

Epileptic and other convulsive affections of the nervous system, their pathology and treatment.

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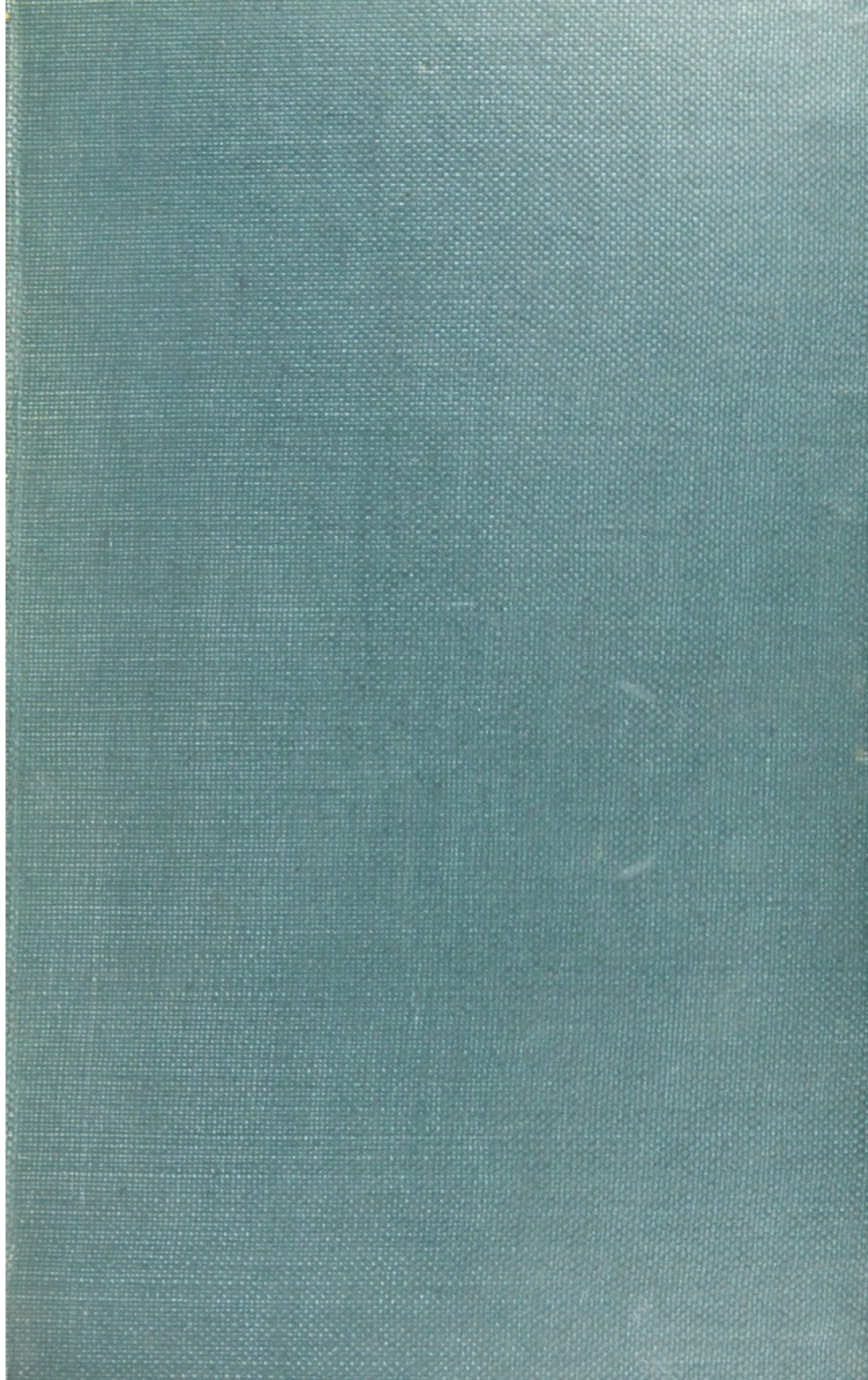
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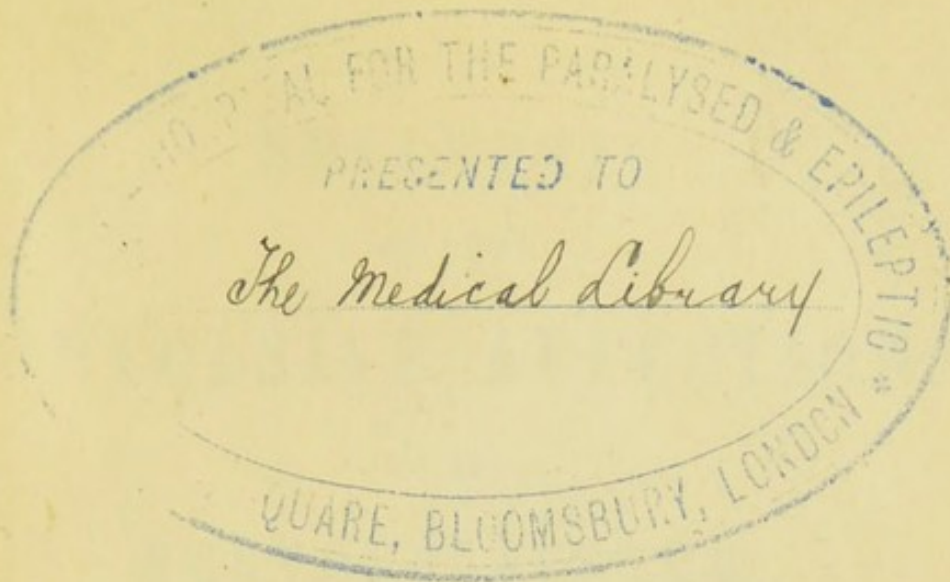
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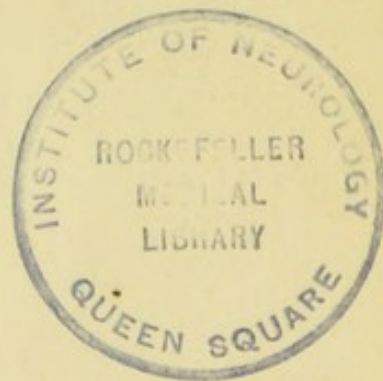
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EPILEPTIC
AND OTHER
CONVULSIVE AFFECTIONS
OF THE
NERVOUS SYSTEM.



PHILIPIC

AND

CONSTITUTIVE APPLICATIONS

OF THE

NEW YORK SYSTEM

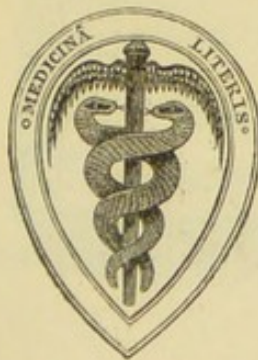
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EPILEPTIC
AND OTHER
CONVULSIVE AFFECTIONS
OF THE
NERVOUS SYSTEM,
THEIR
PATHOLOGY AND TREATMENT.

BY
CHARLES BLAND RADCLIFFE, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; PHYSICIAN TO, AND LECTURER ON MATERIA
MEDICA AND THERAPEUTICS AT, THE WESTMINSTER HOSPITAL, ETC.

THIRD EDITION,
(INCORPORATING THE GULSTONIAN LECTURES FOR 1860.)



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P R E F A C E.

IN the present edition of this work I have incorporated the Gulstonian Lectures which I had the honour of delivering at the Royal College of Physicians in the spring of 1860. I have also so re-written and re-cast the whole as to make a new book rather than a new edition. How far I have succeeded in establishing the principle I have at heart, I must leave my readers to determine; but this much I may say—that the additional thought and experience of the last three years have only served to strengthen my own convictions in the truth of this principle, and to show that my argument, as set forth previously, was a very imperfect statement of the grounds of these convictions.

C. B. R.

4, HENRIETTA STREET,
CAVENDISH SQUARE, W.

LETTER

In the general opinion of this world I have never
perceived the Christian's virtues which I had the
pleasure to observe in the Holy Kings of Castile
in the year of 1500. I have also observed many
of the same kind in the Kings of France and
Spain. This is the reason that I have not
written the history of France. I have only
written the history of Castile and of the
Kings of France and Spain. I have not
written the history of the other Kingdoms of
France and Spain. I have only written the
history of the Kings of France and Spain.
I have not written the history of the other
Kingdoms of France and Spain. I have only
written the history of the Kings of France
and Spain.

O. A. R.

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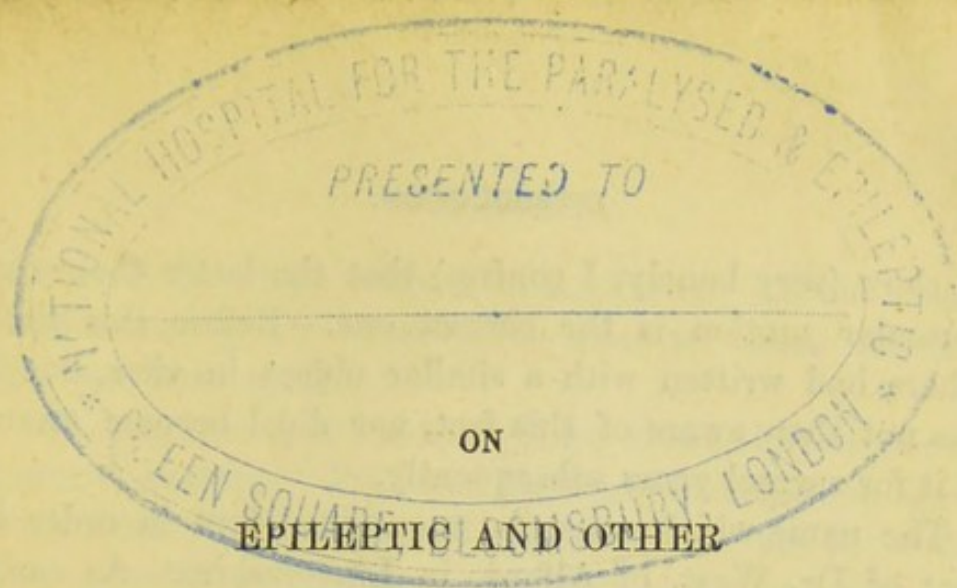
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ON
EPILEPTIC AND OTHER
CONVULSIVE AFFECTIONS
OF THE
NERVOUS SYSTEM.

INTRODUCTION.

IN opposition to the theory which supposes that muscle is endowed with a vital property of contractility, and that contraction is brought about when this property is roused or excited or *stimulated* into action, it may be held that there is a state of polarity in living muscle during relaxation which produces relaxation, and that contraction is nothing more than the necessary result of the muscle being liberated from this state and left to the operation of the attractive force which is inherent in the physical constitution of the muscular molecules. It may be held, indeed, that a change is necessary which shakes to its very centre the pathology of all convulsive affections of the nervous system, for it is evident that the physiology of muscular motion must be the corner stone upon which the pathology of convulsion is built up.

Ten years have now elapsed* since I first endeavoured

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

to show (very lamely, I confess) that the latter theory of muscular motion is the correct one. Before this time others had written with a similar object in view, but I was not then aware of this fact, nor did I become aware of it for several years subsequently.

The name which must be mentioned first in order is that of 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 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 awhile this influence, so as to allow this peculiar property of muscular fibre to show itself." The coexistence 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 adduced in support of the probability of this theory.

Very shortly after the publication of these remarks, a similar idea appears to have been hinted at by Sir Charles Bell in a lecture at the Royal College of Surgeons in London, 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

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

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 contraction, which is in no sense peculiar to muscle, is nothing more than the cessation of expansion—"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 piece of caoutchouc might contract when set free from a previous state of extension; and an analogy is hinted at 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. 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 mus-

* 'Traité de Physiologie comparée de l'Homme et des Animaux,' 8vo, Montpellier and Paris, 1838.

† 'Comptes Rendus,' March 17th, 1847.

cular action, in which he writes:—"Ce fluide développé principalement dans les muscles, s'y répand, et, doué d'une force répulsive entre ses parties, comme le fluide électrique, il tient les éléments de la fibre musculaire dans un état de répulsion analogue à celui présenté par les corps 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 dans la roideur cadavérique. . . . Suivant la quantité de ce fluide qui cesse d'être libre dans la 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 peu 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,

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

sondern den Zusammenziehungen bis auf einen geringen Grad entgegenzuwirken. Im lebenden Organismus, in welchem Ruhe etwas Unmögliches ist, ist auch ein ruhender Muskel eben so wohl wie ein ruhender Nerv undenkbar, der Muskel in seinem beständigen Streben, sich zusammenzuziehen, wird vom Nerven daran verhindert, im Nerven macht sich das fortwährende Streben kund, die Zusammenziehung des Muskels auf ein gerechtes Mass zurückzuführen; das Ergebniss dieser zwei einander entgegengesetzten Eigenschaften der 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 contract 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 the 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 irritability, 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 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 theory of muscular motion—a change amounting to no less than a complete revolution;

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

and I am glad that it is so, for thus supported, I am the more bold to challenge attention to the facts and arguments which will be advanced in the following pages.

Under these circumstances, therefore, it is necessary to aim at acquiring clear views respecting the physiology of muscular motion before entering upon the consideration of the many and difficult questions relating to the pathology of convulsive affections, and thus the primary division of the present inquiry will be into two parts, one physiological, the other pathological—

PART I.—PHYSIOLOGICAL PRELIMINARIES RESPECTING MUSCULAR MOTION.

PART II.—PATHOLOGICAL DEDUCTIONS RESPECTING EPILEPTIC AND OTHER CONVULSIVE AFFECTIONS OF THE NERVOUS SYSTEM.

NOTE.

In the following pages I find it expedient, in many places, to avoid the use of the term IRRITABILITY and its cognates. I do this because these terms are part and parcel of that particular theory of muscular motion from which I am constrained to dissent, and because I cannot use them in a different sense without giving rise to frequent confusion of thought. What I propose to do is to use the words INCITE, INCITATION, INCITABLE, INCITABILITY, in place of irritate, irritation, irritable, irritability. In their derivation the words INCITE and EXCITE are closely akin, but the former word differs from the latter in not having been assigned by

common usage to the current theory of muscular motion, and in not conveying in so full a degree the idea of acting by rousing, or animating, or stimulating. In its primary sense, indeed, the meaning of the verb to incite is rather to stir up simply, and other meanings are secondary to this. At any rate there is no good reason why I should not use the word, as I propose to use it, in this restricted sense. In speaking of an incitable muscle, then, all that I shall mean is a muscle which is capable of transitory contraction in the sense in which an irritable muscle is capable of transitory contraction. In speaking of an incitable nerve, all that I shall mean is a nerve which has that capability of producing contraction or sensation which belongs to an irritable nerve. In speaking of muscular incitability, all that I shall mean is that capability for contraction which is implied in the idea of muscular irritability as contradistinguished from the idea of muscular tonicity. In speaking of nervous incitability, all that I shall mean is that capability for producing contraction or sensation which is implied in the idea of nervous irritability.

