and air which had been for the same time over blood containing a small quantity of strychnia, are examined by Bunsen's method, with this result:—

	Composition of common air.	Composition of air after having been over <i>simple</i> <i>blood</i> for 24 hours.	Composition of air after having been over <i>blood</i> <i>containing</i> <i>strychnia</i> for 24 hours.
Oxygen	20'960	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 blood made use of was collected as it flowed from an opening in the jugular of a calf, and the air and it were in about equal quantities. The plan pursued was to take two tolerably large test-tubes, to fill them half full of blood, to add to one of them two or three drops of solution of strychnia, to cork them both carefully, to set them aside with their mouths downwards, and, except at certain times when they were taken up for the purpose of shaking the air and blood well together, so to leave them until it was time to examine the air which had been bottled up all this while. At the end of twenty-four hours, when the air was removed and examined by Bunsen's method, it was found that the air

which had been over the blood containing strychnia, had been less acted upon by the blood than the air which had been over the simple blood, in that it contained more oxygen and less carbonic acid and nitrogen—a state of things which may be looked upon as pointing to a change in the blood which is equivalent to loss of blood; for it is evident that the strychnia had the effect of diminishing those respiratory reactions between the blood and the air which issue in the absorption of oxygen and the elimination of carbonic acid and nitrogen, and that blood so altered is as good as lost for all vital uses. Nay, it may be supposed that the change so wrought in the blood in fatal cases of poisoning by strychnia or brucia (for the only difference in the action of the two poisons is in the last-named being the less energetic of the two) is equivalent to copious loss of blood, for in the experiment under consideration, as may be seen in the table, a minute quantity of the poison had the effect of lessening the respiratory reactions between the blood and the air to the extent of full two-thirds of the natural amount.

And thus, as in the cases of convulsion already described, so in the case of the spasms produced by strychnia or brucia, there is reason to connect the excessive involuntary muscular action with the withdrawal of arterial blood,—to suppose, in fact, that the action of the blood tells in preventing muscular action rather than in producing it.

There are also two experiments, one by the late Dr. John Reid,* of Aberdeen, the other by Professor

* "Phys., Anat., and Path. Researches." 8vo. Edin., 1848.

Draper,* the younger, of New York, in which it is seen that in asphyxia black blood gets through the smaller vessels less readily than red blood, and from which, for this reason, it may be inferred that the state of vascular contraction is more effectually counteracted by red blood than by black blood.

Taking a rabbit, and laying bare the windpipe and the great vessels at the root of the neck, what is done by Reid is, first, to connect a hæmadynamometer with one of the carotids, and then to suffocate the animal by putting a ligature around the windpipe, and tying it. Before suffocation, the hæmadynamometer marks what may be regarded as the ordinary force and frequency of the pulse, and the artery is easily distinguished from the vein by being somewhat smaller, and by (the difference in the colour of the arterial and venous blood showing very plainly through the coats of the vessels) its red colour: after suffocation, the red colour of the artery rapidly changes into the black colour of the vein, and in about two minutes, when, that is, the process of suffocation is at its height, the artery is as black as the vein, and more distended than the vein, while, at the same time, the force of the pulse, as registered by the hæmadynamometer, is well nigh doubled. This is what is noticed before and after the establishment of suffocation. Black blood finds its way from the veins into the arteries, and is detained there because it does not get through the intermediate vessels with the same readiness as red blood. The state of things, indeed, is one which justifies

* "Lectures on the Physiol. of the Circulation." "Amer. Med. Monthly," April, 1860.

the conclusion that the minute vessels between the arteries and the veins are kept less open by black blood than by red blood.

In Draper's experiment, the heart and its great vessels are laid bare in a rabbit, and then a ligature is put around the trachea and tied tightly. At first, while the ligature remains open, the red and black sides of the heart, and the great vessels near the heart, are of their natural dimensions and colours; afterwards, when the ligature is closed, the blood is seen to accumulate, not in the vena cava and right side of the heart, as was expected, but in the aorta and left side of the heart, in the aorta first in order—to accumulate, that is, not in the venous system, but in the arterial, the arteries becoming larger and larger, and the veins smaller and smaller, as the arterial blood darkens into venous.

And so the fact that the arteries are more and more distended with black blood as the process of suffocation makes headway, and that the pulse becomes fuller and firmer as the blood in the arteries becomes more and more venous—a fact which is altogether at variance with the current belief that the arterial pulse fails rapidly in suffocation, and that the venous system as rapidly becomes gorged with black blood—may serve to show that the action of the blood is to prevent rather than to provoke a state of contraction in the minute vessels between the arteries and the veins; for, after what has been said, it is more easy to refer the contraction in these vessels (in consequence of which the arteries are filled at the expense of the veins) to the simple absence of red blood, than to the stimulating presence of black blood.

A similar conclusion may also be drawn from certain experiments by Dr. Spiegelberg,* of Göttingen, on the action of the blood upon the peristaltic movements of the bowel of a rabbit. In some of these, such movement is seen to be increased by pressing upon the abdominal aorta so as to prevent the admission of red blood into the vessels of the bowels, and to be diminished when, by removing this pressure, the blood is allowed to return into the empty vessels. In others, the same movements are seen to be increased, though not to the same extent, when the intestinal vessels are kept full of venous blood by pressing upon the vena cava or vena porta, and to be diminished when, by removing this pressure, these vessels are allowed at one and the same time to get rid of their load of black blood and to receive fresh supplies of red blood. Relaxation, not contraction, is associated with the presence, and contraction, not relaxation, with the absence of red blood: it is, indeed, as if the disposition to peristaltic movement were inversely related to the supply of red blood to the coats of the alimentary canal.

Again: the muscles which are less vascular are more prone to enter into, and remain in, the state of contraction than the muscles which are more vascular. Thus, the less vascular voluntary muscles of reptiles and fishes are more prone to contraction than the more vascular voluntary muscles of birds and mammals. Thus, the less vascular involuntary muscles of any animal are more prone to contraction than the more vascular voluntary muscles of the same animal. And thus, again—a fact

* Henle u. Pfeuffer's Zeitschrift.—3 Reihe ii, 1857.

which shows that in the two former cases comparative bloodlessness and proneness to contraction are really connected as cause and effect—the muscles of a dormouse, or any hybernating animal, are more prone to contraction in the state of hybernation, when the circulation is almost at a standstill, than in the period of summer life, when the blood courses along the vessels in full stream, and at unbated speed. There are, indeed, many facts, of which these are instances, in which the action of the blood upon muscle and nerve, be this what it may, is exhibited as being favourable to the state of rest, and unfavourable to the state of action; and, so far as I know, the facts which are in any way at variance with these have yet to be discovered.

And lastly, another version of the same story may be found in certain experiments in which Brown-Séquard* and Stannius† tested the action of the blood upon muscle which had passed into the state of rigor mortis.

On the 12th of July, 1851, Brown-Séquard began an experiment which consisted in again and again injecting a pound of defibrinated dog's blood into the principal artery of the arm of a criminal who had been guillotined at 8 o'clock on the morning of that day. The injections were commenced at 11 a.m., the arm then being in a perfect state of rigor mortis. Shortly afterwards, some reddish spots, not unlike those of measles, made their appearance, about the wrist more particularly.

* "Comptes Rendus," Juin 9 et 28, 1851.

† "Untersuchungen über Leistungsfähigkeit des Muskeln und Todtenstarre: "Vierordt's Archiv. für Phys. Heilkunde." Stuttgart, 1 Heft, 1852.

Then these spots went on enlarging, until, by their meeting and merging, the whole surface acquired a reddish violet hue. Then, the skin generally acquired its natural living colour, elasticity, and softness, and the superficial veins stood out distinct and full as during life. Then the muscles relaxed, and recovered their electro-motility, first in the fingers, afterwards in the shoulder. At 4 a.m., when the experiment was discontinued, there was no alteration in these respects. At the commencement of the experiment the temperature of the blood was 75° Fahrenheit, and that of the room 66°.

Another experiment was upon a full-grown rabbit which had been killed by hæmorrhage. In this case, after waiting until rigor mortis had fully set in, Brown-Séquard injected the defibrinated blood of the same animal into the principal vessel of one of the hind limbs. Fifteen minutes afterwards, the muscles of this limb had lost their stiffness, and recovered their electro-motility. From this time, throughout the night, until 3 p.m. on the day following, the injections were repeated at intervals of from twenty to thirty minutes, and all this while the relaxed muscles were highly irritable. From 4.50 p.m. to 7 p.m. the injections were repeated at tolerably regular intervals, with the same results as at first, rigor mortis being fully re-established in the part from which it had been banished when the experiment was resumed. On the following morning, this part was again in a state of cadaveric rigidity, while the rest of the body, which all along had been in this state, was beginning to pass out of it. On the third morning, the muscles everywhere were soft, and in an advanced state of putrefac-

tion, with the exception of those belonging to the limb upon which the injections had been practised, and in which no signs of the departure of rigor mortis were as yet perceptible.

About the time that Professor Brown-Séquard was engaged in these and other experiments of the kind, the late Professor Stannius, without any knowledge of what was being done in Paris, was carrying out an analogous series of inquiries at Rostock.

At 7.30 a.m. on the 21st of July, 1851, ligatures were put around the abdominal aorta and crural arteries of a puppy, and tied tightly. A few minutes after 10 a.m. the muscles had begun to stiffen in all parts from which the blood was excluded. At 10.45 a.m., both hind limbs were stretched out, and perfectly stiff and cold. 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. the natural warmth had returned in some degree to both hinder limbs, and the right limb was a little more flexible than the left. At noon, both limbs had fully recovered their flexibility, and it once appeared as if the left had moved spontaneously; but no sign of pain was caused by pinching the toes. At 12.30 p.m., the muscles which had been rigid contracted everywhere under the action of electricity, and once there seemed to be pain, for the animal, which was then quite quiet, gave a sudden plunge forward when it was electrified. Death happened unexpectedly at 12.45 p.m.

Early in the morning of the day following, a similar experiment was performed upon another puppy. At

noon, the muscles below the knee had ceased to respond to the action of electricity. At 2.15 p.m. both the paralysed hinder limbs were stretched out and rigid. At 2.35 p.m. the ligatures were untied. At 3.35 p.m. electricity gave rise to strong contractions in the muscles of both thighs and to weaker contractions in the muscles of the left leg below the knee; and very few traces of rigidity were to be detected anywhere. At 5.35 p.m. the muscles, now soft and limber everywhere, responded readily to the prick of a scalpel, as well as to the shocks of a coil machine. On the morning following the animal was found dead.

In another experiment in which the abdominal aorta and the crural arteries of a puppy were tied, and left tied to the end, it is shown very clearly that the rigidity of which mention is made in the two last experiments is identical with rigor mortis. In this case, four hours after the operation, the muscles *below* the ligatures were rigid and inactive. In the evening of the day following there was no alteration in this respect. Twelve hours later the animal was found dead, with the parts *above* the ligatures in a state of rigor mortis, and with the parts *below* the ligatures—which parts had been rigid before death—flaccid, moist, and putrescent. In other words, the parts *below* the ligatures were in the state which comes on after rigor mortis; and hence it follows that the stiffness which had existed in these parts before the death of the anterior half of the animal must have been identical with rigor mortis.

And thus it is evident, not only that muscles which have passed into the state of cadaveric rigidity may

be made to pass out of this state by again supplying them with blood, but also that the blood has the power of restoring to muscle the relaxed and mobile condition which is natural to it while alive and at rest—that, in fact (for so long as the supply of blood is kept up the muscles remain relaxed if they are let alone) the office of the blood is, not to cause muscular contraction, but to counteract it.

So far, indeed, the sum of the whole matter seems to be this—that the action of the blood in vital motion may be readily resolved into that of electricity—that, in short, the blood, by supplying oxygen in all probability, may develop the charge which antagonizes the state of action in nerve and muscle, and which in muscle keeps up, in addition, the state of relaxation. The evidence, in fact, is as much in harmony with this view as it is in opposition to the dogma that blood produces contraction by acting as a stimulus to a vital property of irritability which is inherent in living muscle and motor nerve.

XXXI.

That there are several preliminary reasons for believing that the disposition to vital motion in nerve and muscle is inversely related to the supply of "nervous influence" to the parts concerned in the movement, and that "nervous influence" acts, not as a stimulus to a vital property of irritability, but through the instrumentality of natural electricity.

AFTER what has been said about the action of electricity and about the action of the blood in vital motion it is difficult to rest content with the notion that the part played by nervous influence in vital motion is that of a stimulus to a vital property of irritability. In proportion to the supply of red blood to the nerve-centres is the development of nervous influence: and therefore much of what applies directly to the action of blood in vital motion may be supposed to apply indirectly to the action of nervous influence in vital motion. It is necessary, however, to study vital motion as it is manifested in rhythmical vital motion, in convulsion, in spasm, in tremor, and in neuralgia, before it is possible to arrive at anything like a clear conception of the action either of blood or of nervous influence in vital motion: and all that I can do now is to prepare the way a little for what has to be said about the action of nervous influence

in vital motion, and for what remains to be said about the action of the blood in vital motion.

There are sundry facts which seem to show that the disposition to muscular motion is inversely related to the supply of nervous influence to the muscles, and that the tone or tension of muscle is directly proportionate to the supply of nervous influence to the muscle ; and it is to these facts in particular that I wish to direct attention at present.

One of the facts which seem to show that the disposition to muscular contraction is inversely related to the supply of nervous influence is this—that the contractions which are produced in the hind leg of a frog by exposing its sciatic nerve to feeble faradaic shocks are found to become more marked when the animal is decapitated, more marked still when the spinal cord is cut across in the middle of the back, and most marked of all when the muscles are altogether severed from the spinal cord by dividing the sciatic nerve high up in the ham. Before decapitation the contractions are so feeble as to be only just perceptible. After decapitation there is no difficulty in detecting them. After dividing the cord they are tolerably strong. After cutting off the muscles from the cord by dividing the nerve high up in the ham, the very decided increase in the strength of the contraction is quite unmistakable. With each step in the experiment the disposition to contraction is obviously increased, and the only conclusion I can draw from the fact, for fact it assuredly is, is that muscular contraction is prevented or inhibited by nervous influence as it is by electricity.

Another fact akin to this, discovered by Brown-

Séquard, and telling the same story in another and yet clearer way, is exhibited in the reflex contraction which is produced in the hind leg of a frog by pinching one of the toes, for here it is found that a heavier weight is raised after the division of the cord than that which was raised while the cord was intact, that the lifting power increases for some time after the division, and that it remains for a long time unchanged afterwards. In two frogs, which may be distinguished as A and B, the weights actually lifted before and after the division of the cord, expressed in grammes, were:—

		A.	B.
<i>Before</i> division of the cord . . .		60	60
<i>After</i> division of the cord	Immediately after .	20	10
	In 5 minutes . .	45	30
	„ 15 „ . .	60	40
	„ 25 „ . .	80	60
	„ 60 „ . .	130	100
	„ 2 hours . .	140	120
	„ 4 „ . .	140	130
	„ 24 „ . .	150	140
	„ 46 „ . .	150	140

A month later the weight raised was still somewhat greater than that which was raised before the division of the cord. Indeed, it is very possible that the decline of power, which was not perceptible for several days, would have been far less perceptible at this time if due care had been taken to keep the muscles in good trim by exercising them electrically.