PART I.

PHYSIOLOGICAL PRELIMINARIES RESPECTING
MUSCULAR MOTION.



PHYSIOLOGICAL PRELIMINARIES.

IN endeavouring to arrive at a satisfactory theory of muscular motion, the course which seems to be least round-about is that of which the principal stages are marked out in the five following chapters, and this, therefore, is the course which I propose to pursue :—

CHAPTER I.—*On muscular motion in relation to the action of the blood.*

CHAPTER II.—*On muscular motion in relation to the action of nervous influence.*

CHAPTER III.—*On muscular motion in relation to the action of electricity.*

CHAPTER IV.—*On muscular motion in certain other relations, and on the theory which seems to apply to simple muscular motion.*

CHAPTER V.—*On the applicability of this theory to rhythmical as well as to simple muscular motion.*

CHAPTER I.

ON MUSCULAR MOTION IN RELATION TO THE ACTION OF
THE BLOOD.

THE fact, which has not yet lost its novelty, that after the full establishment of rigor mortis the relaxed and incitable condition of the muscle may be restored by the injection of blood into the muscular vessels—a fact which has been abundantly demonstrated by Dr. Brown-Séquard* and Professor Stannius†—may well serve as a text for introducing what has to be said in the present chapter.

One of Dr. Brown-Séquard's experiments by which this fact is established was upon the arm of a criminal who had been guillotined at 8 a.m. on the 12th of July, 1851. The experiment, which consisted in injecting and re-injecting a pound of defibrinated dog's blood into the brachial artery, was commenced at 11 p.m.—fourteen hours after decapitation. At this time the limb upon which the injection was practised was in a perfect state of rigor mortis. As the blood began to penetrate into the vessels, some reddish spots appeared in different parts of the skin of the forearm, of the arm, and, more particularly, of the wrist. Very quickly these spots became larger, and the skin acquired the appearance it has in rubeola. Soon afterwards the whole surface had a reddish-violet hue. A little later, and the skin had acquired its natural living colour, elasticity, and softness, and the veins stood out distinct and full as during life. Then, the muscles relaxed, first in the fingers, lastly in the shoulder, and on examination they

* 'Comptes Rendus,' June 9th and 25th, 1851.

† Op. cit.

were found to have recovered their lost incitability. At 11.45 p.m. the muscular incitability was more decided than it was at 5 p.m., at which time the corpse was first examined; and from 11.45 p.m. until 4 a.m., when fatigue compelled the experimenter to abandon his labours, there was no alteration in this respect. When the experiment was commenced the temperature of the blood was 75° Fahr., and that of the room 66° Fahr.

The subject of another experiment was a full-grown rabbit, which had been killed by hæmorrhage. Dr. Brown-Séquard waited until rigor mortis had fully set in, and then injected the defibrinated blood of the same animal into the principal vessel of one of the hind limbs, which limb had been previously removed from the body. Fifteen minutes after the commencement of the injection the muscles had lost their stiffness, and responded readily to mechanical and galvanic incitation. From this time, throughout the night, until 3 p.m. on the day following, the blood was injected at intervals of from twenty to thirty minutes, and during the whole of this time the muscles remained perfectly soft and incitable. All this while, also, the muscles of the companion hind limb and of the rest of the body were in a perfect state of rigor mortis. Between 3 p.m. and 4.50 p.m. the injections were discontinued, and, in this interval of an hour and a half, the limb (with the exception of a few bundles of fibres here and there) had time to return to the former state of rigidity. Under the second injections the muscles once more and speedily became soft and incitable, and in this state they continued until, from the lateness of the hour, the experiment was again abandoned. On the morning following, the parts of the body which had been left to themselves were beginning to pass out of the state of cadaveric rigidity, but the limb upon which the injections had been practised was perfectly stiff. On the third morning rigor mortis was still undiminished in this

latter limb, and, in marked contrast to this state, the muscles of the rest of the animal were soft and in an advanced stage of putrefaction.

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

On the 21st of July, 1851, for example, at 7·30 a.m., Professor Stannius put a ligature around the abdominal aorta and crural arteries of a puppy. A few minutes after 10 a.m. the muscles had begun to stiffen in the parts from which the blood was excluded. At 10·45 a.m. both hinder limbs were stretched out, and perfectly stiff and cool. At 11·40 a.m. the ligatures were loosened, and the blood was seen and felt to penetrate into the empty vessels. At 11·45 a.m. the natural warmth had returned in some degree, and the right hinder limb was a little more flexible than the left. At noon both these limbs had recovered their flexibility, and it appeared once as if the left had moved spontaneously, but no sign of pain was caused by pinching the toes. At 12·30 p.m. the muscles of the paralysed limbs contracted everywhere upon the application of the galvanic poles; and at one time the galvanism seemed to cause pain, for the animal, which was before quiet, gave a sudden plunge forwards when touched by the poles. Death happened unexpectedly at 12·45 p.m.

Early upon the morning of the day following, a similar experiment was performed upon another puppy. At noon, in this case, there was no evidence of stiffness in either of the hind limbs, but the muscles below the knees had ceased to respond to the touch of the electrodes. At 2·15 p.m. both these limbs were stretched out and rigid, and all evidence of incitability was at an end. At 2·35 p.m. the ligatures were untied. At 3·35 p.m. the application of the electrodes caused strong contraction in the muscles

of both thighs, and weaker contractions in the muscles of the left leg below the knee, and there were but few traces of rigidity anywhere. At 5.35 p.m. the muscles, now perfectly soft and pliable, responded readily to the prick of a knife as well as to the touch of the electrodes. On the following morning the animal was found dead.

In another experiment, in which the abdominal aorta and the crural arteries of a fine puppy were tied and left tied to the end, Professor Stannius shows very clearly that the rigidity of which mention is here made is perfectly identical with rigor mortis. In this case, four hours after the operation, the muscles *below* the ligatures were perfectly rigid and devoid of incitability. In the evening of the day following there was no alteration in this state. 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 had been rigid before death, flaccid, moist, and partially putrescent. In other words, the parts *below* the ligature 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.

Here, then, are certain experiments which would seem to show that the influence of the blood, be this what it may, is exercised in counteracting rigor mortis. And this inference, which I had drawn from the experiments of Dr. Brown-Séguard before my attention was directed to those of Professor Stannius, is the same as that which the last-named physiologist has drawn from his own experiments.

There are, however, certain facts* which seem to show that muscle is affected differently by arterial and by venous blood, and these facts have led Dr. Brown-Séguard to think

* 'Comptes Rendus,' No. 16, 1857.

that the office of arterial blood is to minister to the nutrition of muscle and other tissues, and to the storing up of contractile and other forms of power, and that the office of the venous blood is to supply a stimulus by which the power derived from the red blood is called into action—a view according to which the function of the black blood is no less important to the interests of the economy than that of the red blood.

The chief argument in favour of the idea that venous blood is endowed with these stimulating properties is based upon the well-known fact that the muscles contract violently when the whole mass of blood becomes venous, as in asphyxia. Another argument, also found among the phenomena of asphyxia, is derived from the fact that the pulse is fuller and stronger, and that the mercury rises to a higher point in the hæmadynamometer, when black blood is being pumped into the arterial system. Other arguments are found in some original experiments in which Dr. Brown-Séguard finds that certain involuntary muscles may be thrown into or out of a state of contraction by injecting alternately black and red blood into the vessels. In one of these experiments, for example, the uterus of a pregnant rabbit was separated from its connexions with the cerebro-spinal centres and blood injected into the aorta. On injecting black blood the uterus contracted, and two or three fœtuses were expelled; on injecting red blood these contractions immediately passed off.

But, it may be asked, is it not possible that these contractions may be due to the want of arterial blood rather than to the presence of venous blood? And is not such an explanation the less difficult of the two?

It is certainly true that an animal is as violently convulsed when it is bled to death as when it dies by strangling. In other words, it is certainly true, that the convulsion which depends upon the vessels of an animal being suddenly

emptied of blood is every whit as violent as the convulsion which is brought about by causing the vessels to become suddenly filled with black blood. And thus, on taking the two cases together, the convulsions would seem to be related to the absence of arterial rather than to the presence of venous blood, for absence of arterial blood is a cause which must operate, not in one case only, but in both. At any rate, with this fact of convulsion from hæmorrhage to account for, it is evident that venous blood cannot be regarded as an essential element in the process of convulsion.

Nor is the fuller and stronger pulse of the first moments of suffocation a certain sign that the left ventricle is then being stimulated to more vigorous contraction by the black blood. On the contrary, this phenomenon may be little more than the necessary consequence of the difficulty which the black blood experiences in getting out of the arteries. That this difficulty is a reality was well shown years ago by the late Professor John Reid.* If, for example, the carotid and jugular of a rabbit be exposed, and a ligature placed around the windpipe, the blood in the artery is seen to become rapidly darker and darker, until it is not to be distinguished from the blood in the neighbouring jugular. From two minutes to two minutes and a half are occupied in this transformation of scarlet into black blood. It is found, also, that the arteries are more distended, and that the blood will escape with greater force and in fuller stream when the process of suffocation is at its height—phenomena which are readily explained, as Dr. Reid explained them, by supposing that black blood moves through the systemic capillaries with far greater difficulty than red blood. Recently, moreover, some new facts have been elicited by Professor Draper† (the younger), of New York,

* 'Phys., Anat., and Pathol. Researches,' 8vo, Edinb., 1848.

† "Lectures on the Physiology of the Circulation," 'American Medical Monthly,' April, 1860.

which are calculated to shed not a little light upon the question under consideration. After the stoppage of the respiration, as Professor Draper shows, the left ventricle and auricle, as well as the right ventricle and auricle, become enlarged from the accumulation of blood within them. At first, the cavities on both sides of the heart are gorged to an equal degree, or thereabouts; a little later, on comparing the two sides, the left is even more distended than the right. It is evident, also, that the distension of the left side of the heart is preceded by an unmistakable engorgement of the aorta and arterial system generally, and that the great veins in the neighbourhood of the heart do not participate in the engorgement of the arterial system and of the cavities of the heart. At the time, indeed, when the arteries are most distended, the venæ cavæ, both superior and inferior, would seem to contain less than their usual amount of blood. And, further, it is found that this is the state of things until the elastic resiliency of the coats of the arteries has an opportunity to come into play and empty these vessels—an opportunity which happens when the pumpings of the heart come to an end. In a word, the facts are in direct opposition to the commonly received idea that the veins are gorged with blood and the arteries comparatively empty in the earlier moments of asphyxia; and being what they are, they afford a ready means of explaining why, in these earlier moments, the arterial pulse may be fuller and stronger, without supposing that the left ventricle is then stimulated to more vigorous contraction by the stronger stimulus of venous blood. What is the complete explanation of the fuller and stronger pulse at this time is another matter. It is certain, however, that the ordinary contraction of the ventricle under these circumstances will, in some measure, explain the phenomenon, for the force which ought to be expended in propelling the blood through the capillaries must, just

in proportion as it fails to do this, be expended in dilating the coats of the intermediate arteries. At the same time, it is possible that the ventricle may at this time contract with unusual force, for it is not unlikely that this muscle may participate in the violent contractions which are then convulsing the entire muscular system.

It is also possible, without assuming the existence of any hyper-stimulating properties in the venous blood, to explain the contraction of the uterus which is brought about by injecting black blood into the aorta of a pregnant rabbit; for is it not a well-known fact that the uterus has often contracted and expelled its contents when pregnant animals have been bleeding to death in the service of science? There are, moreover, certain experiments by Dr. O. Spiegelberg,* of Gottingen, which afford special grounds for supposing that the contraction which happens in a case like the one just mentioned is due to the want of arterial rather than to the presence of venous blood. These experiments, which were often repeated upon rabbits, cats, and guinea-pigs, exhibit the uterus as remaining at rest so long as the circulation is not interfered with, as contracting when the flow of blood along the descending aorta is interrupted by pressure, and as relaxing as soon as the pressure is removed. They exhibit the uterus and intestines as contracting with especial vigour in animals which had been bled to death; and, in one or two instances, they show in addition a cornu uteri which had been separated from its vascular connexions as contracting more decidedly than the fellow cornu which had not been so separated. They exhibit, also—and this is the fact which is of special interest in the present inquiry—the uterus and intestines as contracting in a much more marked manner after the tying of the abdominal aorta than after the tying of the vena cava or vena porta—as contracting in a much more marked manner, that is to say,

* Henle and Pfeuffer's 'Zeitschr.,' 3 Reihe, ii, 1857.

when the vessels are left without blood than when the vessels are gorged with black blood. From these experiments Dr. Spiegelberg concludes that this form of muscular contraction is caused by want of blood in the part, and from the last experiment in particular he argues, as I had already done from the previous considerations, that it is the absence of arterial, and not the presence of venous blood, which favours contraction in these and analogous cases.

It would seem, also, that a strong argument in favour of the idea that the contraction in these several cases is really due to the absence of arterial and not to the presence of venous blood, is supplied by Professor Harley,* in his admirable investigations upon the mode in which the blood is affected by agents which, above all others, have the power of provoking contraction, namely, strychnia and brucia. In these investigations, two graduated tubes of the same size are filled half full of calves' blood, and a minute quantity of strychnia is added to the contents in one of them. Then, after frequently shaking the blood in each tube with fresh quantities of air, both tubes are well corked and set aside, half full of blood, half full of air. During the next twenty-four hours the

	Composition of Common Air.	Composition of Air after having been in contact with <i>Simple Blood</i> for 24 hours.	Composition of Air after having been in contact with <i>Blood containing Strychnine</i> for 24 hours.
Oxygen	20·96	11·33	17·82
Carbonic acid...	·002	5·96	2·73
Nitrogen	79·038	82·71	79·45
	100·000	100·00	100·00

blood and air thus corked up are occasionally well shaken together; at the end of this time the corks are removed,

* 'Lancet.' June 7th and 14th, and July 12th, 1856.

and the air, which has been all this while in contact with the blood, is examined by Bunsen's method. The result, as is shown in the accompanying table, is that the air which has been in contact with the poisoned blood contains more oxygen and less carbonic acid than the air which has been in contact with the simple blood. In other words, the strychnia has prevented the blood from absorbing oxygen—prevented the blood from becoming thoroughly arterial; and thus the action of the poison upon the circulation may be said to be equivalent to a copious loss of blood, for in the experiment of which the results are given above the addition of a very minute quantity of the poison reduced by full two thirds the power which this blood has of becoming arterial by absorbing oxygen. When brucia is used the only difference is one of degree, the power of preventing arterialization in this case being somewhat less energetic.

Nor is a different inference to be drawn from the state of the blood in rigor mortis. In this case, the first feeling may perhaps be one which prompts us to assign this contraction to the stimulation of venous blood, for at this time every drop of blood may be supposed to be permanently venous; but, on second thoughts, it is not less evident that this blood is not in the position in which it ought to be in order to allow us to entertain this view. For what is the case at this time? The case is simply this—that the blood is found, not in the fine capillary vessels in which it could alone react effectively upon the muscular fibre, but in the venous reservoirs into which it is passed on after it has done its work in these vessels. The case, in fact, is one which would seem to connect the contraction of rigor mortis with a state of emptiness of the fine muscular vessels rather than with the presence of venous blood in these vessels.

It may be mentioned, also, as in some degree bearing upon the questions which have been under consideration,

that the muscles which exhibit the highest degree of incitability are precisely those which are least liberally supplied with blood. Thus, the voluntary muscles of fishes and reptiles receive very little blood when compared with the voluntary muscles of birds and mammals, and yet these muscles are far more incitable in the former classes than in the latter; thus, the involuntary muscles in any animal receive very little blood when compared with the voluntary muscles, and yet these former muscles are more incitable than the latter; and thus, again, the muscles of any hibernating animal, voluntary and involuntary, are far more incitable during the syncope of winter life than they are during the fever of summer life. It would seem, in fact, as if the degree as well as the duration of the transitory form of contraction were inversely related to the supply of blood in the muscle.

There is also an interesting experiment by Professor Claude Bernard* which may be cited in this place in confirmation of the same conclusion—an experiment which shows that the incitability of the muscles of a warm-blooded animal is exalted to a reptilian standard when the circulation and temperature are depressed to a reptilian standard. In this case, the spinal cord of a rabbit was divided between the fifth and sixth cervical vertebræ—at a level, that is to say, which corresponds to the upper limits of the brachial plexus. With the exception of some faint relics of the power of moving the fore paws, the trunk and limbs were completely paralysed by the operation, and the animal was left lying upon its side without power to stir, breathing by its diaphragm alone, or by the diaphragm with such small aid as might be derived from very partial respiratory movements in the upper part of the chest, panting, passing fæces continually, but soon

* 'Leçons sur la Physiologie et la Pathologie du système nerveux,' 8vo, Paris, 1858, vol. ii, p. 12.

recovering so far as to eat with avidity a carrot which was offered to it. Seven hours after the operation, the breathings had fallen considerably below the natural rate of frequency, and at the same time the paralysed trunk and limbs had become cold and comparatively bloodless—a state contrasting in a very marked manner with the state of the ears, for, owing to the section of the cord having trenched upon the cilio-spinal region, these organs were hot and red. The cold and paralysed parts had also undergone a change by which the incitability of their muscles had acquired the characters naturally belonging to the muscles of a cold-blooded animal—a change which was evident at the time by the readiness with which strong reflex movements could be provoked, and which was confirmed afterwards by the tardy appearance and the tardy departure of rigor mortis. Thirty minutes after death, when the muscles of the non-paralysed head and neck had ceased to furnish any signs of incitability and were beginning to pass into the state of rigor mortis, the muscles of the paralysed parts were still prompt to enter into strong contraction under the slight touch of a bistoury. Nay, so much was the incitability of the parts increased, that on separating one of the hind limbs, and preparing it in the same way as that in which the rheoscopic limb of a frog is prepared—taking, that is to say, the leg without the thigh, but leaving a long portion of the sciatic nerve in attachment—it was found to be possible to repeat all the experiments which are ordinarily performed with the rheoscopic limb.

— Looking back, then, at the evidence which has been advanced in the present chapter, there would seem to be little reason for supposing that blood is in any sense a stimulus to either form of muscular contraction. There would seem to be little reason for supposing that blood is

concerned as a stimulus in producing that prolonged contraction of rigor mortis which is referred to the form of contractility which is called tonicity, for this prolonged contraction has been seen to pass off when blood is injected into the vessels of the rigid muscles. Nor would there seem to be any very good reason for supposing that blood is concerned as a stimulus in producing the transitory contraction, which is assigned to that form of contractility which is specially designated irritability, for it has been seen from more than one point of view, that the proneness of the muscle to enter into, and to remain in, this state of transitory contraction is inversely related to the supply of arterial blood to the muscle. So far from being a stimulus to contraction, it would seem, indeed, as if the influence of the blood is exercised in producing the opposite state of relaxation.

CHAPTER II.

ON MUSCULAR MOTION IN RELATION TO THE ACTION OF
NERVOUS INFLUENCE.

THE agency of nervous influence in muscular motion is a problem of which a satisfactory solution can scarcely be hoped for in the present state of physiological science. It is a problem, also, of which the consideration of one part, and this not the least important, namely that which concerns the "nerve-current," had best be transferred to the chapter which treats of the agency of electricity in muscular motion. At the same time it is not difficult to see, even now, that the facts tally very ill with the idea that muscular contraction is produced by any stimulation on the part of the nervous system.

As justifying this remark, it may be mentioned, first of all, that muscle contracts more forcibly and more readily after it is cut off from the influence of the nervous centres. This is no new fact, but it is one of which the clearest demonstration is to be found in two comparatively new experiments by Dr. Brown-Séquard.*

In one of these experiments a frog is suspended by its fore feet, and the muscles of one of its hind limbs are put upon the stretch by hanging a weight upon a hook which has been previously attached a little above the ankle—a weight which is just a little heavier than that which can be raised by these muscles when they are thrown into contraction by pinching the toes. After this, the spinal cord is divided immediately behind the second pair of

* 'Comptes Rendus,' May 10th, 1847.

nerves, and the power of the contraction which can be provoked in the now paralysed limb by pinching the toes is measured at different times after the division by taking from or adding to the weight upon the hook. What follows is very strange. Immediately after the division of the cord, the muscular power put forth by the weighted leg is sometimes *nil*, but generally it is about a third or fourth of what it was before the operation. A few minutes later, this power is evidently rallying. In twenty or twenty-five minutes all that had been lost is recovered. At the end of an hour the muscular power is greater than it was before the operation—perhaps doubled. An hour or two later, it is certainly doubled, possibly trebled; and from this time up to the twenty-fourth hour, when the augmentation has generally reached its maximum, it goes on steadily increasing. In the case of two very fine frogs, A and B, the actual results were as follows, the weights raised being expressed in grammes :

Frogs experimented upon	A.	B.
Weight raised <i>before</i> the division of the spinal cord				60...	60
Weight raised <i>after</i> the division of the spinal cord	}	Immediately afterwards		20...	10
		In five minutes	...	45...	30
		In fifteen minutes	...	60...	40
		In twenty-five minutes	...	80...	60
		In one hour	...	130...	100
		In two hours	...	140...	120
		In four hours	...	140...	130
		In twenty-four hours	...	150...	140
		In forty-eight hours	...	150...	140

At the last point the muscular power may remain stationary for six, ten, fifteen or twenty days. In a month, if the animal lives, the power in question will, in all probability, have fallen to its original value before the operation. In six, seven, or eight months, it may not be more than a third or fourth of this value; but it is not improbable that some part of this failure might have been prevented if care had been taken to exercise the paralysed limb by galvanism.

In the second experiment, in addition to dividing the

spinal cord behind the roots of the brachial plexus, as in the first experiment, the principal nerve of *one* of the hind limbs is divided high up near the spine. Two hours afterwards, both hind limbs are separated from the body, and their incitability is compared by pinching or galvanizing the nerves. This is the experiment: the result is—that the “irritability is augmented” in both limbs, but mostly in that which had been previously cut off from the influence of the spinal cord by the division of its nerves.

Here, then, are two experiments, interesting individually, most interesting in connexion. In the first, the muscles are seen to contract with greater power when they are cut off from the influence of the brain and medulla oblongata. In the second, the muscles are seen to be more apt to enter into a state of contraction after they are cut off from the influence of the spinal cord than they were before they were cut off from this influence—a fact which of itself may do more than suggest a doubt as to the correctness of Marshall Hall’s notion that the increase of muscular power after the division of the spinal cord is due to increased stimulation on the part of that portion of the cord which is still in connexion with the muscles, which increased stimulation is brought about by the removal of the spinal cord from the controlling influence of the brain.

There are, moreover, sundry difficulties of a very grave character which must be surmounted before it can be allowed to be even probable that the increased aptitude to contraction which exists in these and similar cases is owing to “augmented irritability” in nerve or muscle.

If, for example, the two hind limbs of a frog, prepared in the usual way, are placed as they are placed in one of Dr. Harley’s experiments, the one in simple distilled water and the other in a weak solution of strychnia or brucia, it is found that the former limb may be made to

contract strongly for some time after the other limb has lost its incitability and passed into a state of rigor mortis. In this experiment the nervous incitability of the two limbs is tested by means of induced currents applied to the nerves, and care is taken not to act upon the nerve of one limb more than upon its fellow; and thus the more speedy loss of incitability in the nerve and muscles of the limb immersed in the poisoned solution is not to be accounted for by supposing that a vital property of irritability has been more rapidly expended in contraction.

The result is also the same when, as in another of Dr. Harley's experiments, the hearts of two frogs are similarly immersed, the one in simple water and the other in a weak solution of strychnia or brucia, for in this case it is found that the former heart will go on pulsating for some time, and be capable of being provoked into renewed pulsation for hours, after the other heart has passed into a state of rigor mortis. It is found, indeed, that the heart immersed in the poisoned solution ceases to beat almost immediately, and that it passes into a state of rigor mortis before the heart immersed in simple water has lost any perceptible amount of its power of contracting rhythmically.

It would seem, indeed, as if these poisons acted upon the muscular tissue, not by augmenting the irritability, but in the same way as that in which they have been seen to act upon the blood; for, as Dr. Harley remarks,* "the destruction of the irritability of the muscle may be supposed to imply the suspension of the process of absorbing oxygen, and giving off carbonic acid—the so-called respiration of the muscle." But be the explanation what it may, the fact remains that the incitability of both nerve and muscle dies out most rapidly in the case where the irritability is supposed to be most augmented (for in no case is

* *Op. cit.*

the irritability supposed to be more augmented than in cases of poisoning by strychnia or brucia), and that this more rapid dying out of incitability is not owing to a contractile power having been more rapidly expended in contraction.

Nor is there any lack of evidence to show that muscular contraction may occur in its most exaggerated form under circumstances in which a large amount of nervous influence would seem to be withheld from the muscles.

The convulsion from hæmorrhage is a case in point—a case, too, of no doubtful significance. For if it be true, as it undoubtedly is, that the functional activity of an organ is directly proportionate to the activity of the circulation in that organ, it follows that the supply of nervous influence from the nervous centres to the muscles must be at zero when, as is the case in convulsion from hæmorrhage, the vessels are almost emptied of blood, and the beatings of the heart well-nigh still.

And, certainly, the inference which may be drawn from the occurrence of convulsion during hæmorrhage is not contradicted by the experiments of Sir Astley Cooper and Drs. Kussmaul and Tenner, to which reference has now to be made.

“I tied,”* says Sir Astley, “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 effectually excluded. Respiration stopped almost directly, convulsive struggles succeeded, the animal lost its 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

* ‘Guy’s Hospital Reports,’ No. iii, 1836.

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 a third time, and with the same effect. In seven hours it was cleaning its face with its paw. In nine hours the vertebral arteries were compressed for the fifth time, and the result was the same; namely, suspended respiration, convulsions, and loss of motion and consciousness. On removal of the pressure, violent and laborious respiration ensued, and afterwards the breathing became very quick. After forty-eight hours, for the sixth time, the compression was applied with the same effect."

The tale which is told by this well-known experiment appears to be, that convulsion may co-exist with a state of things which implies inaction of the great cranio-cervical nervous centres,—for such a state of inaction must necessarily be brought about by arresting the flow of blood through the cervical arteries. And this tale is also that which is told in still plainer terms, and at greater length, in the following experiments by Drs. Kussmaul and Tenner.*

In one of these experiments, the first thing is to place ligatures around the common innominate and left subclavian arteries of a rabbit,† and to tie them in a manner which will allow them to be untied at a moment's notice. Upon tying the ligatures, the animal is instantly thrown into a state of convulsion. A few moments afterwards,

* 'Untersuchungen z. Naturlehre der Menschen u. d. Thiere,' von J. Moleschott, vol. ii, Frankfort, 1859.

† In a rabbit, the right subclavian and both carotids usually commence in a common innominate artery, while the left subclavian springs independently from the arch of the aorta.

while the convulsion is still raging at its height, the ligatures are untied, and the convulsed muscles relax suddenly—so suddenly that the animal seems to have been struck with a stroke of paralysis. This is the experiment; these are the results. Convulsion is brought on, that is to say, by preventing the flow of blood to the brain, the medulla oblongata, the upper part of the spinal cord, and the cervical ganglia of the sympathetic system; and convulsion is as instantly suspended by allowing the blood to return to these organs. It seems, indeed, as if this convulsion is connected with a state of complete inaction of one or more of the nervous centres named, for what action can there be when blood is entirely withheld from the part?

In a second experiment, the subclavian arteries of a rabbit are tied at their origin, and another ligature is placed around the arch of the aorta, a little beyond the opening of the left subclavian artery. The ligatures are placed, that is to say, so as to bring about a result which is the very opposite of that which was secured in the last experiment. In this case the blood is cut off from the trunk and limbs, and the circulation confined to the head and neck; in that case the blood was cut off from the head and neck, and the circulation confined to the trunk and limbs. In this case, the vessels of the brain, the medulla oblongata, the upper part of the spinal cord, and the cervical ganglia of the sympathetic nerve, receive more than their proper share of blood, for all the blood of the body is diverted in this direction, while the rest of the spinal cord and the thoracic and abdominal ganglia of the sympathetic system receive no blood at all. Upon tying these vessels, the parts of the body below the ligatures are paralysed, and the paralysis is not preceded by any convulsive movements: upon compressing the carotids, so as to close the only channels through which

the circulation is carried on. This state of paralysis is immediately changed for one of convulsion. This is the experiment; these the results. It is found, also, that the paralysis and convulsion may be made to alternate for an indefinite number of times by removing and renewing the pressure upon the carotids.