There are also certain experiments of Marshall Hall

upon rabbits and turtles which seem to show, not only that the effect of decapitating the animal is to diminish the firmness of the muscles, but also that this diminution in firmness becomes very much more marked after removing the spinal cord, or after destroying it by pithing. In the latter case, indeed, the muscles are found to lose their resiliency altogether, and to become perfectly limp. In the decapitated turtle, for example, the effect of removing or destroying the cord was to cause the sphincter ani to lose its circular form and contracted state, and to become flaccid and shapeless.

After what has been said it is easy to find in these facts a reason for believing that the muscles are kept in a state of tone or tension by nervous influence and that the disposition to muscular contraction is inversely related to the supply of nervous influence to the muscles. Indeed, in themselves and by themselves, the facts are almost sufficient to justify this conclusion, and at the same time to show that the part played by nervous influence in muscular motion cannot be that of a stimulus to a vital property of irritability.

XXXII.

That during the state of rest every cell and fibre of nerve and muscle may react electrically with every other cell and fibre of nerve and muscle and help in keeping up the state of molecular tone or tension which opposes or inhibits the state of action, and that there is no occasion to believe in the separate existence of an inhibitory system of nerves.

BETWEEN the case of the nerve and the case of the muscle in the two opposite conditions of rest and action there is electrically no difference whatever. In each case every fibre and cell is the seat of an electromotive action which keeps it charged during the state of rest; in each case this charge disappears when the state of rest changes into that of action. In each case this charge would seem to be the cause of this state of rest; in each case the discharge of this charge would seem to be the cause of this state of action. It is scarcely possible to regard the two cases as separable electrically. It is scarcely possible to escape the conclusion that electrically the fibres and cells of nerve and muscle interact mutually, with the result of intensifying the state of molecular tone or tension which opposes or inhibits the state of action in both nerve and muscle, in very much the same way as that in which the several cells

of a galvanic battery interact in augmenting the tension of the galvanic circuit. At all events the case is one in which it is not difficult to find a sufficiently good reason for believing, as I do, that in muscle and nerve alike *the state of rest* must be regarded as a state of active tension thus brought about—that in this state of rest every cell or fibre, of nerve and muscle alike, is prevented from passing into a state of action by being kept in this state of electrical tension—that *all* cells and fibres, in nerve and muscle indifferently, interact in producing this result—and that, in fact, there is no occasion to complicate matters by talking about a special system of inhibitory nerves.

XXXIII.

That the physiological history of rhythmical vital motion is in strict accordance with the previous conclusions respecting the action of blood and "nervous influence" in ordinary vital motion.

IN the Croonian Lecture delivered by Sir James Paget, in 1857, "on the cause of the rhythmic motion of the heart," it is made evident that this motion is closely connected with the action of certain nerve-ganglia, discovered by MM. Bidder and Rosenberger within the substance of the heart, and, at the same time, much light is shed upon the cause of rhythmic vital motion generally; and I cannot do better now than follow in the track which is here marked out for me.

It is not necessary to go far along this track in order to see that the cause of the cardiac movement lies within the heart itself, for the simple fact that the heart of the frog or tortoise goes on beating for some time after removal from the body is in itself a sufficient proof to this effect. Nor is it necessary to do more than attend to the evidence advanced in this lecture in order to be satisfied that the cause of this rhythmic motion may be further localised in the cardiac ganglia, to some of which the name of "rhythmic centres" has been given.

If, for example, the heart of a frog or tortoise be removed from the body, and divided into two portions by cutting transversely through the substance of the ventricle a little below the line of junction with the auricles, it is found that rhythmic motion goes on in the portion which comprises the auricles and the rim of the ventricle, but not in the other portion. When pricked, or otherwise "stimulated," the rimless portion of the ventricle may be made to contract like any ordinary muscle, but nothing serves to recall the lost rhythmic motion; whereas, except in being somewhat retarded, this motion goes on as it did before, and that, too, for some time, in the other portion. Again, if the heart of a frog be placed upon a board, with some of its own blood within it and around it, and scissors be then used (as Heidenhain used them) so as to snip away bit by bit, from above downwards, the auricles first of all, and the rim of the ventricle afterwards, the mutilation of the auricles is found to have little or no effect upon the pulsation, but not so that of the ventricle, for, as this is proceeded with, the pulsation is seen to

diminish progressively in force and frequency until—an event which usually happens when the rim of the ventricle has been snipped away to the depth of a couple of lines or thereabouts—it ceases altogether:—so that in this case it is evident that the cause of the rhythmic motion is not in the rimless portion of the ventricle, nor yet in the auricles, but in the rim which is cut away from the cup of the ventricle. Moreover, there is another way to the same conclusion through certain experiments (always upon frogs or tortoises) in which, without removing the heart from its place, ligatures are tied in various positions—experiments devised by the late Professor Stannius, and often repeated with the same results by Sir James Paget and others. In one of these experiments the ligature is tied around the great veins at some distance from the auricles, with the effect of producing little or no disturbance in the rhythmic motion which is going on in the heart on the one side, and in the great veins on the other side. In another experiment, what is done is to use two ligatures so as to close the great veins at the line where they enter the auricles with one, and to shut off the ventricle from the auricles with the other: and this is what happens. On tying the ligature which closes the great veins at their entrance into the auricles, it is found that the ventricle ceases to beat for a few moments and then begins to beat again at a much slower rate than usual, that the auricles cease to beat along with the ventricle and remain motionless afterwards, and that the great venous sinuses go on beating slowly and steadily as if nothing had happened. On tying the ligature which shuts off the

ventricle from the auricles—which ligature is placed around, or rather a little below, the boundary ring between the auricles and ventricle, so as to include the bulbus arteriosus in the frog or the two aortæ in the tortoise—it is found that the beating of the ventricle is accelerated, or renewed if it had come to a stop, that the auricles still remain motionless, and that the great venous sinuses still go on beating slowly and steadily at their natural rate of movement. Now, the nerve-ganglia, to some of which the name of “rhythmic centres” is given, lie chiefly in the boundary lines between the auricles and the ventricle, and between the auricles and the great veins; and, therefore, it is not difficult to see how the cutting away of the rim of the ventricle may put an end to rhythmic motion in the ventricle by removing rhythmic centres,—how the crushing of rhythmic centres under the ligature may put an end to rhythmic motion in the auricles in the experiment where the ligature is tied around the boundary ring between the auricles and great veins,—and how even the apparently contradictory fact of acceleration of rhythmic motion in the ventricle which comes to pass when the ligature is tied around the boundary line between the auricles and ventricle, may be explained, for here it may be supposed that the rhythmic centres are not wholly crushed by the ligature, that the parts which remain uncrushed are not sufficient to *inhibit* action to the full extent, and that by the lessening of the periods of diastole thus brought about, the systolic beats may be quickened. And thus, the facts being as they are, it becomes almost a matter of certainty that the source of

the rhythmical action of the heart must be sought, not in the cardiac muscular tissue alone or independently, but in the nerve-ganglia, to which the name of rhythmic centres has been given.

In speculating upon the *modus operandi* of these rhythmic centres in the production of cardiac motion, Sir James Paget has much to say which cannot be disregarded, but I find it difficult to accept the conclusion at which he arrives. He believes that the rhythmic action of the heart in the vertebrata "is due to the 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 that these discharges are themselves brought about by the nutrition of these ganglia "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, contracting." For my own part, however, I prefer to think that the cause of the cardiac rhythm is to be found in an unstable condition of the rhythmic ganglia as to *charge*. As it seems to me, the *inducing* charge (which gives the measure of the *induced* charge) has its origin in the oxygenation of the fibres and cells of these ganglia, and its measure in the extent to which this oxygenation can be carried out. As it seems to me, the act of charging may at a certain moment end in discharge, because the dielectric walls of these fibres and cells (in consequence of their thinness it may be) are not capable of holding all the charge which is supplied to them. As it

seems to me, the state of the cardiac muscle is that of relaxation (diastole) as long as the charge is present, and that this state changes into that of contraction (systole) when this charge is so far augmented as to lead to discharge. And there is, as I think, reason enough for so thinking. The beating of the heart is certainly dependent upon oxygenation, for it is found that the heart of a frog or tortoise, or a fragment of this heart in which there is a rhythmic ganglion, which is beating in the air, will cease to beat when it is placed in a vacuum, or in an atmosphere of hydrogen or carbonic acid or nitrogen, and resume its beating when air is re-admitted into the vacuum or substituted for the gases which have just been named, and also that this heart or this fragment, which has just ceased to beat in common air, will begin to beat again and go on beating for sometime afterwards, when it is removed into an atmosphere of oxygen or nitrous oxide gas. And the occurrence of electrical changes during cardiac action is put beyond question by Dr. Burdon Sanderson in an experiment upon the palpitating heart of a frog which may easily be repeated. What has to be done in this case, after arranging the clay electrodes of the galvanometer, not side by side in the usual way, but one above the other, is to place the heart between them, with its apex resting in a hollow upon the upper surface of the one, and with the auricles, or the bare rim of the cup of the ventricle, so arranged as to be in contact with the under surface of the other, and then to watch the spot of light upon the graduated scale, which spot tells of what is happening in the current thus introduced into the coil from the heart as the heart

goes on beating. And what happens is simply this—that as the heart goes on beating this spot of light moves forwards and backwards on the scale synchronously with the beats, forwards at the diastole, backwards at the systole, forwards in obedience to the current proceeding from the cardiac muscles and nerves during the state of rest, backwards when—in consequence of that disappearance of current to which the name of “negative variation” has been given—this state of rest changes into that of action. It is so when the entire heart is experimented on; it is so equally when a ventricle, from which all traces of the auricles have been removed, is made to take the place of the entire heart. In either case indifferently the ray moves forwards and backwards in the same way and to the same degree coincidently with the diastolic and systolic movements of the ventricle; indeed so it must be, for in the cut-out heart the only unmistakable rhythmic movements met with are, not in the auricles but in the ventricle singly. And so in supposing, as I do, that the cause of the rhythm of the heart is to be sought in certain electrical rather than in certain nutritive changes within the heart, I may make a firm stand on the fact that the beatings of the heart are really accompanied by synchronous up-and-down changes in the cardiac electricity.

Nor is it necessary to change the point of view in order to see how it is that the diastole of the auricle should correspond to the systole of the ventricle, and *vice versa*.

The absence of valves at the auricular openings of the great veins is to me a strong reason for believing

that the systole of the auricles cannot, by possibility, play that active part in the propulsion of the blood which is played by the systole of the ventricles; and, do what I may, I can come to no other conclusion than this—that the auricular systole is, in the main, due to the passive falling-in of the thin auricular walls upon the blood being suddenly gulped away from the auricles into the ventricles at the ventricular diastole,—and that the auricular diastole is, in the main, due to the simple passive filling-out of the auricles, arising partly from the regurgitation of blood produced by the backward movement of the auriculo-ventricular valves at the ventricular systole, and partly from the pressure of the column of blood which is continually setting-in towards the auricles through the valveless openings of the great veins. I cannot well be in doubt here. If the auricular systole had to drive the blood into the ventricles in the same way as that in which the ventricular systole has to drive it into the arterial system of vessels, then surely there *must* have been valves at the openings of the great veins into the auricles; and, therefore, the simple fact that there are no such valves is, as I think, a good reason for concluding that the auricles may be little more than elastic cisterns for feeding the ventricles,—that their work, like that of the great venous trunks, may be chiefly that of balancing the moving column of blood,—and that, in short, the auricular movements are, in fact, little more than passive consequences of the movements of the ventricles.

Moreover, I find another and perhaps still more cogent reason to the same effect in another direction.

Thus (as in one of the experiments already mentioned) the effect of tying a ligature around the boundary ring between the auricles and the great veins in the heart of a frog or tortoise, is this—that rhythmic motion goes on in the ventricle on the one side, and in the great veins on the other, *but not in the auricles*. And thus, as in an experiment which I have more than once tried upon the heart of a tortoise, the effect of tying a broad ligature tightly around the ventricle, a little below its line of junction with the auricles, is to put a stop to rhythmic motion in the ventricle, and to leave the auricles and the great veins opening into them beating at the same slow rate. It is evident, that is to say, that the rhythmic motion in the ventricle may go on without any movement exactly corresponding to it in the auricles, and also that the rhythmic motion which is manifested in the auricles when there is no such movement in the ventricle, has nothing in it to distinguish it from the slow rhythmic motion which belongs to the great venous sinuses ; and, therefore, I do not see that there is anything to prevent me from finding in these experiments much that is corroborative of the conclusion which I have ventured to draw from the absence of valves at the auricular openings of the great veins.

In this way the explanation of the rhythmic motion of the heart generally may be made to resolve itself into the explanation of the rhythmic motion of the ventricles mainly, and this again, perhaps, into that which applies equally to capillary movement and to vascular movement generally. For, after what has been said, a very natural conclusion appears to be this, that the ordinary

vaso-motor centres may act upon the coats of the capillaries and other simple vessels as do the rhythmic centres of the heart upon the walls of the heart, that is, by way of charge alternating with discharge, the charge producing a sort of vascular diastole, the discharge leading on to a sort of vascular systole.

And so likewise when the enquiry is extended so as to include other cases of rhythmic motion, as, for example, the case of the respiratory movements of the chest, or that of the mantle in the cuttle-fish, or that of the gelatinous body-disk of the pulmonograde *acalephæ*, or that of the lateral leaflets of the *desmodium gyrans*, or that of the *oscillatoria*; for in each and all of these cases it is easy to see that there is no occasion to call in any other principle of interpretation than that which has been made use of hitherto.

There is no reason why the view which has been taken in explaining the movements of the heart should not also be taken in explaining the movements of the chest. The rate of movement in the chest is slower than the rate of movement in the heart, and this is the only difference of moment. And this difference may soon be disposed of by supposing that the dielectric walls of the fibres and cells of the respiratory nerve-centres, though still incapable of holding all the charge which is supplied to them, are, simply by being thicker it may be, capable of holding more than is held by the dielectric walls of the ganglia which have to do with the movements of the heart. In both cases discharge happens when the point is reached at which the walls can hold no more charge; in the case of the chest more time is spent in reaching

this point than is spent in the case of the heart ; and, therefore, the stage of muscular relaxation is more prolonged and the moment of muscular contraction more deferred in the case of the chest than in the case of the heart. This is all.