Here, then, are two experiments from which, as it appears, more than one important deduction may be made with respect to the operation of the great nervous centres in muscular motion.

One deduction, arising equally from both experiments, is—that a state of general muscular contraction or convulsion is caused by cutting off the supplies of blood to the great nervous centres of the head and neck. Thus, convulsion is instantly brought about—in the one experiment by tying the great arteries of the head and neck, in the other by employing pressure so as to close these vessels; and convulsion is as instantly brought to an end—in the one experiment by untying the ligatures which had closed the cranio-cervical vessels, in the other by removing the pressure which had answered the same purpose as the ligatures. There is, as it would seem, a double argument in favour of the connexion of convulsion with a state of inaction in one or more of the cranio-cervical nervous centres, for, to repeat the question already asked, what action can there be in any of these centres when the circulation in the part is completely suspended?

Another deduction, which may be said to confirm the first from an opposite point of view, arises from the second experiment. It is that convulsion is not caused by “rush of blood to the head.” In the second experiment the ligatures close the great channels which convey the blood to the trunk and limbs, and all the blood of the body, or at any rate a great portion of the whole, is, as a matter of course, diverted to the head. If convulsion were dependent

upon determination of blood to the head, the circumstances in this case are eminently favorable to the production of convulsion, and yet the result is simply paralysis of the parts below the ligatures, not convulsion. In this case, indeed, everything would seem to show that convulsion is not connected with increased determination of blood to the head, for, on proceeding with the experiment, the animal is instantly thrown into a state of violent convulsion by pressing upon the vessels so as to arrest altogether the flow of blood to the head. In a word, this second experiment appears to show, in a very conclusive manner, that overaction of any one of the cranio-cervical nervous centres is no cause of general convulsion, for it may fairly be assumed that the increased flow of blood to these centres, which takes place when the great arteries of the head and neck are not pressed upon, and which does not result in convulsion, is attended by a corresponding increase in the functional activity of the nervous centres receiving the blood.

And as a collateral argument in favour of the same deduction—that there is wanting blood, and therefore wanting action in one or more of the cranio-cervical nervous centres during convulsion—it may be mentioned that drowsiness and not convulsion is found to happen in certain other cases in which there is excess of blood in these centres—as in the capillary injection arising from the division of the sympathetic nerves in the neck, or in the venous engorgement brought on by tying the jugular veins, or in the arterio-venous congestion which happens when the jugulars are tied and the cervical sympathetics divided in the same animal.

It is evident, also, from Drs. Kussmaul and Tenner's second experiment, that the spinal cord has not to play the same part in the production of convulsion as that which is played by the cranio-cervical nervous centres, for, with the spinal cord paralysed as it must be paralysed by cutting off

the supply of blood from the trunk and limbs, it is still possible to bring about violent convulsion by cutting off the supply of blood to the head and neck. No doubt, the spinal cord is necessary to the production of general convulsion; it is necessary as a conductor by which the changes in the cranio-cervical nervous centres which bring about convulsion may tell upon the trunk and limbs; but that it is not necessary in any higher sense appears sufficiently evident in the experiment under consideration, for in this case convulsion is seen to happen when, from want of blood, any special action in the spinal cord, as an independent centre, is not to be thought of. Looking at the experiment under consideration, indeed, it seems to be difficult to suppose that any nervous centre *below* the medulla oblongata can have to do with the production of general convulsion, and this being the case it may be supposed that cessation of the action of the medulla oblongata, and the consequent suspension of the supply of nervous influence which naturally emanates from this centre to the muscles, is the chief cause of the convulsion which is brought about by arresting the flow of blood to the head, for it is a well-established fact that convulsion will continue with little or no abatement after all the nervous centres *above* the medulla oblongata have been removed in succession.

And, certainly, there is little reason for supposing that the prolonged contraction which comes on sooner or later in paralysed muscles, and which is characteristic of rigor mortis, is dependent upon the stimulation of nervous influence. With respect to rigor mortis there can be but one opinion, for the simple fact that the nerves have lost all their incitability before its occurrence, is a sufficient proof that this form of prolonged muscular contraction is not dependent upon the stimulation of nervous influence. And with respect to the prolonged contraction which comes on eventually in muscles which have been paralysed by

dividing the nerves which connect them with the nervous centres, it would seem to be equally impossible to refer the contraction to the stimulation of nervous influence. If the muscle had been paralysed by some injury to the nervous centres, it might perhaps have been different, but where, as in the case under consideration, the paralysis is due to a cause which effectually cuts off the supply of nervous influence to the muscles, this idea of stimulation on the part of the nervous influence, as the cause of the contraction, is surely inadmissible. It is also to be borne in mind that the prolonged contraction which comes on eventually in paralysed muscles, is transformed at death into rigor mortis without any preliminary process of relaxation, and that the kinship to rigor mortis, which is thus brought to light, is itself an argument of some value in proof of the position that the prolonged contraction of paralysed muscle is really independent of any stimulation on the part of the nervous system. Nor is it an objection to this view that the muscles do not become permanently contracted at the moment of paralysis, for as will be seen presently, there is for some time in muscle itself a polar condition which, so long as it lasts, must effectually antagonise the state of permanent contraction.

— As with the blood, then, so with “nervous influence,” the whole tenor of the evidence would seem to contradict the theory which seeks to explain muscular contraction by supposing that a property of contractility has been stimulated into action. It is perfectly plain that such stimulation can have little to do in the production of rigor mortis, for this form of prolonged muscular contraction does not make its appearance until every trace of nervous influence is annihilated. It is probable, to say the least, that any stimulation on the part of nervous influence can have little to do with that form of prolonged muscular con-

traction which comes on eventually in paralysed muscles. And with respect to the transitory contractions which belong to the incitable condition of muscle, a similar conclusion would seem to be necessary. For has it not been seen that the hind limbs of a frog are more prone to contract in this manner after they are deprived of cerebral nervous influence by dividing the spinal cord? Nay, more, has it not been seen that this disposition to contract is still more marked when these limbs are cut off from spinal as well as from cerebral nervous influence, by dividing the sciatic nerves? And has it not been seen that general convulsion is the consequence of suddenly arresting the development of nervous influence in one or more of the great cerebral nervous centres, by suddenly interrupting the supply of arterial blood to the head? And has it not been seen that strychnia, which is supposed to have the effect of augmenting the irritability of nerve and muscle in a very remarkable manner, has in reality an opposite effect, namely, that of destroying the incitability of nerve and muscle, and of hastening in an unmistakable manner the occurrence of rigor mortis? Has it not been seen, in fact, that the proneness of the muscle to enter into and to remain in the transitory form of muscular contraction is inversely related to the supply of nervous influence to the muscle? In what has been said, however, no mention has been made of an important element in nervous influence, of which a good deal will have to be said in the next chapter, and until something is known of the "nerve-current" and its workings, it is impossible to have any clear idea respecting the action of "nervous influence" in muscular motion.

CHAPTER III.

ON THE OPERATION OF ELECTRICITY IN MUSCULAR MOTION.

THE operation of electricity in muscular motion is, without doubt, a problem of no ordinary difficulty. Often it has engaged the thoughts of physiologists, often it has foiled them. Each year, however, facts are brought to light which justify a better hope of arriving at a satisfactory solution. Thanks to MM. Matteucci, Du Bois-Reymond, Eckhard, and others, little is now wanting to complete our knowledge of what is necessary to be known respecting animal electricity and its workings in muscular motion. Thanks to M. Chauveau, of Lyons, our knowledge of the mode in which continuous and instantaneous currents bring about muscular motion has been amazingly simplified within the last few months. At present, indeed, everything augurs favorably for the possibility of arriving at a satisfactory solution of the problem under consideration.

In entering upon this inquiry, there appear to be sundry advantages in discussing the operation of continuous and instantaneous electrical currents before speaking of the character and action of the electricity which is inherent in living muscle and nerve; and for this reason I shall adopt this course, and speak first of all—

I. Of muscular motion in relation to the action of instantaneous and continuous electrical currents.

A year ago I should have begun to treat this part of my subject in a very different manner. Now I begin by confessing that a new and clearer light has been thrown

upon the path by M. Chauveau,* and that the safest and wisest course is to walk for the present in this light, beginning at the point where it falls upon the action of *induced currents* in muscular motion.

1. Upon taking hold of the poles of an ordinary induction coil, the well-known shock is experienced. When the current is powerful, this shock passes through the body; when less powerful, the hands only are affected; when sufficiently feeble, the effect is limited to the hand out of which the current passes—the hand which grasps the negative pole, or zincode. And so also for every person when several persons are included in the circuit, the current, according to its strength, making itself felt through the whole body of each person, or in the two hands of each person, or simply in the one hand by which it passes out of each person.

Nor is the case different when the action of an induced current is directed specially upon muscle or musculo-motor nerve.

Applying a sufficiently feeble current directly to a muscle, the contraction which follows is limited to the fibres in the neighbourhood of the negative pole; increasing somewhat the strength of the current, the contraction shows itself also in the neighbourhood of the other pole; using a still stronger current, the whole muscle is thrown into a state of contraction. And when several muscles are included in the same circuit it is still the same, each muscle being convulsed throughout its whole substance, or in the neighbourhood of the two poles, or only at the negative pole, according as the strength of the current is properly regulated.

Applying the poles of the coil to the facial nerves of a horse (the facial nerves, from their superficial position,

* Brown-Séquard's 'Journal de la Physiologie,' July and Oct., 1859, and Jan., April, and July, 1860.

are especially suited for the experiment), the negative pole being upon one nerve and the positive pole upon the other nerve, the facial muscles are convulsed on one side only if the current be sufficiently weak. If, indeed, the current be sufficiently weak, the convulsion is confined to the side of the face from the nerve of which the current makes its exit—the nerve which lies under the negative pole, or zincode. When, moreover, the facial nerves of several horses are included in the same circuit, it is still the same, the contraction being on both sides of the face of each horse if the current be of ordinary strength, or only on the side of the face of each horse from the nerve of which the electricity makes its exit, if the current be sufficiently feeble.

In the experiment with a *feeble* current upon the facial nerve of a horse it is found also that the positive pole may be moved to the same side as the negative pole, and placed so as to cause the current to pass up or down or across the nerve, without producing any change in the result so far as contraction is concerned. What is necessary to contraction in this case is for the negative pole to be upon the nerve, and if this pole be in this position it matters not where the positive pole is placed.

Comparing the induced current which attends upon the beginning of the inducing current, with that which attends upon the ending of the inducing current, the latter, as might be expected from its greater strength, produces a more marked action upon the muscle, but with this exception there is no difference whatever between the two induced currents.

2. The physiological effects produced by *discharges of statical electricity* are in all particulars analogous to those which are produced by the action of induced currents. If the charge be sufficient and the discharge be made by the two hands the shock extends across the body; if the

charge be smaller, the effects of the discharge may be limited to the two hands; if the charge be sufficiently reduced in strength, the only sign of the discharge is felt in the hand out of which the electricity issues—the hand in relation to the negative pole. And so, also, when the discharge is made to tell upon a single muscle or nerve, or upon several persons or muscles or nerves included within the same circuit. The muscular phenomena attending upon discharges of statical electricity are indeed strictly analogous to those which attend upon the passage of induced currents, and—what is particularly to be noticed—this analogy does not fail in the matter of the contraction being irrespective of the course of the discharge along the nerve.

3. The physiological effects of a *galvanic current* are of a more complex character, but with a little attention they are reducible, not only to rule, but to the same rule.

If, for example, the poles of a Daniell's cell be applied to the cheek of a horse, so that one of them is upon the trunk of the facial nerve and the other somewhere else, the facial muscles contract when the negative pole is upon the nerve, and not when the positive pole is in this position, *if the current passing be sufficiently weak*. It is of no moment where the positive pole is placed. It may be placed so as to cause the current to the negative pole to pass up the nerve or down the nerve or across the nerve, any way or every way, and all that appears to be essential to the production of contraction in this case is to have the negative pole upon the nerve.

Nor are we at liberty to suppose that the direction of the current is of greater moment in that common experiment upon the nerve of a detached frog's leg in which it is held to be all important. In this case, at first, the muscles contract at the beginning as well as at the ending of a current of ordinary strength; a little later, and contrac-

tion is only at the beginning or only at the ending of the current—at the beginning if the direction of the current along the nerve be centrifugal or *direct*, at the ending if the course of the current along the nerve be centripetal or *inverse*. At this period, indeed, it seems as if the direction of the current was all important; but this notion is contradicted by several of the new facts which are furnished by M. Chauveau.

One of these new facts is this. The hinder half of a frog is prepared as Galvani was wont to prepare it—the thighs, that is to say, are completely separated by snipping through the pelvic structures at the median line, and the lumbar structures are removed so as to make the lumbar nerves the only bond of connexion between the thighs and the remaining fragment of the spine. The two limbs, thus prepared, are then hung astride upon the edge of a plate of glass, and a *feeble* galvanic current is passed through both sets of lumbar nerves, by applying one pole to one set, and the other pole to the other set. An arrangement is made, that is to say, by which the current passes up the nerves on one side, through the connecting piece of spine resting upon the edge of the insulating plate of glass, and down the nerves on the other side,—by which the current is *inverse* in one set of nerves and *direct* in the other set. The result of this, the first stage of the experiment, is—contraction at the beginning of the current in the limb along the nerves of which the current is *direct*, contraction at the ending of the current in the limb along the nerves of which the current is *inverse*. It seems as if the direction of the current is essential to the result, but such a notion is inconsistent with what remains to be told. In the first stage of the experiment the two poles were separated, one being upon one set of lumbar nerves, the other upon the other set; in the second stage of the experiment, which has now to be explained, both poles are

placed upon the same set. They are placed sometimes in one position, sometimes in another, so that the current along the intervening portion of nerve is sometimes direct, sometimes inverse, and now the result is—contraction at the beginning of both currents, and not contraction at the beginning of the direct and at the end of the inverse current, as it was in the first stage of the experiment.

The same frog which has served for the demonstration of the fact which has just been mentioned will serve also for the demonstration of that which has now to be noticed. In this case the sciatic nerve of one of the limbs is exposed in its inferior third, and the corresponding portion of the thigh is cut away so as to leave the leg hanging to the thigh by the nerve simply. This being done, and a *feeble* direct current being passed along the nerve by placing the negative pole upon the sciatic and the positive pole upon the lumbar nerves, the contraction which results in the muscles of the leg is at the commencement of the current, and at this time only. In the next place, a *feeble* inverse current is passed along the nerve by placing the positive pole on the sciatic and the negative pole on the lumbar nerves, and the contraction which results is still at the commencement of the current and at this time only, but it is now exhibited in the muscles of the thigh, as well as in the muscles of the leg. As in the last experiment, contraction happens on the closure of the circuit irrespectively of the direction of the current, and the presence of contraction in the leg simply, or in the thigh as well as in the leg, would seem to be determined by the position of the negative pole upon the nerve.

In demonstrating the third and last fact which has to be noticed here, it is necessary to expose the sciatic nerve of a frog, to raise it in a loop over one of the poles of a Daniell's cell, and then to place the other pole within the same loop so as to make it rest upon the muscles of the thigh in a line

directly under the first pole. If the nerve be looped over the positive pole, a *direct* current (the current employed is still a very *feeble* one) passes in the portion of nerve intervening between the pole and the muscles of the leg, and, according to theory, it is to be expected that the muscles of the leg will contract when the current begins to pass. In fact, however, these muscles are alike quiescent at the beginning and at the ending of the current. If, on the other hand, the nerve be looped over the negative pole, the current along the portion of nerve intervening between the pole and the muscles of the leg is *inverse*, and hence it is to be expected that these muscles will contract when the current ceases to pass. This is what ought to be the case according to theory; what actually happens is that these muscles contract when the current begins to pass, and then only. It is still the same—contraction when the negative pole is on the nerve, and not when the positive pole is upon the nerve.

Subjecting a musculo-motor nerve to the action of a somewhat stronger galvanic current than that which was employed in these experiments, the muscles are seen to contract at the beginning and at the ending of the current. Proceeding with the experiment, and still using the same current, a change is presently noticed in which the contraction is found to forsake the ending or the beginning of the current, and to occur at one of these times only. Now how is this? Why should there be this change? Why should contraction be present only at the moments when the current begins and ends, and absent in the interval during which the current is passing quietly and continuously? M. Chauveau has an answer to these questions—an answer which refers partly to the constitution of the current acting, and partly to the changing condition of the nerve acted upon.

That there is something peculiar in the galvanic current

at the moments when the current is closed and opened is more than probable. There is, indeed, reason to believe that current electricity begins and ends in rushes of high tension which are of the same character as induced currents or discharges of statical electricity. Thus: a stretched wire conductor is thrown into sonorous vibrations at the moments when the galvanic circuit is opened or closed just as it would be by induced currents or by discharges of statical electricity. Thus again: an ordinary wire experiences sudden changes in length and breadth at the same moments, just as it would do under the action of these currents or discharges. The spark at making and breaking the circuit would also seem to tell the same tale, and so may the induced currents which attend upon the beginning or ending of the galvanic current, for it is difficult to understand how these high-tension induced currents could originate except as outflowings of currents of the same character as to tension. There are reasons, indeed, and good reasons too, for supposing that the galvanic current begins and ends in instantaneous rushes of electricity of high tension—rushes to which Professor Faraday has given the name of *extra-currents*.

These extra-currents are generally spoken of as agreeing with induced currents in their course and relative strength, and those who (myself among the number) have sought to explain the contractions attending upon the closing and opening of the galvanic circuit by connecting them with instantaneous currents of high tension, prevailing at these moments, have spoken of these currents as being induced currents. As M. Chauveau shows, however, there is good reason for supposing that extra-currents differ from induced currents both in their course and relative strength, and that the recognition of this difference is of fundamental importance in explaining many of the points which have to be explained in due time.

In determining the course and relative strength of the extra-currents, M. Chauveau appeals to the muscular movements which attend upon the beginning and ending of a galvanic current when a musculo-motor nerve is included in the circuit. Placing one pole upon the right facial nerve of a horse, and the other pole upon the left facial nerve, and using a *weak* current, he finds that the contraction attending upon the closure of the circuit is on the side from which the galvanic current is making its exit—the side of which the nerve is under the negative pole; and hence he argues that the *initial* extra-current (*initial*, because attending upon the beginning of the galvanic current) must make its exit from the nerve into the negative pole, and that, so doing, it must pass in the same direction as the galvanic current. He finds, also, that the contraction which attends upon the opening of the circuit in the same experiment is on the opposite side of the face—the side of which the nerve lies under the positive pole; and for the same reason he argues that the *terminal* extra-current (*terminal*, because attending upon the ending of the galvanic current) must make its exit from the nerve into the positive pole, and that, in order to this, it must have passed in a contrary direction to that of the galvanic current. And this must be allowed to be a fair inference from what has gone before, for as with *weak* induced currents and *weak* discharges of statical electricity, so with *weak* extra-currents, it is to be expected that only that nerve will be acted upon from which the electricity makes its exit. With the same experiment M. Chauveau also finds that the contraction attending upon the closure of the circuit is more marked and much slower to die out than the contraction attending upon the opening of the circuit, and hence he argues that the initial extra-current is more powerful than the terminal.

Thus exhibited, then, the extra-currents differ altogether, both in course and relative strength, from induced currents. The initial extra-current is in the same direction as the galvanic current, the initial induced current is in the opposite direction. The terminal extra-current is in the opposite direction to that of the galvanic current, the terminal induced current is in the same direction as the galvanic current. The initial is the stronger of the two extra-currents; the terminal is the stronger of the two induced currents.

Assuming, then, the existence of extra-currents such as these at the beginning and ending of the galvanic current, and knowing the marked influence of induced currents and discharges of statical electricity in producing contraction, M. Chauveau concludes that high tension is a necessary quality in the electricity which produces contraction, and that, for this reason, contraction is present at the beginning and ending of the galvanic current, because it is only at these moments that the electricity of the current has the requisite degree of tension, and absent in the interval during which the galvanic current is passing quietly and continuously, because the electricity then passing is deficient in tension. And certainly it is difficult to connect the power of producing contraction with the polar, or thermal, or chemical workings of electricity. For is it not true that these workings are at a minimum where (as in induced currents and discharges of statical electricity) the power of producing contraction is at a maximum? And is it not equally true that these workings are at a maximum where (as in the ordinary galvanic current between the moments of beginning and ending) the power of producing contraction is altogether absent? Moreover, M. Chauveau believes that contraction is produced by the passage of high tension electricity in the same way as that in which it is produced by the pinch of a forceps or

the prick of a knife, that is, by the mechanical commotion which is set up in the nerve or muscle, one or both; and this idea has much to recommend it, for a current or discharge which, when strong, will pierce cardboard and window-glass, or bend, break, shiver, and pulverise a conductor of insufficient dimensions, must give rise, even when very weak, to no small amount of mechanical commotion in a living nerve and muscle.

The change which comes over the order of contraction when a musculo-motor nerve is acted upon for some time by an ordinary galvanic current varies in different cases, but the simplest and most typical case is that in which the nerve of a rheoscopic limb* is subjected to this treatment. In this case, what happens may be conveniently divided into four periods, as in the following table:

	Direct Current.		Inverse Current.	
	Beginning.	Ending.	Beginning.	Ending.
First period . .	Contraction	Contraction	Contraction	Contraction
Second period . .	Contraction	—	—	Contraction
Third period . .	Contraction	—	—	—
Fourth period . .	—	—	—	—

In the first period, contraction occurs at the beginning and ending of the current, and it is of no moment, so far

* The *rheoscopic limb* (ῥεῖν, to flow, and σκοπεῖν, to examine) is the preparation which was first introduced into notice by M. Matteucci under the name of "grenouille galvanoscopique," and which is called "stromprüfenden Froschschenkel" by M. du Bois-Reymond. It consists of the foot and leg of the hind limb of a frog, removed from the thigh at the knee, and having the whole length of the sciatic nerve remaining in attachment. It furnishes the most delicate test of the presence of electricity; for if the veriest trace of a current is made to act upon the nerve, the muscles are at once thrown into a state of contraction.

as this result is concerned, whether the course of the current along the nerve be inverse or direct. In the second period, contraction occurs only at the beginning of the direct and at the ending of the inverse current. In the third period, contraction is absent except at the beginning of the direct current. In the fourth period, contraction has ceased altogether to attend upon either current. It seems as if the direction of the current along the nerve was an essential element in the problem; but in reality this is of as little moment here as heretofore. The problem is somewhat complicated, but, as M. Chauveau shows, it is one which may be solved by remembering what has just been said about extra-currents, and by realising the fact that, during these four periods, the incitability of the nerve included between the galvanic poles is progressively dying out from the divided end towards the muscles. What is to be supposed with respect to the incitability of the nerve is this—that, in the first period, this condition is unimpaired everywhere; that, in the second period, it has disappeared to a point which is further down the nerve than the place to which the *outer* pole (as the pole which is furthest from the muscles may be called) is applied; that, in the third period, it has partially disappeared also as far down the nerve as the place to which the *inner* pole (as the pole which is nearest to the muscles may be called) is applied; and that, in the fourth period, it has altogether disappeared from all parts included within the galvanic circuit.

In the first period, then, the incitability of the nerve is unimpaired everywhere, and there is contraction at the beginning and ending of both currents, for the simple reason that the extra-currents cannot make their exit at either pole without acting upon the nervous incitability.

In the second period the departing incitability of the nerve has departed to a point which is further down the nerve than that to which the *outer* pole is applied, and

hence the nerve no longer responds to the extra-current which makes its exit from the nerve into this pole. *In the case of the direct current*, as the table shows, there is contraction when the circuit is closed, and none when the circuit is opened. There is contraction, that is to say, under the action of the initial extra-current, and not under the action of the terminal extra-current. And so it ought to be, according to the premises. Passing in the same direction as the galvanic current, *i. e.* centrifugally, the initial extra-current makes its exit from the nerve into the *inner* pole, and contraction results—because the nerve at this point has not lost the power of responding to the action of electricity. Passing in the opposite direction to that of the galvanic current, *i. e.* centripetally, the terminal extra-current makes its exit from the nerve into the *outer* pole, and hence there is no contraction—because the nerve at this point has lost the power of responding to the action of the electricity. *In the case of the inverse current*, on the other hand, as the table shows, contraction is at the beginning, and not at the ending, of the galvanic current; and why it should be so is not difficult to understand. In this case the initial extra-current will cause no contraction, because its direction is inverse, like that of the galvanic current, and because it must, for this reason, make its exit from the nerve where the nerve has already lost its incitability, that is, under the *outer* pole. In this case, the terminal extra-current will cause contraction; for being in the opposite direction to that of the galvanic current, it must make its exit from the nerve where the nerve still retains its incitability, that is, under the *inner* pole.

In the third period the change which has taken place is the disappearance of contraction upon the ending of the inverse current. In the last period the departing incitability of the nerve had departed under the *outer* pole; in this period this condition is upon the point of departing

also under the *inner* pole. It must be understood, however, that the incitability of the nerve under the *inner* pole is departing only, not altogether gone, and that for this reason the nerve at this point may be able to respond to the action of the *stronger* initial extra-current, and yet be unable to respond to the action of the *weaker* terminal extra-current. Now, as was shown when speaking of the second period, the initial extra-current of the direct galvanic current and the terminal extra-current of the inverse galvanic current both make their exit from the nerve into the *inner* pole, and hence it is not difficult to see that contraction may still attend upon the beginning of the direct current, because the initial extra-current has sufficient strength to act upon the impaired incitability of the nerve, and that contraction may not attend upon the ending of the inverse current, because the terminal extra-current has not sufficient strength to act upon the impaired incitability of the nerve.

In the fourth period all contraction is at an end—at an end because now the incitability of the nerve has departed from all parts included within the circuit.

In this way, then, without taking into consideration the inverse or direct course of the galvanic current along the nerve, the presence or absence of contraction at the opening or closing of the circuit may be accounted for, and the natural conclusion would seem to be that this contraction is brought about by the extra-currents in precisely the same way as that in which it is brought about by induced currents and by discharges of statical electricity.

Nor is there any evidence of a contradictory character in the background.

After what has been said it is easy to understand how it is that there may be contraction both at the beginning and ending of a strong current, when (in consequence of the incitability of the nerve having become impaired in the

manner which has been described) a weak current may be attended by contraction at its ending only or at its beginning only; for the nerve, which is not incitable enough to respond at both poles to the action of the weak extra-currents connected with the weak galvanic current, may still be sufficiently incitable to respond at both poles to the stronger extra-currents of the stronger galvanic current.

It is easy, also, to account for the remarkable fact recently discovered by M. Claude Bernard,* that contraction occurs at the beginning of the direct and inverse currents, and at this time only, if the nerve retain the full measure of its incitability, and if the current used be of the very feeblest. It is easy to do this, for with a *very weak* current it may be supposed that the initial extra-current may still have strength to react upon the nerve when the terminal extra-current, which is always far weaker than the initial, is too weak to have this power.

The law, moreover, would seem to be the same in every particular in those complicated cases in which, as in the case where, one pole being applied to one foot and the other pole to the other foot of a frog prepared as Galvani prepared the parts, the current has to pass through a compound conductor of muscles and nerves. In this experiment it is necessary to realise the fact that the conducting powers of nerve and muscle are very different, and that in passing from nerve to muscle and from muscle to nerve, the current is in reality passing through different bodies, each of which has its own special points at which the current enters and emerges. The problem is more complicated, but M. Chauveau shows very clearly that it may be solved satisfactorily when a clear idea is obtained of the points at which the electricity enters and emerges in the several parts of the compound circuit, and

* 'Leçons sur la Physiologie et la Pathologie du Système Nerveux,' 8vo, Paris, 1858, vol. i, p. 163.

of the condition of the nerves as to incitability at the points of emergence.

And so, also, in those cases in which, as M. Rousseau (de Verzy) has so well shown,* the contraction is due to the action of a *derived* current, for here, as heretofore, the facts show that the nerve is acted upon or not acted upon according as the derived current, or rather the extra-currents belonging to the derived current, make their exit from the nerve at a point or points where the nerve retains or does not retain its incitability. Here, as heretofore, the inverse or direct course of the derived current along the nerve is not found to play that essential part in the problem which it is supposed to play.