This explanation would also seem to be readily applicable to the rhythmic motion of the mantle of the cuttle-fish—a motion by which, as may now-a-days be seen in any aquarium without any trouble, the water is alternately drawn into the branchial cavities through the slit-like aperture at the base of the funnel, and ejected through the funnel ; and, in fact, I only refer to this instance of rhythmic motion in order to point out in it what seems to me to be a cogent proof that the state called muscular relaxation is something more than simple cessation of contraction—something very like active expansion. I have often watched this rhythmic motion in the mantle of the cuttle-fish, and I am satisfied that the diastolic movement by which the water is sucked in has something in it which must be of the nature of active dilatation or expansion. It cannot be due to the resiliency of elastic fibres upon the cessation of a previous state of contraction, for such fibres form no part of the texture of the mantle. It cannot well be due to simple cessation of a state of contraction, for, so far as I can see, this would lead, not to the water being drawn underneath the mantle, but simply to the limp mantle being firmly pressed inwards by the weight of the circumjacent water. In point of fact, I can only see in this movement of the mantle of the cuttle-fish a proof that the state of muscular fibre to which the name

of relaxation is given is, not merely cessation of contraction, but something more akin to active elongation or erection. Nay more, I cannot look at the movement in question without coming to the conclusion that the diastolic movement may be equally active in other forms of rhythmic muscular motion—that, for example, the blood may be *drawn into* the ventricle of the heart at the diastole as well as *driven out* at the systole, and that the force concerned in carrying on the circulation may be *doubled* in like manner in the capillaries and other vessels, the blood being *drawn into* the vessels at the time of vascular dilatation, as well as *driven out* of them at the time of vascular contraction.

To a certain extent, also, the same explanation may meet the case of rhythmic motion as exhibited in the jelly-like body-disk of a pulmonograde acalaphe, for here there is a neuro-muscular apparatus which may be supposed to behave in the same way as that in which this apparatus has been seen to behave in the cases of rhythmic motion which have just been under consideration. In this case, however, it is not easy to believe that the neuro-muscular system is sufficiently developed to do all the work which must be done in order to account fully for the rhythmic motion which has to be accounted for, and it is difficult to escape the conclusion that here there is another cause at work which is the sole cause of rhythmic motion in the mobile parts of the lateral leaflets of the desmodium gyrans and in the moving filaments of the oscillatoria. In the swollen petiolules supporting the lateral leaflets of the

desmodium gyrans (which are the mobile parts) are certain peculiar cells in which the soft, jelly-like, or semi-granular cores are surrounded by well-defined walls, and not altogether unlike these cells are the moving filaments of the oscillatoria, and many of the cells which are met with in the mobile parts of the sensitive plant or Venus' fly-trap. Indeed, there is that in these fibres and cells which reminds one of the more or less elongated, somewhat spindle-shaped fibre-cells of unstriated muscle, which fibre-cells have the import of cells. Or rather the case is one in which it may be supposed that here, low down in the scale of being, where no traces of nerve or muscle are to be found, each of these filaments or peculiar cells is potentially at once a rhythmic nerve-centre and the neuro-muscular system belonging to it. At all events, it is quite conceivable that the case may be one in which, as in muscle, the cell may be charged as a leyden-jar is charged, and that the contents may swell and shrink as the charge comes and goes, for in order to allow of this all that is necessary is for the walls to be sufficiently dielectric, and for the contents to be sufficiently mobile. In this way the rhythmical movements of the petiolules supporting the lateral leaflets of the desmodium gyrans, and of the filaments of oscillatoria, will have to be explained in the same way as that in which the rhythmical movements of a detached fragment of the heart of a frog or tortoise has to be explained, for in each case the same electrical basis for the movement is provided—*is* provided, I say, for after what Dr. Burdon Sanderson has told us respecting the electrical history of the *Dionæa muscipula* it may

almost be taken for granted that the movements under consideration are associated with definite electrical changes.

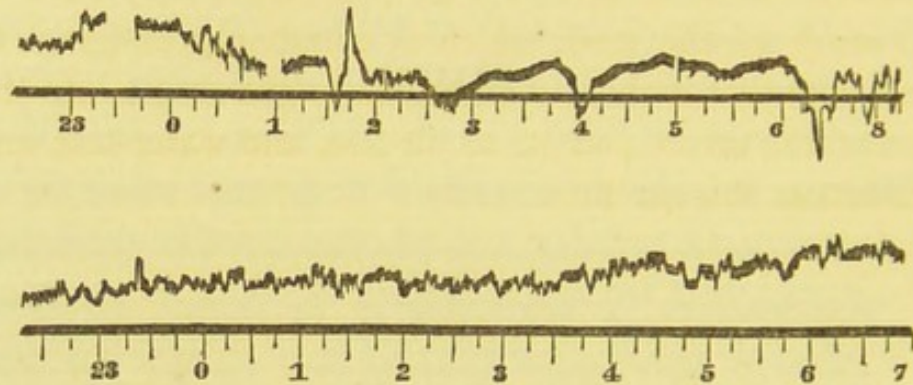
XXXIV.

That an amœba or amœboid body may move as it moves merely because, in obedience to the continual fluctuations in atmospheric potential, its irregularly distributed hyaline and mobile portions swell and shrink to a degree which is far beyond that to which its irregularly distributed granular and comparatively immobile portions are capable of reaching.

THE investigations of the last few years have shown very clearly that the electricity of the atmosphere and earth is in a state of continual unrest. Every day there are two maxima and two minima in this mode of potential, one maximum between 8 A.M. and 11 A.M., the other between 7 P.M. and 11 P.M.; one minimum between 3 P.M. and 7 P.M., the other between 11 P.M. and 3 A.M. Every day also there are minor momentary oscillations in potential which in comparison with these major tidal movements may be looked upon as waves—oscillations of which a vivid picture is presented in the accompanying figure, which figure is a copy of two portions of a daily tracing made by a reflected ray of light upon

the sensitive paper of a self-registering apparatus whose office was to record the movements of atmospheric potential at the point where the water dripped from

FIG. 9.



the nozzle of the pipe of the "collector." There are, indeed, waves as well as tides in atmospheric potential, both of which must tell more or less upon all terrestrial bodies affected by them—upon amœbæ and amœboid bodies among the rest. And, this being the case, it is easy to believe that the movements which are exhibited in amœbæ and amœboid bodies may be nothing more than electroscopic movements—that, in point of fact, amœbæ and amœboid bodies may be only so many natural electrosopes. After what has been said, indeed, it is easy to see that the protrusion and retraction of amœboid processes may be nothing more than the swelling and shrinking of the irregularly distributed hyaline portions of the amœba or amœboid body in obedience to the up-and-down movements of atmospheric potential, which swellings and shrinkings are made all the more conspicuous by the absence, or comparative absence, of such movements in the

granular portions of the same amœbæ or amœboid bodies. Nor can it be objected to this view that the power which is supposed to be at work can scarcely be equal to the task assigned to it, for, in point of fact, there is reason to believe that this power may have to do much of the work which is commonly assigned to heat, and that without it the earth might shrink into the size of the moon, and be as air-less, and water-less, and life-less as this satellite.

XXXV.

That vibratile cilia may move as they do simply because they are acted upon by the instantaneous faradaic currents which are induced in and around them by the movements which are continually happening in atmospheric potential.

INSTANTANEOUS faradaic currents in this direction or that must be induced in and around the vibratile cilia whenever there is any movement in the potential of the neighbouring atmosphere: and it is easy to believe that the cilia may be put in motion by these currents. There is no occasion to suppose that ciliary movement is in any way dependent upon the working of any special irritability in the cilia themselves, or in the part on which they grow. Indeed, after what has been said, the natural conclusion appears to be this—that the cilia and the

cells supporting them are as passive in the matter of ciliary movement as are the blades of grass, or the ground on which they grow, when these blades are set in motion by a breeze.

XXXVI.

That the history of epilepsy and other forms of convulsion, as set forth in the condition of the vascular and nervous systems, is in flat contradiction to the notion that convulsion is caused by an excessive development of "nervous influence" consequent upon an over-abundant supply of blood.

I. THE immediate precursor of the epileptic paroxysm is a sign which is somewhat difficult to catch—corpse-like pallor of the countenance. M. Delasiauve* was the first to notice this phenomenon, and M. Trousseau insists upon it as a mark which distinguishes true epilepsy from feigned epilepsy. "Il est une signe," says he,† "qui se produit au moment de la chute, qui n'est imitable pour personne, c'est la pâleur très prononcée cadaverique, qui couvre pour un instant la face epileptique. Nous ne la voyons pas, parceque nous arrivons trop tard, alors que la face est déjà d'une rouge très prononcée." In fact the

* "Traité de l'Epilepsie." 8vo. Paris, 1855.

† "L'Union Médicale." 28th Apr., 1855.

general form of the epileptic or epileptiform paroxysm, *le haut mal*, begins in the same way as the partial form to which the name of *le petit mal* is often given, for cadaverous pallor of the countenance has certainly a very conspicuous position among the initial symptoms in this latter case.

A habit of sighing, as if the proper balance of breathing could only be maintained by now and then taking breath more deeply than usual, is also observable in many epileptics, and not unfrequently, especially when the fit happens during sleep, the breathing may come to a standstill so complete and so prolonged as to make a bystander fear that the patient is actually dead. More than once I have felt this fear myself; and again and again my attention has been struck by the sighings to which I have referred; and, in short, there is, as I believe, good reason for believing that there is some radical fault in the breathing in many epileptics, and that the fit itself is often ushered in by a complete cessation of all proper respiratory movements.

In the typical epileptic or epileptiform paroxysm, the staring, squinting, out-starting eyes, the black and bloated countenance, the noisy gasps and ghastly distortions caused by ineffectual attempts to breathe, show plainly enough what is happening. The state which accompanies the convulsion is evidently that of suffocation. It is as if some invisible fiend had tightened his strong arms around the chest of the unhappy sufferer. Nor is it really otherwise in those varieties of the disorder, partial or general, in which the face remains pale and shrunken from the beginning to the end of the paroxysm, for here

the face has always a ghastly pallor or lividity which shows very plainly that the convulsive symptoms are accompanied by some grave interruption in the proper aëration of the blood.

In some cases the pulse at the wrist is almost or altogether imperceptible from the beginning to the end of the paroxysm ; in others it rallies speedily and when the fit is at its height it is at once hard and full and frequent. How then is this ? What is the true meaning of this state of seeming vascular over-action ? The common belief on the subject is that an increased quantity of *red* blood is injected into the arteries during the paroxysm, and that this increased quantity of red blood produces the convulsion by provoking a state of increased functional activity in one or other of the great nerve-centres, and among others Schroeder van der Kolk* has given distinct expression to this belief. In reality, however, there is reason to know that the pulse acquires power during the paroxysm, because the condition of the circulation at the time is one of suffocation, and for this reason simply. For the condition of the circulation in suffocation is—what ? It is *not* one in which, as is generally supposed, the arterial pulse fails rapidly for want of blood, and the venous system as rapidly becomes gorged with un-aërated blood ; on the contrary, it is one in which the arteries fill at the expense of the veins, and the pulse in the arteries becomes stronger and fuller as the colour of the blood in the arteries changes from red to black, because black blood does not

* "On the Proximate Cause and Rational Treatment of Epilepsy," New Sydenham Society Series. 8vo. London, 1859.

get through the capillaries with the same facility as red blood. Evidence to this effect is supplied in the experiments of Reid and Draper of which an account is to be found on a former page (p. 96). It is, indeed, certain that the strong and full pulse of the epileptic or epileptiform paroxysm may be nothing more than the natural pulse belonging to the state of suffocation which obtains at the time—the pulse of black blood, the *apnæal* pulse, as it may be called. Nay, this is the only conclusion which is allowable, for with the respiration completely, or all but completely, at a standstill, as it is in fact, it is simply impossible that there can be increased injection of *red* blood into the arteries during the paroxysm. Moreover, a right view in this matter explains many apparent anomalies in the pulse. It explains, for example, how it is that blood drawn from the temporal artery in a fit is often black in colour and projected to an unusual distance, and how in cases of congestion of the lungs, and in some other cases where the aëration of the blood is greatly at fault, the pulse may beat with seemingly contradictory power in the very last moments of life. It shows, in fact, that the pulse may derive a fictitious value from the admission of black blood into the arteries when the respiration is insufficient, and that mere power of pulse, apart from the condition of the respiration, is a very unsafe criterion of true vascular vigour.

Over-activity of the circulation, indeed, forms no part of the history of epileptic or epileptiform disorder, either in the fit or in the interparoxysmal period. Instead of predisposing to these disorders all febrile excitement would seem to have a contrary effect, for it often

happens that fits of frequent recurrence are suspended for days by fever, as in the case where severe symptomatic fever follows a burn or other injury inflicted during a fit, or in that where sharp idiopathic fever happens to be set up, and that they recur as frequently as before when the fever has passed off. And certainly nothing to the contrary is to be gathered from the state of the circulation in the interparoxysmal period, for here if anything at all out of the common is noticeable it is sure to be some indication of defective vascular vigour—a pulse easily flagging, hands and feet readily becoming cold and clammy, and the like.

In short, the history of epilepsy and epileptiform disorder, as set forth in the vascular system, is quite in keeping with all that has been said upon the working of the blood in vital motion, in that the convulsion is always seen to be connected with a state which is very far removed from vascular over-action.

2. In epilepsy all consciousness is suddenly suspended, and memory keeps no record of what happened in the fit. The miserable patient may wake to find himself terribly burnt or mutilated in some other way without knowing when it happened. And in the minor form of the disorder, where the screaming, falling, and struggling of the major form are wanting, and where even walking and talking may go on automatically without interruption, the mind is altogether blank, perceiving nothing, remembering nothing. For the time being, in point of fact, the state during the actual attack of epilepsy, partial and general alike, is nothing less than one of mental obliteration.

And in the interparoxysmal period there is much in many epileptics which is calculated to show that the disposition to epilepsy is inversely related to the degree of mental power. In very bad cases an unmistakable process of mental deterioration may be at work: and in the very worst cases of all, as in those which are gathered together in the epileptic wards of a workhouse or county lunatic asylum, epilepsy may be associated with downright idiotcy or fatuity or lunacy: but it is, I believe, a mistake to look upon these cases as other than exceptional. An epileptic may have had many fits for many years without being more damaged by them than he would be by the same number of bad headaches; and very often there is reason to believe, not only that much of what is wanting mentally may be ascribed to the fact that the mind has been allowed to lie fallow when it ought to have been cultivated, but also that the true relation of epilepsy to idiotcy or fatuity or lunacy, is often that of an effect rather than that of a cause. Indeed, it is easy to see that the very gloomy views which are entertained by many respecting the mental condition of the epileptic are not justified by the facts, and that in the majority of cases there may be in reality very little difference, mental or other, between the epileptic and non-epileptic: but still, when all is said on this side of the question that can be said there still remains a doubt as to the full mental capacity of the epileptic which cannot quite be explained away, and which in a measure justifies the belief that the disposition to epilepsy is inversely related to the possession of mental power.