Nay, it would even seem that the action of the galvanic current upon sensation is equally irrespective of the centripetal or centrifugal course of this current along the nerve. Thus, in the case where the sciatic nerve of a rabbit is looped over the poles of a Daniell's cell, what happens is simply this—that motion and sensation are produced irrespectively of the direction of the current at the moment when the circuit is closed or at the moment when the circuit is opened, so long as the portion of nerve intervening between the poles retains the power of freely transmitting impressions; and that, afterwards, when the intervening portion of nerve is no longer equal to the free transmission of impressions, then pain, and pain only, is produced when the extra-current makes its exit from the nerve into the pole which is nearest to the sensorium, and motion, and motion only, when the extra-current makes its exit from the nerve into the pole which is nearest to the muscles.

It is also shown, very clearly, that the contraction which is brought about by the action of the electricity which is

* Claude Bernard, 'Leçons sur la Physiologie et la Pathologie du Système Nerveux,' 8vo, Paris, 1858, tome i, p. 171.

inherent in muscle and nerve is in every respect obedient to one and the same law.

It is, however, full time to pass on to the second part of the present inquiry, and I must therefore content myself by referring to the original papers for the facts and arguments which are not introduced into this free sketch of M. Chauveau's most admirable investigations.

II. *Of muscular motion in relation to the electricity which is inherent in living muscle and nerve.*

To Galvani* belongs the honour of being the first to indicate by experiment the probable existence of electrical currents in muscle and nerve. Having prepared the hinder parts of a frog in his usual manner, this physiologist saw the limbs convulsed when the lumbar nerves and the muscles of the thigh were connected by a conducting arc of metal, when these nerves were raised upon a glass rod and brought round so as to touch the thigh, and when the nerves of the two limbs were brought into contact; and, seeing these movements, he rightly argued that they were due to the action of animal electricity, opposing the last experiment—that in which the contractions were produced by simply bringing homogeneous nerves into contact—as an answer to the objection of Volta,† that the acting electricity was produced at the moment by the contact of heterogeneous bodies—metal and animal tissue, or muscle and nerve. It is, however, to MM. Matteucci‡ and Du Bois-Reymond§ that we are indebted for the more exact proofs which are required by modern science as to the existence of electrical currents in muscle and

* 'De viribus electricitatis in motu musculari commentarius,' Bologna, 1791.

† 'Collezione dell' opere,' &c., Florence, 1816.

‡ 'Traité des phénomènes électro-physiologiques des animaux,' &c., Paris, 1844.

§ 'Untersuchungen über thierische Electricität,' Berlin, 1848 and 1849.

nerve, and to both these physiologists, but particularly to the latter, a high meed of praise is due. To Professor du Bois-Reymond, indeed, we are indebted, not only for demonstrating in many animals the presence* of a nerve-current in the smallest fragment of nerve-tissue—in the brain, spinal cord, and other great nervous centres, in sensory, motor, and mixed nerves—but also for tracing the current in muscle, which had been already detected by Professor Matteucci, from the entire muscle to the single primitive fasciculus, and for showing that Nobili's "current of the frog,"* instead of being peculiar to this animal, is nothing more than the outflowing of the muscular current of this animal. To him we are indebted for the knowledge that the law of the muscular current in the frog is one and the same with the law of this current in man, rabbits, guinea-pigs and mice, in pigeons and sparrows, in tortoises, lizards, adders, slow-worms, toads, tadpoles, and salamanders, in tench, in fresh-water crabs, in earth-worms—in creatures belonging to every department of the animal kingdom, and that the law of the muscular current agrees with the law of the nerve-current in all particulars, and also with that of the feeble currents—most feeble as compared with the currents of muscle and nerve—which play in tendinous and all other living animal tissues. To him we are also indebted for the discovery of the changes in the muscular and nerve-currents which occur after death, during ordinary muscular contraction, and under the operation of continuous galvanic currents—changes which are of fundamental importance in clearing up much that is dark in the physiology of muscular motion, and to which attention will have to be directed once and again in the pages immediately following. To him, in a word, we are indebted for almost all that is exact and valuable in our knowledge of the muscular and nerve-currents.

* 'Memorie ed Osservazione edite ed inedite,' &c., Florence, 1834.

1. With a proper galvanometer* there is no difficulty in finding unmistakable evidence of the presence of electrical currents in incitable tissues. In the frog, for example, a current, which Nobili thought to be peculiar to the animal, passes from the feet upwards through the limbs and trunk towards the head. In a single muscle, a current, which is called the muscular current, passes *within the muscle* from the tendinous end or ends to the sides of the muscular fibres, or—what is practically the same thing—to the nerve of the muscle, for within the muscle the branches of the nerve are in relation to the sides of the muscular fibres. If the tendinous end or ends be removed by making a transverse section through the muscular fibres, this current becomes more powerful without undergoing any change of direction; and thus it would appear that this muscular current is independent of the tendon, and that its course *within the muscle* is from the ends to the sides of the fibres. In a single nerve, the current, which is called the nerve-current, passes *within the nerve* from the transverse section to the sides of the nervous fibres—passes, that is to say, from the ends to the sides of these fibres. These currents are also found without any difficulty in the very smallest fragments of muscle and nerve, and the general current of a limb, or of the whole animal, is nothing more than the resultant of the special currents belonging to the

* My galvanometer was made by Mr. Becker (Elliot Brothers, 30, Strand), after the pattern of the one used by M. du Bois-Reymond. The coil is composed of 1 lb. 11 ounces of wire, gauge No. 32; the layers of the coil are 154; the number of coilings are 20,020, or upwards of three English miles. The needles are cylindrical with each end sharpened out into a long point; the connecting piece is made of aluminium instead of tortoise-shell, as in M. du Bois-Reymond's instrument—a difference by which the astatic system becomes a little lighter, 4.5 grains instead of 4.9 grains. At first I used the electrodes used by M. du Bois-Reymond—platinum plates immersed in a saturated solution of common salt; lately I have preferred the electrodes recommended by M. Jules Regnault, and adopted by M. Matteucci—plates of amalgamated zinc immersed in a saturated solution of sulphate of zinc.

individual muscles and nerves, or rather to the individual particles of the fibres of the muscles and nerves, with some small addition from the currents belonging to other living tissues; for, in an electrical point of view, muscle and nerve are only peculiar, in the fact that the currents are far stronger in them than in any other tissue. In addition to the currents which pass *within* the muscle and nerve, from the ends towards the side of the fibres, it is found also that feebler currents pass internally between different points upon the ends or upon the sides of these fibres, and that the direction of these feebler currents is towards the point which is nearest to the central point of the side or ends of the fibres. It is found, too, that all these currents die out as the muscles and nerves lose their incitability, that their last trace has disappeared before the occurrence of rigor mortis, and that their direction may change (perhaps more than once) when they are wavering upon the verge of final extinction. In all these particulars it is found, in fact, that the muscular and nerve currents are obedient to one and the same law.

It is the simple fact, therefore, that rigor mortis is absent so long as the muscular and nerve currents are present, and that rigor mortis is present as soon as these currents are absent; and this is the first point to which I would direct attention as likely to bear prominently upon the interpretation of muscular motion from an electrical point of view.

2. In ordinary muscular contraction there is also good reason to believe that a similar change passes over the muscular and nerve-currents to that which passes over them in rigor mortis.

In one of the beautiful experiments by which M. du Bois-Reymond* demonstrates the change which comes over the muscular current in ordinary muscular contraction, the

* 'Untersuchungen,' &c., vol. ii, p. 50 and p. 59.

first thing is to remove the gastrocnemius of a frog with a long portion of the nerve remaining in attachment. After this, the next thing is to include the muscle within the circuit of the galvanometer, and to place the nerve in such relation to the poles of an induction coil that a series of shocks may be passed through it when it is desired to throw the muscle into a state of tetanus. This arrangement having been made, and the needle having taken up the position of divergence corresponding to the force of the current which is passing through the coil from the *relaxed* muscle, the muscle is thrown into a state of tetanus by passing a series of induction shocks through the end of the nerve. This is the experiment; the result is this—that the needle, which had diverged to a considerable distance from zero under the current of the *relaxed* muscle, swings back, and for a moment or two passes to the other side of zero, when contraction is produced, and when, consequently, the current passing through the coil, and acting upon the needle, is derived from the *contracted* muscle. At first sight, it seems as if the muscular current had been reversed during contraction; but that this is not the true explanation of the backward movement of the needle during contraction is made evident by modifying the experiment in a very simple manner. In this instance, after having measured the strength and ascertained the direction of the current of the *relaxed* muscle, the circuit of the galvanometer is opened, and the needle allowed to return to rest at zero. The muscle is then tetanized by passing a series of induction shocks through the nerve, and when the contraction is fully established, the circuit of the galvanometer is immediately closed. In this instance, that is to say, the current of the *contracting* muscle is made to act upon the galvanometer with the needle at zero, and not, as in the first instance, with the needle already diverging under the action of the current of the *relaxed* muscle. And what

is now the result? The result is—that the needle moves under the current of the contracted muscle in the same direction as that in which it moved under the current of the relaxed muscle, but not to the same distance from zero. In other words, the muscular current is *weakened during contraction*. And thus it is evident that the backward movement of the needle, when, as in the experiment as first described, a relaxed muscle is made to contract without first breaking the circuit of the galvanometer, is really due to the needle having been left to oscillate back to zero by the weakening of the current which had previously kept it away from zero. I find, also, that the needle moves back to zero under these circumstances *more slowly* than it does when left to move from the same point in the movement of simple oscillation; and thus, in the simple rate of motion, I have an additional proof that the needle is not acted upon by a reverse current during contraction, for if it were so acted upon it is evident that the motion derived from this current must be added to that belonging to the backward movement of oscillation, and that the needle under the double impulse must swing back with proportionately increased velocity. In a word, I know no means of accounting for the fact of the needle moving *more slowly* back to zero under these circumstances than by supposing that the backward movement of oscillation is resisted by some remains of the current of the relaxed muscle; and thus the mere rate at which the needle swings back during contraction may, in itself, point to a weakening of the muscular current at this time.

In the experiment which best exhibits the change of the nerve-current during muscular contraction,* the ischiatic nerve of a frog is divided in the ham, and dissected out for a sufficient length up towards the spine. Then, having included the nerve within the circuit of the galvanometer by

* 'Untersuchungen,' &c., vol, ii, p. 511.

applying the divided end to one electrode, and a point of the lateral surface to the other, and having allowed the needle to take up the position into which it diverges during the passage of the current, which is derived from the nerve through the coil of the instrument, the animal is poisoned by introducing a few drops of a solution of strychnia under the skin, and all is done that has to be done, except to watch the needle and record the movement which presently happens,—which is that the needle returns three or four degrees nearer to zero during the tetanus, and that it recedes again from zero when the spasms pass off. In other words, the nerve-current is seen to be weakened during contraction.

M. du Bois-Reymond has also established the fact that the same result is brought about in all other cases of muscular contraction—that the muscular and nerve-currents are always weakened during contraction: and thus there is reason to believe that ordinary muscular contraction, as well as rigor mortis, is in some degree connected with the absence of that electricity which is inherent in living and quiescent muscle and nerve. And this is the second point to which I would direct attention as likely to be of service in an inquiry of which the object is to examine the physiology of muscular motion from an electrical point of view.

3. The facts which may be mentioned next in order are some which show that, under certain circumstances, the nerve-current may be strengthened or weakened by the action of a continuous galvanic current, and that these changes have an influence upon the incitability of the nerve, which accords sufficiently well with what has been said about the muscular and nerve-currents in the two preceding sections.

That under certain circumstances the nerve-current may be strengthened or weakened by the action of a con-

tinuous galvanic current, is shown by M. du Bois-Reymond in an experiment* in which a long piece of fresh nerve is so arranged that one portion is included within the circuit of a galvanometer, and another portion laid across the poles of a galvanic pair. In this case, if the two currents, galvanic and nervous, coincide in their direction, the needle of the galvanometer recedes further from zero than it did under the action of the nerve-current singly; if, on the contrary, the two currents do not coincide in direction, the needle returns and takes up a position nearer to zero than that which it had taken up under the action of the nerve-current singly. In other words, the nerve-current is weakened if the two currents differ, and strengthened if they coincide, in direction.

The next fact is one which at first sight appears to have little to do with the one just mentioned—little to do with the electricity of the nerve—but that it is not cited out of place will appear in the sequel. The fact itself is furnished by Professor Eckhard,† of Giessen, in an experiment which will be rendered more easily intelligible by means of the accompanying tables. In the experiment, two different portions of the nerve of a rheoscopic limb are subjected simultaneously, one to the action of a series of shocks from an induction coil, the other to the action of a continuous galvanic current. In the table, each one of the horizontal columns A and B represents the nerve and the muscles acted upon, the divisions to the left and right of the perpendicular dotted line being assigned to the nerve and muscles respectively, P and N being the points to which are applied the positive and negative poles of the galvanic pair, (I I'), enclosed within brackets, being the points correspond-

* 'Untersuchungen,' &c., vol. ii, p. 292.

† 'Beiträge zur Anatomie und Physiologie,' Giessen, 1858, Erster Band, p. 23, &c., and Althaus's 'Treatise on Medical Electricity,' 8vo, London, 1859, p. 111.

ing to the poles of the induction coil, the word tetanus or a cipher showing whether the induced currents do or do not throw the muscles of the limb into a state of contraction. In the experiment, as is apparent in the table, the portion of nerve included between the poles of the induction coil is nearer to the muscles than the portion included between the poles of the galvanic pair. In the first instance, as is represented in the upper column A, the galvanic current

	Nerve.	Muscle.
A	N ← P (I I')	0
B	P → N (I I')	Tetanus.

is *inverse*, or from the muscles, and there is no contraction; in the second instance, as is seen in the under column B, the galvanic current is *direct*, or towards the muscles, and a state of tetanus is the result. Nor is the absence or presence of tetanus at all dependent upon the order in which the continuous and instantaneous currents are applied to the nerve. If the induced currents are applied first, the contractions resulting from their action upon the nerve continue without any appreciable change when the action of the direct galvanic current is superadded, and at once come to a stop under the action of the inverse galvanic current; if, on the other hand, the galvanic current be applied first, the induction shocks are found to have the power of producing contraction when this current is direct, but not when it is inverse.

It is possible also to modify this interesting experiment in such a way as to make it probable that the *direct* galvanic current is really favorable to the production of tetanus. In this case the tetanizing agent employed by M. Eckhard is a drop of a strong solution of salt. First

of all, the muscles are thrown into a state of contraction by applying this drop to the nerve; in the next place the drop is diluted with water until it loses the power of keeping up the state of contraction; after this, the direct and inverse galvanic currents are passed in turn along the nerve. What precedes and what follows the passage of these currents will be rendered more easily intelligible by means of a table constructed upon the same plan as the last. In column A the nerve is acted upon by the strong

	Nerve.	Muscle.
A	(strong solution of salt)	Tetanus.
B	(dilute solution of salt)	—
C	P → N (dilute solution of salt)	Tetanus.
D	N ← P (dilute solution of salt)	—

solution of salt, and tetanus is the result; in column B the solution of salt acting upon the nerve is diluted until it loses the power of keeping up the state of tetanus; in columns C and D the action of the galvanic current is superadded, and tetanus is found to be present when, as in column C, the current is direct, and absent when, as in column D, the current is inverse. It would seem, indeed as if the direct current had a positive power of favouring contraction, for the solution of salt which, as in column B, was too dilute to keep up the state of tetanus which, was kept up by the stronger solution, as in column A, is found to have this power when, as in column C, the action of the direct current is superadded to that of the salt. And this is the only point requiring notice, for that the tetanus should again disappear in column D is not to be wondered at, seeing that the shocks of an induction coil have been seen to lose their power of producing contraction

when the nerve is under the influence of the *inverse* galvanic current.

How, then, are these very different effects of the action of the direct and inverse galvanic currents to be accounted for? Is it by supposing that the irritability of the nerve is exalted by the direct and paralysed by the inverse current? This is the explanation which has been offered, but it is one which cannot be regarded as satisfactory. On the contrary—to say nothing of the tenor of the evidence advanced in the two preceding chapters—there is a fact furnished long ago* by Ritter which of itself appears to necessitate a different conclusion.

The fact in question is the one which explains how, as in the so-called *voltæic alternatives*, the contraction which attends upon the beginning or ending of a galvanic current may be more than once recalled after it has departed, by reversing the direction of the current along the nerve. The fact in question is to be found in an experiment in which a galvanic current of ordinary strength is allowed to pass continuously for twenty or thirty minutes through the hinder parts of a frog, which hinder parts have been prepared after the manner of Galvani, and arranged so that one leg and one pole are in one glass of water, and the other leg and the other pole in another glass of water,—an arrangement in which the current is inverse in the nerve of one leg and direct in the nerve of the other leg. At the end of the twenty or thirty minutes the conditions of the two nerves are in marked contrast, the incitability of the one in which the current has been inverse having undergone little or no change, the incitability of the other in which the current has been direct being almost or altogether extinguished. It is found, also, that the nerve which has thus lost its incitability under the prolonged action of the *direct* current will more than once recover itself in

* 'Beweis dass ein selbstständiger Galvanismus,' &c., Weimar, 1798.

this respect under the prolonged action of the *inverse* current—an experiment which is easily carried on by reversing the position of the poles in the glasses. The experiment under consideration is one, indeed, which seems to be altogether fatal to the idea of the irritability of the nerve being exalted by the action of the direct current, and paralysed by the action of the inverse current; and, this being the case, it follows that the absence or presence of contraction in these experiments will not be sufficiently accounted for by supposing that the irritability of the nerve is paralysed when the current is inverse and exalted when the current is direct.

In seeking for another explanation of the phenomena which are so conspicuously brought to light in these experiments, the thoughts naturally pass from the irritability to the electricity of the nerve, and the question arises whether the nerve-current in the sciatic nerve of a frog (the nerve acted upon in the experiments) may not be affected differently by the direct and inverse galvanic currents, and whether in this way it may not be possible, in some degree, to account for contraction being absent in one case and present in the other. Such questions are suggested by the experiment of M. du Bois-Reymond, which was related immediately before the experiments of M. Eckhard; such questions are hinted at by the considerations which have gone before.

That the nerve-current of the sciatic nerve of a frog is affected differently by the direct and inverse galvanic currents is not only probable, but certain. The way in which the needle moves when a portion of this nerve is included within the circuit of the galvanometer, shows very clearly that the general current of the nerve, like the general nervo-muscular current of the whole limb, has an upward or *inverse* direction. The way in which the needle moves when, without disturbing the previous relations to

the galvanometer, another portion of the same nerve is subjected to the action of a galvanic cell, shows not less clearly that the nerve-current is strengthened or weakened according as its direction happens to agree or disagree with that of the current proceeding from the cell, the previous deflection of the needle being increased by the *inverse* galvanic current and decreased by the direct galvanic current. It is, indeed, precisely as it might be expected to be,—for in the last-mentioned experiment of M. du Bois-Reymond it has been shown that the nerve-current is strengthened when its direction coincides with that of the galvanic current, and weakened when its direction does not so coincide.

That the nerve-current of the sciatic nerve of a frog is affected differently by the direct and inverse galvanic current is therefore certain; that it is affected in a manner which may, in some degree, explain why it is that contraction is absent under the action of the inverse current, and present under the action of the direct current, is by no means improbable. For what are the facts? The facts are simply these, that contraction is absent when the nerve-current of the nerve acted upon is strengthened by the galvanic current, and present when the nerve-current is weakened by this current. The facts, indeed, are in accordance with the premises, for has it not been seen that the nerve current is weakened in ordinary muscular contraction, and extinguished in rigor mortis? Nor is this view of the matter invalidated by the fact that the muscles do not remain contracted when, as in Ritter's experiment, the nerve-current (this dies out *pari passu* with the incitability of the nerve) has become completely suspended by the prolonged action of the direct galvanic current; for this absence of permanent contraction may be sufficiently explained by the presence of the muscular current—for it is a fact that this current is little, if at all, affected by the prolonged passage of the galvanic current along the nerve.

Nor is it necessary to pass from the same electrical point of view in order to understand, in some degree, at least, how, in the experiments under consideration, distant muscles are thrown into a state of contraction by acting upon the trunk of the nerve. On the contrary, the answer which may be obtained by thus regarding the question, appears to be much more satisfactory than that in which the irritability of the nerve is made to play the most important figure.

There is every reason to believe that the living nerve consists of an infinite number of electro-motive molecules, and that the nerve-current is the sum of the actions of these molecules. There is every reason to believe that the activity of the nerve-current is in proportion to the number and energy of these molecules, and that any change of the nerve-current in any part of the nerve will make itself felt, without loss of time, in every part of the nerve, provided the nerve retain its properties as a conductor. There is every reason to believe, also, that the nerve-current cannot begin or end, or experience any change of intensity, without immediately giving rise to the development of instantaneous currents of high tension in the nerve itself (extra-currents) and in the neighbourhood of the nerve (induced currents). In short, there is every reason to believe that the nerve-current will behave in all respects like the galvanic current; for these two currents are analogous, if not identical, in all their characters.

When, therefore, the trunk of the sciatic nerve is acted upon by an induction coil, or by a drop of salt water, it is easy to understand that the nerve-current will have to respond to this action throughout the whole course of the nerve. Under the *pressure of the poles* of the induction coil, some of the electro-motive molecules of the nerve will be pushed asunder, and the nerve-current will be weakened in a corresponding degree—weakened because a certain

number of its electro-motive molecules are pushed out of the range in which they react most effectively, and intensify each other's action most fully. Under *the action of the induced currents proceeding from the coil*, a mechanical commotion will be set up in the nerve; and this commotion will also weaken the nerve-current, partly by deranging the proper relative position of the electro-motive molecules, and partly by disturbing, either temporarily or permanently, the composite structure of these molecules. Moreover, the nerve-current cannot be weakened without giving rise to the development of *extra-currents* within the nerve—of currents, that is to say, which, in instantaneousness and high tension, are precisely akin to the induced currents proceeding from the coil;—and hence another cause by which the nerve-current is weakened, for, like induced currents, these extra-currents cannot pass along the nerve without deranging and disturbing the electro-motive molecules of the nerve. It is evident, also, that this change in the intensity of the nerve-current must spread along the whole course of the nerve, provided the nerve retain its natural properties as a conductor of electricity. In a word, there is no difficulty in accounting for that weakening of the nerve-current which has been seen to happen concurrently with contraction, and which happens equally when the nerve is occupied with the transmission of sensations. Nor is the case different when the action of a drop of salt water is substituted for that of the induction coil; for,—by abstracting water from the nerve, by combining chemically with the nerve, or by acting in some more recondite manner—the salt may weaken the nerve-current, partly by interfering directly with the proper action of a certain number of electro-motive molecules in the part acted upon, and partly by the mechanical commotion which is set up in the nerve, under the passage of the extra-current, which arises in the nerve as a further consequence of the direct weakening of the nerve-current.

It is to be supposed, also, that the weakening of the nerve-current which arises under these circumstances will give rise to *induced currents* in the neighbourhood of the nerve, as well as to extra-currents in the substance of the nerve itself; and that these induced currents—which in instantaneousness and high tension are precisely similar to the induced currents proceeding from the coil—will be manifested along the whole course of the nerve, so long as the nerve retains its natural properties as a good conductor. It is to be supposed, in particular, that the weakening of the nerve-current will necessitate the development of induced currents within the muscle to which the nerve is distributed,—for these muscles are in the neighbourhood of the nerve; and that this development of induced currents within the muscle may be the cause of the contraction which is produced when a distant portion of the trunk of the nerve is subjected to the action of an induction coil, or of a drop of salt water.

Nor is it necessary to call in the aid of muscular irritability in order to account for the contraction which is brought about by the action of these induced currents within the muscle. On the contrary, it is not only possible, but probable, that the real action of the induced currents may be by deranging and disturbing the electro-motive molecules of the muscle in the same way as that in which the induced currents proceeding from the coil have been supposed to derange and disturb the electro-motive molecules of the nerve;—that the muscular current, which is the sum of the actions of these molecules, may be weakened by this derangement and disturbance;—and that the muscle may contract, not because the induced currents have acted as a stimulus to a vital property of irritability, but because they have for the moment suspended the muscular current which antagonizes contraction. It is not only possible, but probable, that the weakening of the muscular current, which has been seen to attend upon this form of contraction,

and which is readily accounted for by the deranging and disturbing action of the induced currents arising from the weakening of the current of the musculo-motor nerve, may be the essential condition of the contraction. It remains to be seen how far this view will be borne out by the facts which have yet to be cited, but in the mean time it is evident that it agrees well enough with what has gone before. For has it not been seen that the muscular current is weakened in ordinary muscular contraction, and extinguished in rigor mortis? And did it not appear, when speaking of the operation of the blood and nervous influence in muscular motion, that the whole body of evidence is against the idea of a vital property of contractility being stimulated into action during contraction?

Why, in the experiments under consideration, the induction coil or the salt should fail to produce contraction when the nerve is simultaneously acted upon by an inverse galvanic current, and why they should not fail to do this when the nerve is simultaneously acted upon by a direct galvanic current, are also problems which are not altogether unintelligible upon the same hypothesis. Under the action of the *inverse* galvanic current, when the nerve-current is strengthened, the addition to the nerve-current may prevent that weakening of the nerve-current which would otherwise result from the action of the induction coil and salt—prevent it, perhaps, partly by compensating for the loss, partly by increasing that tension of the electro-motive molecules which resists this loss. Under the action of the *inverse* galvanic current, that is to say, the nerve-current may be *fixed* in a condition in which it cannot give rise to induced currents, for there can be no such induced currents so long as the inducing current remains constantly at the same level. And hence, perhaps, the absence of contraction. Under the action of the *direct* galvanic current, on the other hand, when the nerve-current is weakened, this

current may be capable of experiencing more decided changes of strength than usual under the action of the induction coil or salt—may be thus capable, perhaps, because the electro-motive elements may have been deprived of some of that tension by which they ordinarily resist the action of any disturbing influence:—and thus it may be that stronger induced currents, and stronger contractions are brought about—stronger induced currents because the strength of these currents will be in direct proportion to the amount of variation in the strength of the inducing nerve-current; stronger contractions because the contraction may be the direct measure of the strength of the induced current acting upon the muscle.

It may be urged, however, that the nerve-current is too feeble to bring about such results, and at first sight there appears to be no little force in this objection. The nerve-current, as measured by the galvanometer, is certainly very feeble, but the portion thus measured is as certainly not the whole nerve-current. On the contrary, this portion may, for anything we know, be altogether insignificant when compared with the amount which is hemmed in within the coats of the nerve, and which is prevented from overflowing by the imperfectly conducting properties of these coats. It would seem also as if a proof positive as to the possibility of strong induced currents being derivable from the nerve-current may be found in the discharges of the torpedo and other electrical fishes, for there is good reason to believe that these discharges are nothing more than induced currents arising at the instant from movements in the currents of the nerves belonging to the electrical organs.

Upon bringing the opposite polar surfaces of the electric organ of a torpedo or other electric fish into connexion with a galvanometer, the needle gives evidence of a momentary current when the discharge takes

place—a current of high tension, as the feelings will sufficiently show if the experimenter care to include himself in the circuit; but in the interval between the shocks the needle remains at zero or thereabouts. Upon further inquiry there is reason to believe that the electricity of the electric organ is supplied by the nerves, and received at the instant when it is wanted. The rich supply of nerves to the transverse partitions between the prisms, the instant renewal of the discharge when the animal is provoked, or when the nerves of the electric organs or the nervous centres connected with those nerves are incited in any manner, the storm of shocks attending the action of a poison which, like strychnia, has a special action upon the nervous system—all point to the nervous system as the source from which the electric organ derives its electricity. The position of the needle of the galvanometer at zero or thereabouts in the intervals between the shocks, and the absence which there must be in the moist membranous organ of an animal living in water of the means of insulation, without which it is simply impossible to suppose that electricity of high tension could remain stored up beyond a single instant, point as plainly as can be to the electricity being received by the electric organ at the instant it is wanted. The facts, indeed, are calculated to show that instantaneous currents of high tension may originate in the action of the nerves of the electric organ. They show, as it would seem, that there was good ground for supposing, as was supposed, that induced currents might originate in movements of the nerve-current, for the whole history of the discharges of the torpedo and other electrical fishes is intelligible upon the theory of induced currents arising in movements of the currents of the nerves of the electric organ, and altogether unintelligible upon any other theory. In a word, they furnish an answer to the objection that the current in the musculo-motor nerve is too feeble to act upon the muscle by the induced currents to which it may

give rise, for be this current never so feeble, it is at least as strong as the current of the nerve of the electric organ.