Many of the facts which have been brought to light by the investigations of pathological anatomists are of little use in serving to fix the seat of epilepsy in any particular part of the nervous system. The changes in the cerebral hemispheres which have been found in epileptics who have also been idiotic or fatuous or lunatic do not serve to fix the seat in these hemispheres, for these very changes are continually met with in idiotic or fatuous or insane persons who have not been epileptic. I am also helplessly at fault as to localization when I consider the exceptional cases in which after death has been found thickening of the bones of the skull or the contrary, or exostosis, or ossification of the dura mater, or caries, or ulceration, or abscess, or tubercle, or malignant or other tumors, or aneurism, or embolism, or cysts, or atheromatous or ossified vessels, or hæmorrhage, or venous effusion, or sclerosis, or softening of the brain substance, for the seat of these and other lesions is as inconstant as their character. Nor is it possible to agree with Wenzel in fixing the seat of epilepsy in the pituitary body,* for Rokitansky† says that he has "frequently failed to discover any change in this body in those who had notoriously suffered from epilepsy and convulsions," and that he has "met with it in others who were thoroughly healthy." Indeed, the only changes which have any real claim to be looked upon as peculiar to epilepsy are those which have been found by Schroeder

* "Beobacht. über den Hernauhang fallsüchtiger Personen.," &c. 8vo. Mainz, 1810.

† "Manual of Path. Anat." Tr. (for the Sydenham Society), by Dr. C. H. Moore. Vol. iii, p. 424.

v. der Kolk in the medulla oblongata. In early cases of epilepsy, it is true, nothing at all characteristic was met with even here, but in confirmed cases this organ was always found to be harder than natural, from the interstitial deposit of a minutely granular albuminous matter, or else softened and swollen and fatty. The posterior half was also redder and more hyperæmic than it ought to be, even when the patient had not died in a fit: and on making a more minute examination, the blood vessels were found to be dilated to thrice their natural dimensions, and with their walls much thickened. These vessels, moreover, were seen to be especially dilated in the course of the hypoglossus nerve and corpus olivare in epileptics who were in the habit of biting their tongues in a fit, and in the course of the vagus in epileptics in whom this habit was absent. These statements are based upon fifteen post-mortem examinations. From these facts, and also from the seat and bilateral character of the spasms in epilepsy, Schroeder v. d. Kolk argues that the especial seat of epilepsy is to be found in the medulla oblongata, and up to a certain point he argues rightly. The post-mortem facts speak for themselves, and so also, to a certain extent, do the spasms. The spasms begin in muscles which receive nerves from the medulla oblongata—in muscles, that is to say, which are supplied by the facial, the accessory, the hypoglossal and the portio minor trigemini: and in slighter cases they are limited to these muscles. And the spasms in the walls of the chest may also be looked upon as pointing to the same nerve centre: for a similar state of spasm is brought about by the action of an induction coil upon the

great afferent nerve of this centre—the pneumogastric. Nor does the bilateral character of the spasm in epilepsy lead to a different conclusion. The lateral halves of the medulla oblongata are connected in the most intimate manner by transverse fibres and commissures: and hence it is that the corresponding nerves belonging to the two sides of the medulla oblongata are under a stronger physical obligation to act together than that which rules the corresponding nerves belonging to the two sides of the brain and spinal cord. And, therefore, the fact that in epilepsy the spasms are always more or less *bilateral* may fairly be looked upon as a reason for supposing that they have some special connection with a nerve-centre of which one lateral half cannot act without the other acting also—with, that is, the medulla oblongata.

As it seems to me, however, Schroeder v. d. Kolk would have been nearer the truth if he had extended the seat of epilepsy so as to make it take in the spinal cord on the one hand and the mesocephalon (or the continuation of the spinal cord in the base of the encephalon) on the other—if, in the latter case, that is, he had taken the pons varolii and cerebellum along with the medulla oblongata, and the corpora quadrigemina and pineal gland along with the crura cerebri, and the island of Reil and the pituitary body along with the thalami optici and corpora quadrigemina. Indeed, the more I look into the matter the more am I inclined to think that the spinal cord is affected in a very special manner in epilepsy. In the fact, for fact it is, that in the seizure the breath is held, not by any spasm of the larynx, but by the walls of the chest being fixed, I find

a reason for supposing that the spinal cord is more concerned than the medulla oblongata in bringing about the state of suffocation which is so marked a feature in the epileptic seizure. In the fact that a very early, if not the very earliest, manifestation of spasm in epilepsy is in the form of trismus, I find a reason to the same effect—if only I may look upon the 5th pair of nerves as belonging to the spinal series: and so also in the fact that the first convulsions in epilepsy are almost as tetanoid in their character as the spinal spasms of tetanus itself. And I do not come to a different conclusion when I call to mind the innumerable experiments in which Dr. Brown-Séquard has shown most conclusively that true epilepsy is producible in guinea-pigs by wounding the spinal cord in various places, and that Dr. Westphal invariably met with minute hæmorrhages in the spinal cord and cervical medulla in the guinea-pigs in which a habit of epilepsy, beginning in an attack of general convulsion, was brought about by hitting them smartly upon the head once and again. I have not yet had an opportunity of testing the correctness of these conjectures by examining the spinal cord of dead epileptics: and it remains to be seen, therefore, whether I am right or wrong in thinking, as I do now, that the reasons for connecting the spasms of epilepsy with disorder of the spinal cord are scarcely less cogent than those which serve to connect tetanus with such disease. Nor am I led to a different conclusion by remembering that in several of the guinea-pigs in which Dr. Brown-Séquard produced epilepsy artificially by wounding the nervous system in various places, the part injured was, not the spinal cord,

but one or other of the principal nerves of the thigh, or the medulla oblongata, or the pedunculus cerebri, or the corpora quadrigemina, and that, still more recently, Dr. Hertzog has found it possible to produce epilepsy in guinea-pigs by removing certain portions of the cortical substance of the brain. I cannot question the accuracy of the facts, and I have no need to do so. I allow, indeed, that the damage to the nervous system of which epilepsy is the consequence may be in various parts of the nervous system, perhaps in any part; but I cannot see in this view anything but another reason for believing that one part of the nervous system may react upon another part, and that in the cases where the spinal cord or its continuation in the mesocephalon is not affected primarily it may be affected secondarily, as in the case of the epileptic guinea-pigs with minute hæmorrhages in the cord and cervical medulla which were made epileptic by hitting them smartly upon the head. Indeed, in all the facts I see only this—that in every case a damage has been done to some part of the nervous system which has lessened the power which this system as a whole possesses of inhibiting or antagonizing contraction, and that it is primarily by this lessening of this general inhibitory power that the convulsion is brought about, even in the cases in which the spinal cord is injured directly. Moreover, I find nothing in any of these facts to make me ready to believe that the convulsion has to do with any inflammatory or hyperæmic change which has been set up by an injury; on the contrary, in the great majority of cases what I find is this—that there has been abundant time for any such

inflammatory or hyperæmic change to pass off, and to be replaced by a state of *irritation*, which in these cases at least would seem to be the reverse of a state of exalted vitality in the part. For in the epileptic guinea-pigs in which Dr. Brown-Séquard was able to produce a fit at any time by tickling, or by irritating in some other way, one of the cheeks, or the antero-lateral part of one side of the neck—the region to which he has given the name of *epileptogenous zone*—it was found that this part or region was always more or less *anæsthetic* in one way or another.

In epilepsy, therefore, the nervous system would seem to be at fault in a way which cannot well be mistaken. There is manifest inaction of those parts which have in an especial manner to do the work of the mind. There is manifest inaction in those parts which have to do with respiratory movement. Convulsion and suffocation evidently go hand in hand. The case is one which is altogether inconsistent with the notion that convulsion has to do with exalted functional activity in any part of the nervous system, for the failure in the supply of arterial blood to the part must, so far as I can see, carry with it an equivalent failure of functional activity in the part. The case is one which is altogether consistent with the notion that the failure in the supply of arterial blood may carry with it an equivalent failure in the electrical activity in the nervous system generally, beginning, perhaps, in the tract which has specially to do with respiration, and that the disturbance in electric equilibrium thus brought about may issue in convulsion in the very same way as that which has been indicated

with sufficient distinctness again and again in the physiological portion of the argument. In all respects the case is as it should be if the premisses are tenable.

II.

1. In the convulsive disorders associated with hysteria and chorea the breathing is not arrested as it is in the epileptic or epileptiform paroxysm, but it is shallow, embarrassed, often broken by sighs, and generally accompanied by a sense of breathlessness almost amounting to actual stifling; and there is nothing in the state of the circulation which can be looked upon as pointing to over-activity of any kind. In hysteria the difficulty is to keep the hands or feet warm, or to avoid chilblains, when the weather is at all cold; and frequent palpitations show how often the heart is called upon to make up for work which is left undone by the "capillary force." There is, in fact, a sort of radical weakness in the vascular system which seems to be not remotely akin to that which is met with in hibernating animals. Nor is it really otherwise in chorea. Here, perhaps, there is a disposition to rheumatic fever—at least in this country, but in this fact there is no reason for supposing that the febrile and choreic symptoms are in any way concurrent. The place of the choreic symptoms is, not along with the febrile symptoms, but before them, or after them—often long before or long after them; and the inevitable inference from the facts is that chorea in itself is essentially a feverless malady. Not unfrequently, also, there

are signs which point to a condition of circulation the very opposite to that which is met with in active fever, such as coldness and clamminess of the hands and feet, pastiness and puffiness of certain parts of the integuments, anæmic vascular murmurs, and the like. Indeed, the very predisposition to rheumatic fever may itself be taken as a reason for thinking that the circulation in chorea is radically weak. Moreover, it not unfrequently happens that the symptoms of chorea are suspended by the accidental development of scarlet fever, or other true febrile disorder, and that they recur when this state of feverishness is at an end.

The true place of the convulsion in connection with any form of febrile disorder, moreover, is in the cold stage before the hot stage, or in the stage of collapse after the hot stage, and not in the hot stage. It seems as if there were something uncongenial or even incompatible between the convulsion and an excited state of the circulation. In the fevers of infancy and early childhood, in the exanthemata especially, convulsion not unfrequently occupies the place taken by rigor in the fevers of later years; and it is confined to this stage, except there happen to be certain brain or kidney complications of which more will have to be said presently. It is rather the fever which is the consequence of the convulsion, than the convulsion which is the consequence of the fever. And certainly it is difficult to connect with anything like fever the convulsion which may happen at a later period in the state of collapse which is too often the immediate precursor of death.

Nor can a different place be assigned to the con-

vulsion connected with teething, or worms, or any other of the many manifestations of the state to which the name of "morbid irritability" has been given ; for here, most assuredly, in the few instances where the history of the convulsion is at all complicated with fever, the place of the convulsion is, not side by side with the fever, but before the fever or after it, precisely as it was in the cases referred to a moment ago.

And surely nothing contradictory to these conclusions is to be found in the sad case in which convulsion takes its most terrible form—the case of convulsion from loss of blood in childbed ; for here any doubt as to the connection between the hæmorrhage and the convulsion, if that were possible, must be set aside by the fact that transfusion of blood must be reckoned as the very chiefest means of arresting the convulsion, and saving life.

In the convulsion which may happen in uræmia and other forms of blood-poisoning, the condition of the vascular system may be somewhat obscure, but there is no reason to think that it is essentially altered. Possibly the great deficiency of blood corpuscles, which is a marked feature of Bright's disease in its advanced stage, is more concerned in the production of the head-symptoms than the uræmic poisoning. Sir Thomas Watson is of opinion that the pale and watery condition to which the blood is at last reduced in albuminuria may have something to do in bringing about the stupor and coma which generally mark the close of the disorder, and he bases this opinion upon the fact that similar symptoms are apt to ensue, in conjunction with a similar deficiency

of hæmatosin in spurious hydrocephalus; and I am quite disposed to accept this view, and to apply it to the interpretation, not only of the stupor and coma, but of the convulsion also. At all events this is certain,—that the convulsion of kidney-disease is associated with a state which is in every way the reverse of that which may be spoken of as vascular vigour—a state in which the blood is pale and watery, and the circulation carried on very imperfectly: and also this—that the whole drift of the argument hitherto is contradictory to the notion that the convulsion is brought about by the urea in the blood (or rather the carbonate of ammonia into which the urea is resolved) having acted as a *stimulus* to a vital property of irritability in the nervous system or elsewhere. And what may be said of the condition of the vascular system in the convulsion connected with uræmic poisoning is also more or less applicable to the cases where convulsion happens in persons whose blood is loaded with bile or other matters which ought to be eliminated.

And thus, convulsion in all its forms, epileptic and epileptiform, hysterical, choreic, and the rest, is found to be associated with a state of the vascular system which is very far removed from hyperæmic or feverish activity—a state in which the supply of *red* blood to the system is either arrested altogether or greatly interfered with—a state in which it is easy to believe that the blood must have acted in preventing rather than in provoking convulsion, the case in all particulars being one which is in perfect accordance with the conclusions arrived at in the remarks upon the work

of the blood in vital motion in the physiological part of the argument.

2. In hysteria the patient is at most semi-conscious during the paroxsym, and in a bad bout of choreic agitation there is no very great difference in this respect. In both cases the will is almost altogether in abeyance. And certainly the mental state which is most characteristic of the inter-paroxsymal period in both hysteria and chorea is one which is the reverse of strong-mindedness—a state of nerveless unrest at the best.

Nor are the disclosures of pathological anatomy in chorea such as to connect this malady with anything like an inflammatory condition in one or other of the great nerve-centres.

In half the cases, perhaps, traces of inflammation, more or less vague, and always of very uncertain seat, are met with in the brain or spinal cord, one or both, and quite as frequently in the cord as in the brain; but in the remaining half the most careful search fails to detect them. Traces of inflammation in these parts are not always present; this is plain; and to be ever absent is in itself a certain proof that the inflammation which left them cannot be regarded as the cause of the chorea. Indeed, there is reason to believe that certain parts of the brain, more especially, perhaps, the thalami optici and the corpora striata, with the grey matter of the convolutions attached to them (the island of Reil), instead of being inflamed, are actually starved for want of blood by the plugging of the minute vessels arising either, as Dr. Kirkes pointed out, from the passage into the arterial system of minute warty vegetations that had become

detached from the cardiac valves (and hearts with valves covered with such vegetations are so common in chorea as to have got the name of choreic hearts), or else, as Dr. Bastian supposes, to white blood corpuscles, altered somewhat, and cohering to the walls of the vessels. In point of fact, the pathology of chorea is, to say the least, quite as much in accordance with the notion of certain parts of the brain being starved for want of blood, from the vessels being plugged in one or both of these ways, as with the notion that these or other parts are inflamed. Nor is there any difficulty in accounting for the traces of inflammation which are undoubtedly met with in some of these cases. On the contrary, it is only necessary to suppose that they are the effect of the disease, and not the cause—that, in cases where they are present, the disease has been prolonged until—in consequence of the vaso-motor nerves being at length exhausted or paralyzed by the continuance of the state of over-action beyond a certain limit—the time has arrived in which the vessels of the part pass out of the state of contraction which belongs to the stage of “irritation” into that of congestion and inflammation. Nay, it is quite conceivable that in the very cases in which these traces of inflammation are met with, the choreic symptoms may have been mitigated when the inflammation was established, for all the evidence so far—and there is more to the same effect further on—goes to show that the change to be expected under these circumstances is this, and no other.

And, as with chorea, so also with hysteria or any other disorder in which convulsion of a non-epileptic

character is a symptom, there is really nothing in any appearance after death to make it necessary to modify this conclusion in any particular.

Convulsion is not a common symptom of inflammation of the brain or its membranes. Now and then, in children especially, it may happen at the onset of this disorder, in the cold stage before the hot stage, or at the end of the disorder, in the stage of collapse after the hot stage, when the patient has all but ceased to strive in the "struggle called living;" but, so far as my experience goes, it never happens during the time when general febrile reaction, with determination of blood to the brain, is fully established.

Neither does convulsion find a place among the symptoms of acute mania. Acute mania may be a consequence of convulsion, and convulsion may return when the maniacal excitement has subsided, but the convulsion and the acute mania are not really concurrent phenomena. Indeed, the simple fact appears to be that here also the convulsion is incompatible with anything like active determination of blood to the brain.

And in the case where it may happen to be associated with cerebral apoplexy convulsion may have to be referred, not to "active determination of blood to the brain," nor yet to engorgement of the cerebral veins, but to the pressure or other damage caused by the extravasated blood. It may well be that convulsion is thus brought about, if only the apoplectic effusion be on or in some part of the brain which has to do with automatic or reflex movement; it can scarcely be that it has any

real connection either with "active determination of blood to the brain," or with engorgement of the cerebral veins. A sufficient contradiction to the notion that it is connected with "active determination of blood to the brain" is supplied in the brief comments on inflammation of the brain which have just been made: and a contradiction to the other notion, that it is connected with engorgement of the cerebral veins, is not far to seek. In whooping-cough, where these veins are often congested to a very high degree during the paroxysm, when convulsion happens it is, not at this time in particular, but rather when the face is pale and the patient spent, and either sleepy or else asleep. In extreme congestion of the lungs, also, where these veins are greatly gorged with dark blood, the most likely consequences are dreamy sleepiness, stupor, coma it may be, seldom or never convulsion. And in cases where extreme venous congestion of the brain is brought about by straining, or in some similar way, the symptoms are coma simply, not coma and convulsion. In these cases, indeed, it is as it is shown to be in the experiments in which the external and internal jugulars of rabbits were tied by Drs. Kussmaul and Tenner (p. 97), and in which the operation resulted, not in convulsion, but in stupefaction, with, occasionally, some slight grinding of the teeth.

Nor do I find anything in the relations of convulsion to the state called "exalted or morbid irritability" which is at all calculated to invalidate these conclusions. For what is this state? It is not inflammation: it is not fever: it is some undefined and negative state occurring frequently in teething, in worm-disease, in uterine

derangement, in spinal irritation particularly, and in many other cases—a state in which the patient is irritable, easily over-balancing on the side of excitement, or on that of depression, as the case may be—a state in which exhaustion is very readily brought about, and for which *nervous exhaustion* would seem to be as good a name as any—a state, in fact, which is more readily accounted for on the supposition that certain nerve-centres are starved for want of blood, than upon the contrary supposition that they are over-fed with blood. In a word, there is nothing in the facts which together go to make up the idea of “exalted or morbid irritability” which is at variance with the view here taken of epilepsy and convulsive disorders generally.

Looking back at the history of epilepsy and other convulsive disorders, as thus delineated, I see nothing to justify the notion that the convulsion is brought about by an over-abundant development of nervous influence in some great motorial nerve-centre consequent upon a hyperæmic state of that centre, and much to lead me directly to a contrary conclusion. I have to do, as it seems to me, with a state of anæmia which must necessitate a defective development of nervous influence in that centre and which may necessitate a defective development of electricity there also. I have to do, as it seems to me, with a state of things which allows me to believe that the convulsion is brought about by the removal of something which had antagonised a state of action in muscles and nerves—that, in consequence of want of arterial blood, there may be a failure of the

charge with which the nerve-centre is ordinarily charged, that, owing to this failure, there may be a *downward* movement of electricity towards a lower level of equilibrium, and that, as long as this movement continues, there is in and around the fibres of nerve and muscle, a development of the instantaneous faradaic currents which are directly concerned in throwing nerve and muscle into the state of action. Upon the supposition that there is a failure of charge consequent upon want of blood—this follows as a matter of course. The case is strictly in accordance with the premisses. But not so necessarily if it be supposed that there is augmentation of charge consequent upon an over-abundant supply of blood. In this case there may be an *upward* movement of electricity until the higher level of equilibrium is reached, and this movement may issue in the development of instantaneous faradaic currents, but it does not follow that the nerves and muscles will be thrown into a state of action by these currents. On the contrary, it is quite possible, and by no means improbable, that the action of these currents may be counteracted by that of the excess of charge which is supplied to the nerves and muscles, and which is caused by the excess of blood in the part. At all events there is, so far as I can see, no reason to believe that this question has to be dealt with in the problem under consideration, for the facts with which I have here to do go to show that convulsion is connected with a failure of nervous influence and nerve-electricity consequent upon an insufficient supply of blood to the nerve-centre, and not with the contrary state of things. Indeed, so far as I can see, there is nothing in the history of

convulsion in any one of its various forms which is not strictly in accordance with the history of vital motion as delineated hitherto.

XXXVII.

That the history of tetanus and other forms of spasmodic disorder, as set forth in the condition of the vascular and nervous systems, is strictly in accordance with the history of convulsion, and with the physiological premisses.

I.

1. A case of tetanus is fresh in my memory in which the state of the pulse as to power seemed to be inversely related to the severity of the spasm and to the heat of the skin ; and this case, so far as I know, is in no way exceptional. And, certainly, I have met with several cases in which the liberal administration of ether and brandy had the effect at one and the same time of invigorating the pulse, of mitigating the spasm, and of reducing the temperature.

There is also as it would seem some reason for believing that in trismus and tetanus the blood is more or less damaged by a virus as it is damaged by strychnia (p. 94), or in hydrophobia. Three years ago I saw in consultation with Mr. Quain a fatal case of trismus in which death seemed to be due to this cause rather than

to any other. The patient, a young lady, recently married, had been operated upon for piles a fortnight previously. At our first interview she laughed at the idea of a consultation being thought necessary in her case, and would have it that the stiffness of the muscles of her jaws which for half-a-dozen hours had prevented her from putting out her tongue or using her teeth easily, would pass away presently; and, save for this stiffness, she seemed to be quite well. In the course of the night following—I had been called out of bed to see her—I found her afraid to go to sleep lest she should dream again a dream in which she was in the agony of drowning, and I had to spend some time in consoling her. Next day, when Mr. Quain and I met again, she smiled at what she called her folly in the night, and was in very much the same state as that in which she was when we saw her first together—not worse apparently. The next night I was again called up to see her, and though I had not far to go and went as quickly as possible, I was too late to find her alive. After remaining quietly asleep for four or five hours she awoke with a start, sat up in bed, tore her night-dress open, pointed to a bottle containing *sal volatile* which stood on a table close by, took the draught which was given her, lay down, and, after lying quietly for half an hour, died unexpectedly. The horrified husband only said that she was breathless and agitated for a few moments after waking, and that then it seemed to him as if she were wishful to go to sleep again. Death happened indeed exactly as it did in two cases of hydrophobia and in two cases of trismus which have been under my care during

the last six years; and the impression left upon my mind in each of these cases was that death had been brought about by damage done to the blood rather than by damage done to the nervous system, the damage done to the blood being due to the working of a virus which had then got the upper hand. And I find much to deepen this impression when I call to mind the cases of trismus and tetanus which have come under my notice at different times, for in the greater number of these cases death was preceded by a state of exhaustion with little or no pain and spasm, which state seemed to be more easily accounted for by referring it to a loss of power in the circulation from a morbid change in the blood, than by referring it to the exhaustion arising from a continuance of pain and spasm.

Nor does the increased heat which may and often does happen in tetanus lead necessarily to a different conclusion, for, as the following abstracts of three of the cases recorded by Dr. Wunderlich will serve to show, the temperature may rise, not only in the moribund state, when the spasm is likely to be greatly mitigated and when the circulation is carried on in a very faltering manner, but also for some time after death, when the blood is altogether stagnant.

The patient in the first of these cases was a butcher, aged 29. The disorder, which was idiopathic or rheumatic tetanus, without any peculiarity as to symptoms, ran its course in five days. There was some delirium, with marked abatement of the spasmodic symptoms for six and thirty hours before death, and towards the end this state differed scarcely if at all from extreme ex-

haustion of mind and body. After death the spinal cord here and there was found to be injected and considerably disorganized, and that was all. The weather was hot—the thermometer in the ward at the time of death standing at 77° Fahr.—and putrefaction was unusually rapid. During the first two days the temperature was not taken; afterwards, the readings of the thermometer were these:—

<i>Before death</i>	July 24, 1861	. . .	102° Fahr.
	" 25 "	. . .	102°
	" 26 "	9 a.m.	104.45°
	" " "	6 p.m.	103.55°
	" " "	9.20 p.m.	110.1°
<i>At death</i>	. . .	" " " 9.35 "	112.55°
<i>After death</i>	2 minutes		112.77°
	5 "		113°
	20 "		113.22°
	35 "		113.55°
	55 "		113.67°
	60 "		113.65°
	70 "		113.22°
	90 "		113°
	100 "		111.8°
	6 hours		106.25°
	9 "		104°
	12 "		102°
	$13\frac{1}{2}$ "		101°

The second case was that of a man, aged 20, who died of traumatic tetanus on the tenth day. Up to

twenty-four hours before death the spasms were well marked, and the mind was quite clear; afterwards, and especially during the last six hours of life, unrest, talkativeness, jactitation, and slight delirium, were the most prominent symptoms. The appearances after death agreed very closely with those noticed in the last case; and the notes of temperature, which are the following, do not differ materially :—

Three hours <i>before death</i>	. . .	105·8° Fahr.
<i>At death</i>		107·6°
Ten minutes <i>after death</i>		107·8°
15 " " "		108°
20 " " "		107·8°
48 " " "		106·45°
58 " " "		105·8°
68 " " "		105·35°
80 " " "		104·45°
95 " " "		103·55°
120 " " "		101·75°
240 " " "		99·3°

The patient in the third case was a young woman in whom on the second day the symptoms of pneumonia became associated with those of idiopathic or rheumatic tetanus, and who died on the third day. As the symptoms of pneumonia became developed the symptoms of tetanus subsided, and from the post-mortem appearances as well as from the symptoms during life, death seemed to be brought about by the disorder of the lungs rather than

by the disorder of the spinal cord. In this case the temperature was:—

3½ hours <i>before</i> death	. . .	102·85° Fahr.
10 minutes <i>after</i> death	. . .	103·32°
21 " " "	. . .	103·55°

Other evidence to the same effect remains in the background, but this is enough to show that the increase of heat in tetanus about which I am speaking has to do with a state of things which is marked by death rather than by life—that the cause may have to be sought, not in over-activity of the circulation, but in a state of passive engorgement of the minute vessels consequent upon vaso-motor paralysis. It is easy to see that vaso-motor paralysis, brought about by damage done to vaso-motor centres in the spinal cord, may lead to annihilation of the “capillary force” which helps the blood along in its course, and thence to the increased development of heat through uncontrolled molecular changes in and around the blood which lags behind in the minute vessels. At all events the fact remains that the temperature may continue to rise in tetanus for some time *after actual death*, and it is with the fact only, and not with the explanation, that I have to do now, for as long as the fact remains I am at liberty to maintain that the increase of heat in tetanus does not really invalidate the statement which I ventured to make in starting—that the disposition to spasm in tetanus appears to be inversely related to the activity of the circulation.

2. There is also no good reason to connect the spasms of tetanus with inflammation in the spinal cord or elsewhere. "Serous effusion with increased vascularity," says Mr. Curling, "is generally observed in the membranes investing the medulla spinalis, and also a turgid state of the blood-vessels about the origin of the nerves," and the same changes may be met with within the cranium, but not in so marked a degree, or so frequently. Out of 70 fatal cases collected by Mr. Curling, there were only two in which changes in the nervous system, unequivocally the result of inflammatory action, were discovered after death, and these two were cases where the back had been injured by a blow or a wound, and where the signs of inflammation found after death were, to say the least, as easily referrible to the injury as to the tetanus. And in no one of the many cases in which the cords of animals killed by strychnia were examined by Majendie, Orfila, and Ollivier, was there any perceptible organic lesion, inflammatory or other. Nor do recent microscopic investigations into the condition of the spinal cord in tetanus bring to light any clearer signs of inflammatory changes in this organ. Lockhart Clarke* found the vessels injected, and the substance of the cord in a state varying from simple softening to complete solution, the softened or dissolved portions forming irregular "areas of disintegration" filled with the *débris* of blood-vessels and nerves, or with a finely granular or perfectly pellucid fluid. These areas of disintegration were chiefly in the grey substance around the canal, but they were also in the white sub-

* "Med. Chir. Trans.," vol. xlviii, 1865.

stance. They were, in fact, in no one part particularly and exclusively. Here and there were extravasations of blood and "other exudations," but pus corpuscles are not mentioned. "In the walls of the blood-vessels," Lockhart Clarke says, "there was no morbid deposit, nor any appreciable alteration of structure, except where they shared in the disintegration of the part to which they belonged; but the arteries were frequently dilated at short intervals, and in many places surrounded, sometimes to a depth equal to double their diameter, by granular and other exudations, beyond and amongst which the nerve-tissue, to a greater or lesser extent, had suffered disintegration." And elsewhere he adds, "the appearances met with are exactly similar in kind to the lesions or disintegrations which are found in various cases of ordinary paralysis, in which there has been little or no spasmodic movement." In short, the cord is broken up, as at a certain time in all cases it is broken up by ordinary putrefaction, and the dilated vessels, and, certain exudations of blood and serum excepted, this is all that is noticed. The facts point, not to inflammation, but to disintegration. And what Lockhart Clarke found in these cases is substantially the same as that found by Dr. Dickinson in the one case recorded by him,* for the only peculiarity in this case is in the presence, in addition, of an excessive quantity of a translucent, structureless, or finely granular, carmine-absorbing material, evidently the sero-fibrinous plasma of the blood, which had escaped from the minute arteries into various parts of the substance of the cord where the

* "Med. Chir. Trans.," vol. li, 1868.

nerve tissue had broken down, and which lay in pools here and there between the cord and its membranes. Nor is a contrary conclusion to be drawn from the condition of the sympathetic ganglia or of the nerves at the wound where there is a wound. In some cases there is, as Mr. Swan points out, marked injection of the minute vessels supplying the sympathetic ganglia, especially the cervical and semilunar, but these cases are few in number compared with others in which all signs of the kind are absent. In some cases, also, there may be traces of inflammation in the wound, and these cases are more numerous than those in which such traces are met with in the spinal cord, or other great nerve-centre; but here again these traces, instead of being constant, are not even common. In the great majority of cases, indeed, the wound, if there be one, is, to all appearance, perfectly healthy, and healing or healed. In a great number of cases, in the majority perhaps, the primary wound was completely healed and almost forgotten when the symptoms of tetanus made their appearance, and Dr. Rush, who had extensive opportunities for observation in the military hospitals of the United States, and who was unquestionably a most competent observer, remarks that there was invariably an absence of inflammation in the wounds causing the disease. John Hunter also says: "The wound producing tetanus is either considerable or slight. * * * When I have seen it from the first, it was after the inflammatory stage, and when good suppuration had come on; in some cases, when the wound had nearly healed, and the patient was considered healthy. Some have had locked-jaw after the healing was com-

pleted. * * * When tetanus comes on in horses, as after docking, it is after the wound has suppurated and begun to heal."

Again, the true history of inflammation of the spinal cord or its membranes, would only seem to lead to the same conclusion by a different way, that is, by showing that where this inflammation is really present the symptoms are not those of tetanus.

Acute general spinal meningitis is often obscure enough in its symptoms at first, and this obscurity is generally increased by the presence of head-symptoms in one form or another. Pain along the spine and in the extremities, produced by movement, must, as I think, be regarded as the most prominent symptom of all. It may be confined to the region of the spine, but more generally it shoots into the extremities, into the legs especially. As a rule, it does not shoot belt-wise round the trunk. It is brought on by any movement of the trunk, and in great measure at least it may be prevented by avoiding such movement. It is brought on also by moving the extremities, and in this case it is very likely to begin in the limbs and shoot thence to the spine. It seems to depend, in part at least, upon the same cause as the pain of pleurisy, viz., the dragging of an inflamed, and, for that reason, exquisitely tender, serous membrane, and its character is certainly more like that of pleurisy than that of rheumatism (to which latter pain it has been likened), for it occurs in the same sharp, sudden, breath-stopping catches. Along with these fits of pain are fits of muscular stiffness in the painful parts, about which latter fits it is

desirable to have very clear notions. It is usual to regard this stiffness as analogous to the spasm of tetanus ; it is necessary, as it seems to me, to look upon it as expressing an instinctive act of muscular contraction, of which the object is to prevent pain by preventing the movements which produce pain. The spine and extremities cannot be moved without causing pain : the stiffness prevents the pain by preventing the movement : this would appear to be the true view. This view, originally applied by Dance to the explanation of muscular stiffness in a case of acute spinal meningitis, observed by himself, and recorded by Ollivier, would seem to apply with the same exactness to all cases of the kind. Indeed, as I believe, there can be no greater mistake than to confound the stiffness in question with the spasm of tetanus, or to regard, with Ollivier, spasm "*comme indiquant positivement la phlegmasie des membranes de la moelle,*" for the rule is, that as long as the patient can keep still, so long is he, comparatively at least, free, not only from fits of pain, but from fits of stiffness also, the intervals of freedom being sometimes of considerable length, even for days—a rule which is very different from that which obtains in tetanus. The differences between acute spinal meningitis and tetanus are indeed so marked as to make a mistake in diagnosis somewhat difficult. Muscular rigidity continuing without intermission when once set up, together with occasional fits of painful spasm, are the two characteristic symptoms of tetanus. The muscles, even in the intervals between the spasms, are very much as they are in rigor mortis, curiously hard. They are also somewhat tender. The spasms

become more frequent as well as more violent and painful as the disease progresses, at the worst recurring every ten or fifteen minutes and continuing from one to two and a half minutes, and being now and then violent enough to crack or even displace a tooth or two, or to snap a thigh bone, or to tear across a big muscle like the *psoas* or *rectus femoralis*. As a rule, the rigidity shows itself, first in the muscles of the jaws, then in those of the neck and back, and then in those of the face and limbs, the upper limbs being usually affected before the lower: and in the end the only voluntary muscles (the involuntary muscles are not affected) which are found to remain limber are those of the tongue, throat, and hands. Sometimes, as in some cases of rapidly fatal tetanus from strychnia-poisoning, there may be no trismus, and the disorder may manifest itself chiefly in strong spasms which keep the arms and legs apart and on the stretch, and which clench the fingers; and sometimes, as after an amputation, the rigidity may begin in the muscles of the stump; but usually the muscles set in the order which I have indicated, and *opisthotonus*, to a certain extent, must be regarded as scarcely less constant than trismus. In acute spinal meningitis, on the other hand, the jaw, if it be set at all, is set rather at the close of the disease, and then only to a very inconsiderable degree, and muscular rigidity and spasm are neither constant nor conspicuous phenomena. In acute spinal meningitis, indeed, the occasional muscular rigidity and the seeming spasms are in great measure voluntary or semi-voluntary acts to prevent the pain in the back and limbs which is produced by movement, and the muscles are relaxed—

with the exception perhaps of those behind the neck—as long as the patient can keep perfectly still. *In a word, the true involuntary fits of spasm and the permanent muscular rigidity which are constant and characteristic phenomena in tetanus are not met with in acute spinal meningitis.*

A case of acute spinal meningitis which was admitted into one of my beds in the National Hospital for the Paralysed and Epileptic only a few days ago corroborates in a remarkable manner what I have just been saying about the history of this disorder. The patient was a married woman, 42 years of age, who for three years had been an out-patient under the care of my colleague Dr. Gowers for bulbar paralysis with a certain degree of disseminated spinal sclerosis. The illness began eight years ago with difficulty in speaking and swallowing, and with weakness in the upper limbs. Two years later this weakness extended to the lower limbs. Up to Christmas Day last she could walk about after a fashion, and do most things for herself in which she had to use her hands. On Christmas Day, after being violently sick, she became so weak as to have to keep her bed for a week. Then she rallied a little, and was able to be about for a short time every day. On the 23rd of January last, when she became an in-patient, she was very weak and very feverish, her temperature late in the evening being 104·6° Fahr., but she did not complain of pain anywhere, or of anything but weakness. For the next four days she remained in very much the same state. On the 27th her only answer when spoken to was "yes" or "no," and she had, in fact, become semi-conscious. She

had perspired a good deal in the night, but there had been no kind of fit. On the 30th, without any material change in the symptoms, she died. While in the hospital the pulse ranged from 120 to 140: the respiration from 44 to 64: the temperature from 99.7° Fahr. to 106.6° Fahr., varying greatly from day to day, and reaching its maximum, 106.6° Fahr., in the moribund state. During life nothing in the shape of disease was found out to account satisfactorily for the high temperature. After death the enigma was solved, not by the state of the lungs, which were only a little congested posteriorly, not by the state of the kidneys, which were small and granular, with adherent capsules, not by the state of the spinal cord, although here the arteries of the lower part were found to be considerably injected, but by the state of the spinal membranes, which opposite the six lower dorsal vertebræ were deeply injected and thickly covered externally (not internally) by blood-stained, jelly-like lymph, and by the inside of the spinal canal corresponding to the inflamed membranes being much redder than it ought to be. No microscopic examination of the great nerve-centres has yet been made, but this is not necessary in order to make it evident that in the few days immediately preceding death there had been a state of acute spinal meningitis which had not betrayed itself during life by pain or spasm or any other symptom, and that it was not necessary to inquire further in order to see why the temperature rose so high as it did during life. According to the premisses there is nothing unintelligible in the facts which have to be dealt with. Indeed, after what I have said already, I

may fairly leave this case to tell its own story without further comment.

Among the symptoms of acute general myelitis, no place is found for trismus, or spasm in any form. Paraplegic anæsthesia, ushered in often by tingling or some other disagreeable sensation in the parts which eventually become anæsthetic; paraplegic paralysis, preceded frequently by great restlessness of the legs; a feeling as of a tight belt or garter or armlet; absence of pain in the spine or extremities—of pain produced by movement especially; retention of urine; involuntary stools; absence of spinal tenderness; increased sensitiveness to differences of temperature, by which moderately warm or iced water gives rise to a feeling of burning over the vertebra which marks the upper limit of the myelitis; annihilation of reflex excitability in the paraplegic parts; priapism; comparative voicelessness; impeded respiration; engorgement of lungs and other viscera; tendency to bed-sores; loss of electro-contractility and electro-sensibility in the paralysed muscles; absence of "head-symptoms;" absence of fever; absence of trismus, or any other convulsive or spasmodic symptom—these are the points which call for special notice in the history of general acute myelitis. The symptoms are very different from those of spinal meningitis—so different as to make it difficult to confound them, if only moderate care be taken to realise them. In spinal meningitis, the most prominent symptom is pain in the back and extremities, produced or aggravated by movement; in myelitis, pain of any kind has scarcely a claim to be reckoned among the symptoms, pain produced by move-

ment certainly not. In spinal meningitis the sensibility is somewhat exalted ; in myelitis it is abolished. In spinal meningitis there is muscular weakness, and the movements are fettered by pain, but there is no true paralysis ; in myelitis paralysis is the symptom of symptoms. In spinal meningitis there is occasionally a state of muscular stiffness, half voluntary in its character, of which the object is to prevent certain movements which give rise to pain. In myelitis there is, for the most part, an utter absence of any symptom akin to spasm or tremor, or convulsion. Ollivier, it is true, speaks of continuous contraction of the limbs as being met with, "*assez ordinairement*," in chronic myelitis ; but the cases cited by this excellent observer do not substantiate this statement. Thus, out of nineteen cases of myelitis, complicated and uncomplicated, acute and chronic, there are three only in which these contractions were present, and not one of the three can be correctly cited as a case in point. In one of the three (89), the sensibility was intact, and the disease of the cord confined almost exclusively to the anterior columns ; in the second (93), there was obtuse sensibility, and the disease was chiefly in the grey matter ; and in the third (94), sensibility remained, and there was no post-mortem examination to show what the disease in the cord really was. In each one of these cases, also, there were head-symptoms which do not figure in uncomplicated myelitis. Dr. Brown-Séquard says—"When the dorso-lumbar enlargement is inflamed, reflex movements can hardly be excited in the lower limbs, and frequently it is impossible to excite any. On the contrary, energetic reflex move-

ment can always be excited, when the disease is in the middle of the dorsal region, or higher up." And again, when speaking of the reflex convulsions which may happen in the cases where the inflammation is in the middle of the cord, or higher up, he says, "convulsions do not take place at the beginning of the inflammation, but some time after, and they recur by fits for months and years after." And this is precisely what does happen. The truth, in fact, would seem to be, that these reflex spasmodic movements must be referred, *not* to inflammation in the lumbar enlargement of the cord, nor yet to inflammation higher up the cord, for in this latter case, to enforce what has just been said by repeating it, the "convulsions do not take place at the beginning of the inflammation, but some time *after*, and they recur by fits for months and years *after*." They happen, as it would seem, *after* the inflammatory disorganization has interrupted the continuity of the cord, and produced a state of things analogous to that witnessed in the guinea-pig whose cord has been cut across experimentally—a state of things in which increased reflex excitability in the paralysed parts is one of the concomitants. Nor is a different conclusion to be drawn from the occasional presence in the paralysed muscles of a state which is analogous to, if not identical with, the "late rigidity," of Todd. This "late rigidity" is very different from "early rigidity." In "early rigidity," the electro-motility of the muscles is increased, and the muscles relax during sleep, and to a less degree under the influence of warmth. The contraction is evidently of the nature of spasm. In "late rigidity," on the contrary, the muscles are wasted,

their electro-motility is reduced to a minimum, and sleep and warmth scarcely tell in causing relaxation. This form of contraction, indeed, if not identical with rigor mortis, is, as it would seem, more akin to this state than to spasm. In a word, absence of spasmodic symptoms would seem to be the rule in all cases of myelitis, acute or chronic. In children, it is true, myelitis may be ushered in by convulsions—in which case the convulsion may be supposed to take the place of the rigor which may usher in the same disorder in adults, and to belong to the precursory state of irritation, and not to the stage of actual inflammation—but, even in children, unless there be some meningeal complication along with the myelitis, this preliminary convulsion would seem to be of rare occurrence.

Prolonged muscular contraction, on the other hand, is one of the many symptoms belonging to the state which is known under the name of spinal irritation. The lower extremities appear to be the parts most commonly affected, one or both of them; but the upper extremities can claim no exemption, nor yet the muscles of the jaws and neck, trismus and torticollis being among the forms it may take. This contraction, which is generally painless, may be prolonged for weeks or months continuously, even during sleep, or it may have occasional intermissions of uncertain duration; and the attacks, secondary as well as primary, are usually found to begin and end suddenly and unexpectedly. It cannot well be confounded with tetanus; it may in some instances be difficult to distinguish between it and the somewhat vague disorder to which Trousseau gave the name of tetany (*tétanie*). In tetany as in tetanus, the con-

traction is painful, but in tetany the order in which the body is attacked is different from that which is observed in tetanus, centripetal not centrifugal, first the extremities, then the trunk or head, the contraction in fact being confined to the extremities except in cases of unusual severity. In the way in which it affects the extremities first, and often exclusively, the contraction of tetany agrees with the contraction under consideration, but in other respects it differs. It differs especially in being ushered in, and accompanied by, symptoms which do not seem to be part and parcel of simple spinal irritation, viz., tingling with some degree of anæsthesia, and also (so it is said) in the form of the hand being peculiar when the contraction is in this part, this form being like that which is taken in order to put on a tight glove, and also in the possibility of bringing on the contraction when it is absent by firm pressure upon the principal arteries and nerves of the part in which the contraction is about to be manifested. It may be questioned, however, whether there are absolutely fixed lines of division between these different forms of prolonged muscular contraction, and whether the differences which exist may not be accounted for as the result of different degrees of "irritation," affecting, it may be, different parts of the spinal cord. It may be questioned, also, whether a sufficient case is made out for describing tetany as a distinct disorder, and whether it is not rather a form of spinal irritation complicated with some graver spinal disease—myelitis, meningitis, or congestion—in varying proportions. The association of tingling and numbness with the prolonged contraction is, as it would seem, a

reason for so questioning. At any rate be its significance in tetany what it may, prolonged contraction in various sets of muscles must be looked upon as a not unfrequent symptom in simple spinal irritation—a state which points, not to organic disease, but to functional disorder, of which one most characteristic feature is the way in which one symptom or group of symptoms may change, and change suddenly, into another symptom or group of symptoms. In spinal irritation, indeed, it is now this disease which is simulated, now that, there being scarcely any disease which may not be copied. At one time the head is affected, at another the chest, at another the abdomen or the extremities, and the only thing constant among these ever-shifting phenomena appears to be the presence of spinal tenderness, of which the seat changes from one part to another as this or that set of spinal nerves is chiefly affected. The pain or disorder of any particular organ is altogether out of proportion to the constitutional disturbance; and the local tenderness of the spine has plainly nothing to do with a cause so mechanical and fixed in its nature as inflammation. In point of fact, the subjects of spinal irritation, with few if any exceptions, may be spoken of as hysterical, hypochondriacal, or nervous. They have that *nervous constitution* which Whytt, following in the steps of Sydenham, showed to be the common basis of hysteria and hypochondriasis, and of which the signs are sufficiently obvious. First in order among these signs in that sign which Sydenham regarded as pathognomic of hysteria and hypochondriasis—a proneness to pass, under or after strong emotion, large quantities of pale,

limpid urine. Then come other signs scarcely less characteristic: proneness to tenderness, not only in some part of the spinal column, but also in the epigastrium and left hypochondrium—*le trépiéd hystérique* of Briquet; proneness to sudden and distressing flatulent distension of the stomach and bowels, with loud rumblings and explosions, and with a feeling as of a ball rolling about, first in the left flank, and then mounting, or tending to mount, into the throat, where it gives rise to a sense of choking and to repeated acts of swallowing; proneness to bursts of laughing or crying and sobbing; proneness to yawning, sighing, and stretching of the arms—which phenomena are rarely ever present in acute organic disease; proneness to fits of convulsive agitation and struggling. Then come a promiscuous series of signs, among which are—erratic pains of a neuralgic character, breathlessness, nervous cough, palpitation, throbbings in the temples, epigastrium, and elsewhere, “flushes and chills,” syncope, hiccough, nausea, vomiting, aversion to, or unnatural craving for, food, heartburn, oppression at the præcordia, languor, debility, fidgetiness, tremulousness, vertigo (especially on rising hastily), ringing in the ears, fancifulness, a habit of romancing, undue lowness of spirits or the contrary, and other symptoms whose name is legion. Nay, not only is the name of these symptoms legion, but there is ever going on a process of mutual metamorphosis in the symptoms themselves; and, in short, it is this very variability and mutability of the symptoms which must be looked upon as the great characteristic of the nervous constitution, with which, and not with any inflammatory or structural

change, the prolonged muscular contraction which has to do with spinal irritation is associated.

A like inference is also to be drawn from the fact that the traces of inflammation after death which may be met with in cases of tetanus are, not in the spinal cord exclusively, but in various parts of the brain, in the nerves, and in other parts of the body as well. There is, in truth, a vagueness in the seat of these traces which makes it certain that inflammation in any one nerve-centre cannot be looked upon as essential to the spasm, and which suggests the notion—which, as will be seen presently, arises more particularly in connection with the history of the spasm in hydrophobia—that the inflammation which has left these traces may have had to do with some secondary, and, it may be, depurative process, by which the system has striven to rid itself of some virus, rather than with the spasm.

II.

I. As in tetanus, so in the other forms of spasmodic disorder about which I have yet to speak, there is reason to believe that the spasm is associated with a condition of the vascular system which is the very reverse of anything like over-activity, and that the increased temperature met with in some cases must have the same significance as in tetanus. During the attack of catalepsy the appearance of the patient is so corpse-like that it may even be necessary to put the ear to the chest to know of a certainty that the heart has not ceased to beat. In cholera the cramps are coincident with a state closely approaching to pulselessness, and any increase of

temperature before or after death is evidently of the same nature as the analogous phenomenon in tetanus. In hydrophobia the circulation flags from the very beginning; and in spasmodic ergotism there is no evidence of vascular excitement throughout the whole course of the disorder. And, certainly, no contrary conclusion with respect to the state of the circulation is to be drawn from the history of the seizures of cramp in the leg and elsewhere which occur so frequently in old people, and in those in whom old age is anticipated by chronic degeneration of the brain.

In hydrophobia, as in tetanus, it appears to be the rule for the spasm to gain ground as the pulse loses in power, for on analysing the histories of a considerable number of cases, I find that there was less agitation, less convulsion, less spasm, when the state was less feverish than in the ordinary run of cases. Nor is a different conclusion to be drawn from the history of spasm as set forth in whooping cough. For the fact is simply this—that the whoop, which is the audible sign of the spasm, does not make its appearance until the febrile or catarrhal state has passed off; that it disappears if pneumonia, bronchitis, or any other inflammatory disorder be developed in the course of the malady; and that it returns again when the inflammation has departed. Taken by itself, this evidence, it is true, may not amount to much; taken in connection with what has gone before, and with what has yet to be set forth, it helps not a little to strengthen the conviction that spasm, like convulsion, is antagonized rather than favoured by an excited condition of the circulation.

2. As in tetanus, too, the vagueness in the seat of the inflammation which may be developed in the course of various other spasmodic disorders would seem to show that spasm is not to be regarded as a symptom of inflammation of the spinal cord, or of any other part of the nervous system. Thus, in 46 cases of hydrophobia, of which the histories were carefully analysed by my brother, Mr. J. Netten Radcliffe,* "the morbid appearances were in the dura mater in 8, in the arachnoid membrane in 10, in the pia mater in 16, in the velum interpositum in 2, in the choroid plexus in 12, in the cerebral hemispheres in 28, in the spinal cord and membranes in 18, in the pons varolii and medulla oblongata in 4, in the tongue in 8, in the palate in 3, in the salivary glands in 2, in the pharynx in 19, in the æsophagus in 16, in the stomach in 20, in the intestines in 6, in the larynx, trachea, and bronchial tubes in 31, in the ultimate ramifications of the air passages in 24, in the heart in 4. These lesions consisted in every gradation of injection of the blood-vessels, from the slightest blush to the most vivid red or dark black congestion; of alteration in the consistency of the tissues, principally softening; of effusion of blood, and certain products of perverted secretion and nutrition. In several of the cases the lesions were of such a character that they have been classed with those resulting from common idiopathic inflammation; in a greater number of cases they were of that character which is found in structural changes occurring in asthenic conditions of the system." Now, this vague-

* "Lancet," Sept., 1856.

ness in the seat of these inflammatory and other structural changes is a very curious and significant fact—a fact which, perhaps, more clearly than any other single fact, is calculated to show the true relations of inflammation to spasm. It is calculated to show that inflammation of any one particular nerve-centre cannot be essential to the existence of the spasm. It is calculated to show that the cause of the inflammation may be as general as the cause of the obscure febrile condition which may be developed in the course of the disorder—that, in fact, it is little more than accident which fixes the seat of the inflammation in one part of the nervous system rather than in another, or in one part of the body rather than in another. In the case of hydrophobia, indeed, it is calculated to put any inflammation which may be developed in the course of the malady in the position of a depurative process—a process which, as in the inflammation developed in connection with the fever of small-pox, is intended to rid the system of a morbid virus. And thus it may be that spasm is connected *not* with a state of inflammation in any part of the nervous system or elsewhere, but with a state which may, or may not, issue in such inflammation—a state to which the name of “irritation” is commonly given, and which is marked, not by relaxation of vessels and hyperæmia, but by contraction of vessels and anæmia; and, so far as I know, there is nothing in the history of any form of spasmodic disorder other than hydrophobia and tetanus which is in any way calculated to exhibit the relations of inflammation to spasm in a different light.

As in convulsion so in spasm the paroxysm would seem to be brought about by failure of charge in some great motorial nerve-centre consequent upon a defective supply of arterial blood to the part, and by the development of instantaneous faradaic currents dependent upon the movement of electricity to a lower level of equilibrium. The only difference is in this—that in the case of spasms the nerve-centres affected are the hindermost portions of the thalami optici, the corpora quadrigemina, a great part of the superior and middle cerebellar peduncles, some undefined portions of the cerebellar lobes, a great part of the crura cerebri, a great part of the pons varolii, the olivary bodies more especially, in part the lateral columns of the medulla oblongata, the upper ends of the anterior columns of the spinal cord, and very probably the entire length of the lateral columns of the cord with the exception of their uppermost extremities. Nor is it altogether unintelligible that permanent contraction should be brought about by injury to one or other part of this nerve-tract,—that it should come on immediately and continue without abatement not only for hours and days but for weeks and months, and that it should be on the same side of the body as the part injured. For it may be that the natural working of the inherent electricity of this tract of the nervous system is especially to keep up a state of muscular tension,—that an injury to any part of this tract may lead to permanent contraction, not by subjecting the muscles to any kind of irritation, but simply by lessening the electrical tension of the muscles and so leaving the muscular molecules more at liberty to yield to the attractive force

which is inherent in their physical constitution,—and that the contraction is on the same side of the body as the part injured because the nerves belonging to one half of the tract in question do not decussate with those belonging to the other half—because, that is to say, the nerves of one half of the tract are distributed to muscles on the same side of the body. Nay it is possible that the diminution of electrical tension thus brought about may have something to do in making the paroxysm of spasm more continuous than the paroxysm of convulsion, for it is easy to see that, owing to this cause, the muscles may be less prompt to get out of the state of contraction when once in it.

And thus, in looking back at the history of spasm as exhibited in these various ways, I find nothing to justify the notion that the spasm is caused by excessive development of nervous influence in the spinal cord, or in any other nerve-centre, consequent upon an overabundant supply of blood to the part, and much which is strictly in accordance with the history of convulsion already set forth, and with the physiological premisses.

XXXVIII.

That the history of tremor in its various forms, as set forth in the condition of the vascular and nervous systems, is strictly in accordance with the history of convulsion and spasm, and with the physiological premisses.

i. THERE is manifestly something altogether uncongenial between tremor, as exhibited in ordinary trembling or shivering, and an excited condition of the circulation. The skin is cold and pale or dusky, and the muscular disturbance is at an end as soon as the state of vascular reaction is established. Indeed, the history of tremor in these forms is in harmony with the history of rigor and subsultus in fever, for here it is found that the rigor, which marks the cold stage of the disorder, departs during the hot stage, and returns again in the form of subsultus when the vascular reaction of the hot stage is at an end.

2. Nor is it possible to connect tremor in any form with an over-abundant supply of nervous influence. In tremulous old age, in paralysis agitans, and in the exceptional form of this disorder in which the tremor shows itself only or chiefly when an attempt is made to stand or move about on the feet, and in which the spinal cord after death is found to be in that state of degeneration to which the name of disseminated spinal sclerosis

has been given, there is manifest deficiency in this supply. And it is not less certain that delirium tremens loses its characteristic trembling if acute head-symptoms and high fever make their appearance in the course of the disorder.

And, therefore, as in the case of convulsion and spasm so in the case of tremor there is good reason to discard the current doctrine of vital motion as exhibited in nerve and muscle, and to cling to the belief that the true view of this motion is that which is set forth in the physiological premisses.

XXXIX.

That the history of neuralgia in its various forms, as set forth in the condition of the vascular and nervous systems, is strictly in accordance with the history of convulsion and spasm and tremor, and with the physiological premisses.

I. IN the neuralgia so frequently met with in agueish districts the state of the pulse and of the circulation generally during the paroxysm is not unlike that which belongs to the cold stage of ague, and these resemblances are further borne out in this—that rigors often go along with the pain in close companionship, and that the pain as often begins and ends with strict periodicity at a given time, and is followed by an obscure hot fit. It

would seem as if the neuralgia and the shivering had both of them to do with the state of the circulation, to which, in ague, the name of cold stage is given—a state, that is, not of over-activity, but of under-activity. And what holds good in this particular case is also true in all cases of neuralgia, for in all these cases there is, together with a state of the circulation, which, to say the least, is not unlike that of the cold stage of ague, the same disposition to periodicity, and the same accompaniments of shivering and shuddering.

In rheumatic fever the rule, I believe, will be found to be this—that the pains, which had been torturing the patient for weeks or months previously, come to an end when the feverish reaction and local inflammation of the fully formed disorder make their appearance. After this, the joints are *tender* enough, but if the patient keep still, as he is very likely to do under the circumstances, he is comparatively at peace so far as the pain is concerned. Or, if it be otherwise, the pain will generally be found to be in a part in which the signs of rheumatic inflammation are imperfectly established or absent, or else at a time in which there is a decided remission in the inflammatory symptoms—an event which happens more frequently in this disorder than is commonly supposed.

It is also difficult to look upon the local inflammation of gout as essential to the existence of the racking pain of this disorder. “About two o’clock in the morning,” says Sydenham, who from personal experience knew full well what he ought to say, “the patient is awakened by a severe pain in the great toe, or, more rarely, in the heel,

ankle, or instep. This pain is like that of a dislocation, and yet the parts feel as if cold water were being poured over them. Then follow chills and shiverings, and a little fever. The pain, which was at first moderate, becomes more intense; and with its intensity the chills and shivers increase." After tossing about in agony for four or five hours, often till near daybreak, the patient suddenly finds relief, and falls asleep. Before falling asleep, the only visible change in the tortured joint is some fulness in the veins: on waking in the morning, this part has become swollen, shining, red, tender in the extreme, and more or less painful, but this painfulness is as nothing in comparison with the torture of the night past. It seems, indeed, as if the pain now existing must be referred to the mere tension and stretching of the inflamed ligaments, for it may be relieved, or even removed, by judiciously applying support to the toe and to the sole of the foot. On the night following, and not unfrequently for the next three or four nights, the sharp pain in all probability returns, reappearing and disappearing suddenly, or almost suddenly, and resulting in the discovery of additional inflammatory swelling upon awaking in the morning. The pain in these relapses, like the primary pain, is accompanied by chills and shiverings, and by the most distressing irritability and excitability, but until unequivocal signs of inflammation are developed in it the painful part is not tender in the true sense of the word. The inflammation is attended by no fever, or by very little; or, if it be otherwise, as happens occasionally, and the inflammation runs higher than usual, *the characteristic pain is less*

urgent than usual. Dr. Garrod points out this latter fact in his excellent work on gout,* and says that he has seen several illustrations of it. From its history, then, it would seem as if the inflammation of gout were not essential to the pain of gout. It would seem as if the pain went hand in hand with the rigors which are preliminary to the development of the inflammation. It would seem as if the inflammation had little to do with the pain, for if it were otherwise, it is scarcely to be supposed that the pain should be least urgent in the cases of gout in which the inflammation is most marked, and that the unequivocal signs of inflammation should make their appearance during sleep without waking the patient. Nay, it would even seem as if the pain were put an end to by the establishment of the inflammation—as if, in fact, the pain were antagonized rather than favoured by the inflammatory condition. Moreover, the suddenness with which it begins and ends in the majority of cases must be looked upon as a reason for referring the pain to the category of neuralgia—a category in which, to say the least, it is not a little difficult to find any place for inflammation.

There is also reason to believe that pain holds the same relation to fever and inflammation in other kinds of fever besides the rheumatic, and in other kinds of inflammation besides the gouty.

A few years ago, I had a patient in the Westminster Hospital, who, when I saw him first, complained of violent pains all over the body, in the back and loins

* "Gout and Rheumatic Gout." Post 8vo. London: Walton and Maberley, 1859, p. 39.

especially, and also of chills and shiverings. Shortly afterwards he was in a state of high fever, and the pains and chills and shiverings had all taken their departure. The case was one of small-pox ; and the impression left upon my mind was that the pains and rigors were symptoms which ought to be classed together, and regarded as belonging to the cold and not to the hot stage of the fever. And this case would seem to be a fair illustration of what happens in other fevers ; for it seems to be the rule rather than the exception for the pains which attend upon the onset of these disorders to pass away, or to become greatly mitigated, when the cold stage gives place to the hot stage. Nay, it would even seem as if pain gave place for the time to what may be called artificial feverishness. At any rate, I have more than once felt *tic-douloureux* pass away as soon as I could set my blood fairly in motion by violent bodily exercise ; and on two occasions I have derived a similar benefit from a practice which is not unfrequently adopted in the hunting field, and put an end summarily to a sudden attack of lumbago by leaning forwards in the saddle and beating the loins with the hands until the whole body was aglow and the perspiration dripped from the forehead.

Nor is it different with inflammation. In the case of a dislocation or sprain, for example, the acute pain of the accident—the pain to which Sydenham likens that of gout—does not, as a rule, remain after the parts have begun to be hot and swollen and tender ; and this case is certainly no exception in the history of inflammation. It would seem, in fact, as if the proper place for the

pain was among the phenomena of the preliminary cold stage—the stage of “shock,” and not among the phenomena of actual inflammation. And it is not impossible that the efficacy of blisters in the relief of many kinds of pain may furnish another passage in the same story ; for it is a fact, which is as well established as any fact in therapeutics, that blisters are most effectual means of relieving pain, and that this relief is usually coincident with the blistering—that is, with the inflammation set up by these agents. Nor is a contrary conclusion to be drawn from the history of certain cases in which pain continues as a permanent symptom after the full establishment of inflammation, as, for example, in deep-seated inflammation of the mamma ; for in these cases it is a fact that this persistent pain is immediately relieved or removed by those operative measures which diminish the tension of tender parts arising directly or indirectly from the inflammation. It is a fact, that is to say, that the persistent pain in these cases is an accidental and not an essential accompaniment of the inflammation—a consequence, as I have just said, of the inflamed and tender tissues being kept on the stretch, and not a necessary part of the inflammation itself.

How far these inferences will be confirmed or set aside by the histories of those forms of pain in which the nervous system is more especially implicated remains to be seen ; but, so far, there seems to be good reason for believing that pain of a neuralgic character is connected with a depressed state of the circulation rather than with anything like true fever.

2. Pain is no very conspicuous symptom in the com-

mon form of cerebral meningitis—that is, in the tubercular form; and in simple meningitis there is reason to believe that any severe pain in the head is precursory to, rather than attendant upon, the actual inflammation. A few years ago, for example, I had in the Westminster Hospital a well-marked case of acute simple cerebral meningitis in a boy aged 15. On my first visit, the face was pale and perspiring, the ears and head felt cold to the touch, the pupils were dilated, the pulse was contracted and feeble, and what was complained of chiefly was agonising pain in the head, with frequent chills and shiverings. On my second visit, eight hours afterwards, the face was flushed, the head burning hot, the pupils contracted, the eyes ferrety, the skin hot and dry, the pulse quick and hard, and fierce delirium had taken the place of the pain. And this, so far as my experience goes, is the regular history of the pain in this disorder. It is pain ceasing, not pain beginning, as the signs of active determination of blood to the head make their appearance. It is pain in association, as it would seem, with an ænemic rather than with a hyperæmic condition of the membranes of the brain.

In the same hospital, about the same time, there was also under my care another case, in which, after death, were found unmistakable signs of recent spinal meningitis of an acute character, the patient being a young man aged 23. The illness began three days before admission with sharp pain in the back and legs, with shivering and retention of urine, the patient beginning to suffer in this way shortly after sleeping for some time flat on his back upon the grass. The back was

stiff, and the head somewhat drawn back, and on any attempt at movement, and now and then without such attempt, severe pain was experienced along the whole course of the spine, in the legs, in the lower part of the abdomen, and, to a lesser extent, in the head also, this pain being always accompanied by increase of stiffness. Death happened at the end of a week. During the last three days of life the bouts of pain and contraction were very occasional and of very short duration ; and in many instances even these, there is reason to believe, might have been avoided if the patient could have been kept perfectly still.

And certainly this is the conclusion which must be drawn from the history of those painful disorders which come under the head of spinal irritation, and which are so often met with in hysterical patients, for here severe pain of a neuralgic character is a prominent symptom, and yet the collateral symptoms, and the issue of the disorder in nineteen cases out of twenty, make it impossible to ascribe the pain to inflammation of the substance or membranes of the cord.

With respect to neuralgia in all its manifold forms one thing is certain, and this is, that neuritis is not necessary to its production.

In the cases where extreme local tenderness with some degree of swelling along the track of the sciatic nerve would seem to show that sciatica has become complicated with neuritis, the neuralgic pains are not aggravated. On the contrary, the plain fact would seem to be rather this—that these pains, which had been such prominent symptoms previously, come to an end

when the local tenderness and swelling give evidence of the establishment of inflammation in the course of the sciatic nerve, if only the affected limb be kept still and all pressure upon the tender parts be avoided.

It is also the rule, rather than the exception, for toothache to come to an end when the face becomes swollen and inflamed, and for the stabbing pains which so generally precede the inflammatory eruption of herpes to give place to tenderness when the eruption is fully formed.

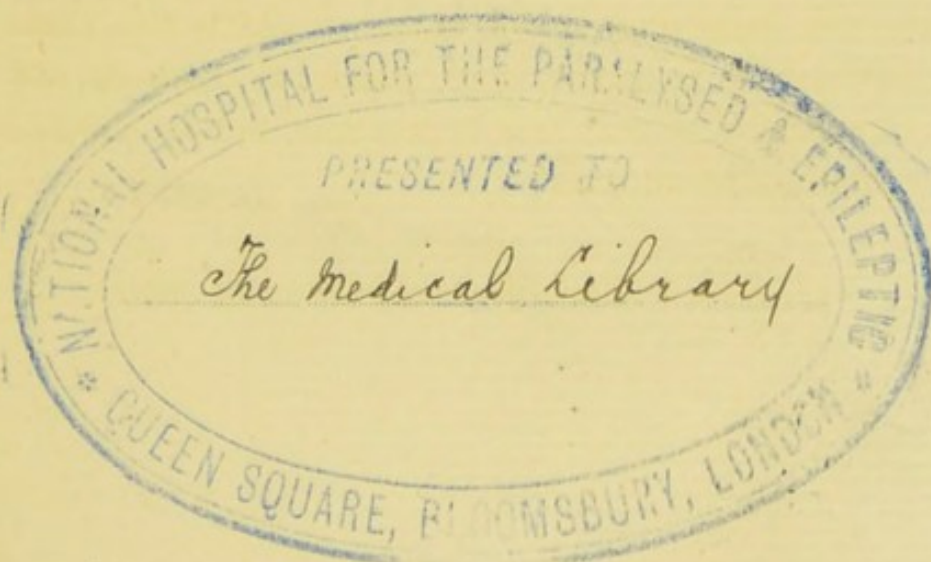
Again, I can testify to this being the true history of facial neuralgia, or *tic-douloureux*, in many cases : first, neuralgia without local tenderness and swelling and redness, and with frequent chills and shiverings, and a decidedly depressed condition of the circulation ; then, after an interval more or less prolonged, local tenderness, redness, and swelling, with some slight feverish reaction, without chills and shiverings, and without neuralgia, the true neuralgia for the most part coming to an end coincidently with the establishment of the local inflammation.

In short, neuralgia and pain of a neuralgic character would seem to be connected, not with a state of inflammation in any part of the nervous system, but with a state the reverse of this, which may or may not issue in such inflammation—a state to which the name of “irritation” is given, and which is marked, not by relaxation of vessels and hyperæmia, but by contraction of vessels and anæmia.

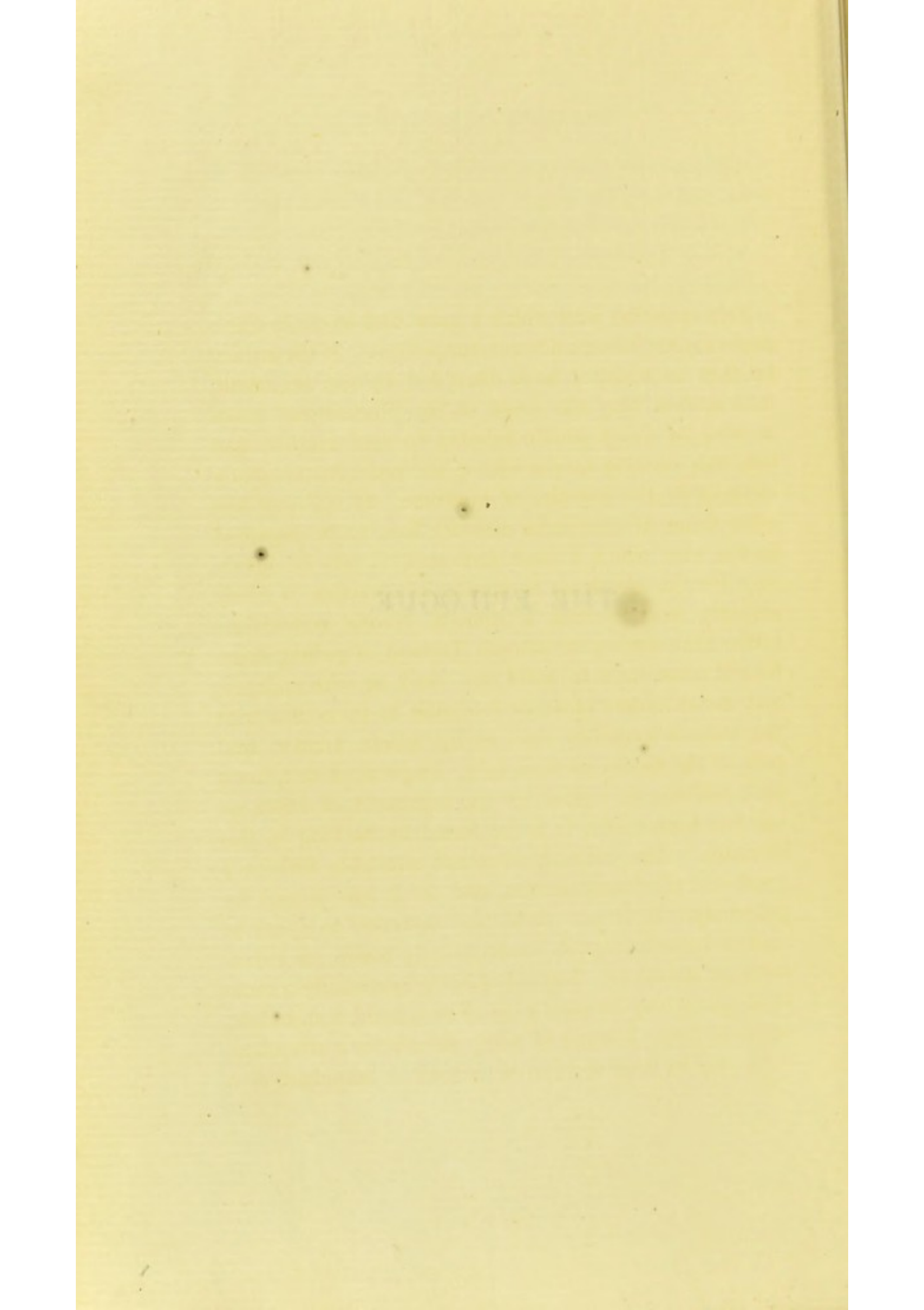
And so it is that the real history of neuralgic dis-

order is none other than that of convulsion and spasm and tremor. There is always a state of vascular inactivity which is altogether inconsistent with the current notion that this form of pain is connected with a state of exalted vitality in the parts of the nervous system which have to do with sensation. The case is the very opposite of what it is commonly supposed to be. The case, indeed, is one for which no other key would seem to be needed than that of which use has been made all along.

All has not been said which might have been said, but enough has been said, I trust, to make it not improbable that vital motion must be regarded in a very different light to that in which it has been regarded for so long a time—that a complete revolution is necessary in the doctrine of vital motion—that vital motion is, in fact, merely a mode of physical motion. I have not, so far as I know, indulged in anything like special-pleading, and, in fact, I could not have spoken differently if I had been sworn on all that I hold most sacred to speak the truth, the whole truth, and nothing but the truth.



THE EPILOGUE.



THE question with which I have had to do in these pages cannot be set aside as unimportant. If the answer be that to which I have been led by the argument, it is evident that the creed of the physiologist needs revising in every article relating to vital motion, and that this revision carries with it the necessity for many changes in the practice of medicine. If epilepsy and other forms of convulsive disorder have to be regarded in the way which I have indicated, it will be necessary for the physician to modify his practice in many respects, and to seek a remedy among restoratives rather than among sedatives. Instead of pulling down he will often have to build up. And, as with epilepsy, and the disorders more or less akin to it, so also with the various maladies marked by spasm, tremor, and pain of the nature of neuralgia. In point of fact, not a little evidence in support of the argument, of which no use has been made, is to be found by seeking in this direction. The evidence was not available without a good deal of circumlocution, and so it was passed by purposely. It is not right, for instance, to speak of various remedial agents as necessarily tonics, or stimulants, or sedatives. Instead of being necessarily a sedative, opium may be used so as to be a tonic, a stimulant, or a sedative. Instead of being necessarily a stimulant, wine may be used so as to be a tonic, a stimulant, or a

sedative. Instead of being necessarily a tonic, quinine may be used so as to be a tonic, a stimulant, or a sedative. And until clearer views are arrived at in these matters it is difficult to find in the results of treatment anything which is really calculated to confirm or contradict the conclusion arrived at in the argument. Indeed, all that I can now do is to assert—and this I do without any hesitation—that the result of carrying out in practice the view in question has been to convince me that I have right on my side, and to make me hope that in setting forth this view I may have done something which will at one and the same time increase the power of the physician and quicken the insight of the physiologist.

FINIS.