There are, moreover, certain analogies which justify an appeal to these facts in the present case. Thus, the nerves of the electric organ arise from the anterior tract of the spinal cord, and terminate in loop-like plexuses; and so do the nerves of the muscles. The electric organs are paralysed by the division of their nerves; and so are the muscles. The electric organs are made to deliver their shock by inciting the ends of the divided nerves which remain in connexion with the organ, and the discharge is limited to the region to which the incited portion of nerve belongs; the muscles are made to contract under similar circumstances, and the contraction is equally localized. The electric organs are exhausted by exercise and recruited by rest; so are the muscles. The action of strychnia upon the electric organs and upon the muscles agrees in this, that it gives rise to a storm of involuntary tetanic spasms in the one case, and to a storm of involuntary discharges in the other. And, lastly, there is a similar agreement in the effects of the action of a galvanic current upon the nerves of the two organs, for the attending shocks or contractions are equally confined to the moments when the circuit is closed or opened, and equally banished from the interval during which the current is passing continuously.

It would seem, also, as if the phenomena of *secondary contraction*, discovered by MM. Matteucci and Du Bois-Reymond, may be looked upon as a direct argument in favour of the sudden passage of currents of high tension in the neighbourhood of a musculo-motor nerve concurrently with that change in the nerve which determines contraction. When three or four rheoscopic limbs are arranged in a series in which the nerve of the second is placed upon the muscles of the first, the nerve of the third upon the muscles of the second, and so on, nerve upon muscle for the succeeding limb or limbs, secondary contraction takes place in the

second, third, or fourth limb, whenever the first limb is made to contract. Secondary contraction is also exhibited in a rheoscopic limb of which the nerve is laid upon the *nerve* of another rheoscopic limb, when the latter limb is thrown into a state of contraction. The phenomenon is very curious, but it is one which may be readily explained by supposing that the contraction is attended by induced currents which pass, as such currents may and must pass, beyond the range of the contracting muscle into that of the nerve lying upon the contracting muscle, and *vice versá*. It is one which appears to admit of no other explanation than this, for in the case where the contraction is produced by placing nerve upon *nerve*, the only other explanation which deserves attention is excluded, namely, that which supposes that the nerve of the limb exhibiting the secondary contraction has been incited mechanically by the motion of the contracting muscle. In a word, the phenomenon of secondary contraction, interpreted by what has gone before, would seem to furnish a direct argument in favour of the actual presence of induced currents in the neighbourhood of the nerve, when there is that change in the nerve-current which determines contraction. It would seem, indeed, that the nerve is for the nonce placed in the position of the electric organ, specially so called, and that the secondary contraction is the natural effect of the discharge.

— And if this be the way in which contraction is brought about when the nerve of a muscle is subjected to the action of electricity, it is to be supposed that this will also be the way in which contraction is brought about when muscle is subjected to the direct action of electricity. It is to be supposed, that is to say, that induced currents, and discharges of statical electricity, and extra-currents will produce contraction, not by acting as a stimulus to a vital property of muscular irritability, but simply by deranging and disturbing the polar condition of the muscle, and in

that way removing the influence which antagonises contraction.

3. In speaking of the way in which the nerve was affected by the direct and inverse galvanic currents, no mention was made of certain facts which, at first sight, may seem to modify the conclusion then arrived at. To have considered these facts in that place, however, would have added not a little to the complication of an argument already too complicated, and for this reason, as well as on account of their own intrinsic importance, it seemed to be better to place them in a section by themselves.

Of these facts those which may be mentioned first in order are furnished by Professor Eckhard * in some experiments which differ from those already quoted only in this, that the position of the poles of the induction coil and of the galvanic pair upon the nerve are relatively different. In the former instance, the portion of the nerve acted upon by the induced currents was between the muscles and the portion of nerve included between the poles of the galvanic pair; in the present instance, the poles of the induction coil are applied, as the accompanying table will show at a glance, either between the poles of the galvanic pair, as in columns A and B, or upon a portion of nerve which is more distant from the muscles than that to which the last-named poles are applied, as in columns C and D.

	Nerve.	Muscle.
A	P → (I I') → N	No tetanus.
B	N ← (I I') ← P	No tetanus.
C	(I I') P → N	No tetanus.
D	(I I') N ← P	No tetanus.

* 'Beitrage,' &c., Erster Bd., p. 23, &c.

The relations of the poles, as represented in the four columns, are different in each case, and yet there is no difference in the result, for contraction is equally absent in the cases where, as in columns A and C, the galvanic current is direct, and in the cases where, as in columns B and D, the galvanic current is inverse.

Another fact, belonging evidently to the same category, is to be found in the behaviour of the spinal cord under the action of a constant galvanic current. Under ordinary circumstances, as is well known, violent convulsions of the body and limbs are produced by inciting the spinal cord in any way; but it is not so well known that it is possible to prevent the incitation from acting in this manner by passing a continuous galvanic current along the cord. Of the fact, however, there can be no doubt. At the moment when the spinal cord is included within a galvanic current, and at the moment of breaking the circuit, there is a convulsive shock in the muscles receiving nerves from the cord; in the interval between these moments these muscles are not only at rest, but the circumstances are so altered that it is no longer possible to provoke them to contraction by acting upon the cord. Even induced currents are powerless in this respect. It is of no moment, also, whether the course of the galvanic current be direct or inverse, for in either case it is equally impossible to produce any impression upon the muscles by inciting that portion of the cord which is between or above the galvanic poles. In order to obtain this result, however, it is necessary to apply the incitation to a part of the cord along or above which the galvanic current is actually passing; for if it be applied to the cord *below* the part which is included between the galvanic poles, or to a nerve after it has left the cord, the direction of the current is found to tell upon the muscle in the same way as that in which it told in M. Eckhard's first experiment, the inverse

current counteracting and the direct current favouring contraction.

The last fact which has to be mentioned is supplied by Professor Matteucci,* in certain experiments in which a constant galvanic current is passed along the spine of frogs poisoned with strychnia. In some of these animals tetanus had supervened before the circuit was closed, in others the muscles were still relaxed. In the former the spasms passed off as soon as the circuit was closed, and the muscles remained in a relaxed state so long as the current continued to pass continuously; in the latter the animal died in the course of time from the effects of the poison, but without manifesting the slightest trace of muscular rigidity if care were taken to pass the current steadily throughout the whole experiment. M. Matteucci also refers to the case of a man suffering from tetanus, who became able to open his jaws, to breathe freely, and to move his body and limbs without much difficulty, so long as a strong galvanic current was passed along the spine from the occiput to the sacrum.

In reflecting upon these interesting facts it is easy, without proceeding very far, to find additional reason for believing that the *direct* galvanic current does not augment the irritability of the nerve, for, if it did, it is obvious that contraction could not be absent in the experiments of M. Eckhard which are represented in columns A and C of the last table, or in the experiments upon the spinal cord in which tetanus either fails to appear or else disappears under the action of the direct current. It would also seem as if the absence or disappearance of contraction in these several experiments, may be more readily explained by referring to the electricity of the nerve than by referring to the irritability of the nerve. It has been argued that instantaneous currents of high tension are induced in the muscle

* 'Traité des Phénomènes Electro-Physiologiques,' &c.

and elsewhere in the neighbourhood of a nerve by any change in the strength of the nerve-current, and that contraction is the sign of the action of these induced currents upon the muscle. It may now be argued that contraction is absent, or that contraction disappears, in the experiments under consideration, because the artificial current prevents those changes in the strength of the nerve-current which are necessary to the production of induced currents in the muscle and elsewhere in the neighbourhood of the nerve—prevents these changes because, under these circumstances, the weak nerve-current is, as it were, fixed at the same constant level as that of the constant galvanic current by which it is overruled. And thus there is no necessity to alter the conclusion arrived at in the last section. On the contrary, there is additional reason for its adoption in that it would now appear that contraction is prevented or put an end to in cases where the changes in the nerve-current which give rise to induced currents are prevented or put an end to.

— Looking back, then, at the arguments which have been advanced in the present chapter, it would seem to be no improbable idea that muscular elongation is coincident with the presence of electrical action in muscle and nerve, and that muscular contraction is coincident with the absence of this action. It would seem to be no improbable idea that there is a state of polarity in living muscle during relaxation which produces relaxation, and that contraction is nothing more than the necessary result of the muscle being liberated from this state, and left to the operation of the attractive force which is inherent in the physical constitution of the muscular molecules.

After death, the simple fact is, that the muscles are relaxed so long as the muscular and nerve-currents continue to be manifested, and contracted as soon as these currents

have taken their departure; and hence there is no difficulty in supposing that this form of contraction is antagonised by the state of polarity, and that the muscle contracts as a matter of course when it is freed from this state and left to itself. The difficulty, indeed, is to entertain any other supposition.

With respect to the transitory contraction of the incitable muscle, the case is not quite so plain, but, when examined into, it is found to be not unlike that of rigor mortis in its essential features. It is found that the muscular and nerve-currents are weakened during this form of muscular contraction. It is found that the incitability of a nerve is diminished when the nerve-current is augmented by passing a continuous galvanic current in the same direction, and augmented when the nerve-current is diminished by passing a galvanic current in the opposite direction. It is found that momentary currents of high tension—discharges of statical electricity, induced currents, extra-currents—are necessary to throw an incitable muscle into a state of contraction, and there is reason to believe that these currents and discharges act by the mechanical commotion which they set up in the muscular and nervous fibre. It is found that instantaneous currents of high tension, induced within the muscle, upon the interruption of the nerve-current, are the probable means by which the nerve exercises its influence in producing contraction, and that there is no contraction when the nerve-current is placed, as it were, in a state of fixity by the galvanic current, because, under these circumstances, it cannot exhibit those movements which are necessary to give rise to induced currents. It is found that these different forms of instantaneous currents of high tension may produce contraction, not by stimulating a vital property of muscular irritability into action, but by deranging and disturbing the electro-motive molecules of the nerve and muscle, and in that way

weakening the nerve and muscular currents which are the sum of the joint actions of these molecules. It is probable, in fact, that the transitory suspension of the polar state of the muscle which is brought about in this manner is the essential condition of the transitory contraction of the incitable muscle. The case differs from, and yet agrees with, that of rigor mortis. The difference is in the muscular polarity being extinguished in the one form of contraction, and only suspended for a moment in the other form; the agreement is in this state of polarity being absent in both forms. In the case of the incitable muscle, contraction must be transitory, and relaxation must be the prominent condition. Contraction must be transitory for a double reason. It must be transitory, because the induced current upon which each particular contraction depends is momentary in duration. It must be transitory, because the nerves which are engaged in producing contraction must lose their incitability after a time, in consequence, perhaps, of the nerve-current becoming reduced to an amount which is too insignificant to allow the development of induced currents of sufficient strength to act upon the muscle. And relaxation must be the prominent condition of the incitable muscle, for contraction must be antagonised by the action of the muscular and nerve-currents, so long as the muscle is left to itself. In the case of rigor mortis, on the contrary, relaxation must be absent, and contraction must be the sole and abiding condition, for the simple reason that that polar state is now at an end which antagonises contraction in the incitable muscle. There is, indeed, an indissoluble bond of connexion between the prolonged contraction of rigor mortis and the transitory contraction of the incitable condition, and there is no wonder if, at times, the one form of contraction should run into the other. There is no wonder, for example, that in animals which have been poisoned by

strychnia, rigor mortis should often follow upon the last tetanic spasm with scarcely any interval.

The view which is here taken of the operation of electricity in muscular motion is indeed one which supports and explains what has already been said respecting the operation of blood and nervous influence in muscular motion. That the blood should antagonise contraction is to be expected if the nerve and muscular currents have the power of producing this effect; for it may be supposed that the chemical changes which are wrought by arterial blood in muscle and nerve are as inseparable from the nerve and muscular currents, as the chemical changes in the galvanic cell are inseparable from the galvanic current. And that nervous influence should antagonise contraction, is assuredly no matter of wonder, if this be the action of the nerve-current. Nay, if this be the action of the nerve-current, it follows that nervous influence must antagonise contraction, for the nerve-current is a component part of, and the most intelligible idea in, nervous influence. Electrically considered, indeed, the operation of nervous influence in muscular motion becomes not a little simplified. Thus, for example, it is not difficult to understand that convulsion may be brought about by arresting the supply of arterial blood to one or more of the nervous centres of the head, and that in the case of death this convulsion should be separated from the final contraction of rigor mortis by an interval of relaxation. For may it not be assumed—that the electrical activity of these centres will fail when arterial blood is withheld,—that this failure will tell upon the whole nervous system, and set up a movement in the nerve-current which will lead to the induction of instantaneous currents of high tension in the muscles and elsewhere in the neighbourhood of the nerves,—that a succession of these induced currents will be developed so long as the nerve-current is oscillating towards the lower level,

which it is obliged to settle into for want of arterial blood, —that convulsion will result from the action of these induced currents upon the muscular fibres, — that relaxation will follow the convulsion when the induced currents cease to be developed, because the muscles will be then left to the undisturbed operation of the muscular and nerve-currents,—and that rigor mortis will come on eventually, because these currents are then extinguished. And does not the fact that an incitable muscle is more prone to contract when it is cut off from the influence of the nervous centres, or when it is deprived of a portion of its proper supply of blood, become somewhat less intelligible when the idea of the nerve-current is substituted for that of nervous influence, and when the arterial blood is supposed to act by producing the nerve and muscular currents? For if these currents antagonise contraction, it will follow, as a matter of course, that there must be a greater proneness to contraction when a large amount of nerve-current is cut off by separating the muscles more or less completely from the nervous centres, or when a diminished supply of arterial blood necessitates a more scanty development of the nerve and muscular currents? In a word, the view which is here taken of the operation of electricity would appear to be the view which is demanded by all that was said in the two preceding chapters, for it furnishes the key which is wanted for the full explanation of this operation of blood and nervous influence in muscular motion.

CHAPTER IV.

ON MUSCULAR MOTION IN CERTAIN OTHER RELATIONS, AND
ON THE THEORY WHICH SEEMS TO APPLY TO SIMPLE
MUSCULAR MOTION.

LOOKING back at the facts and considerations advanced in the three preceding chapters, the conclusion which seems to be forced upon the mind is this—that relaxation, rather than contraction, is the chief peculiarity of incitable muscle. It seems as if there is during relaxation a state of polarity which produces relaxation: it seems as if contraction is nothing more than the necessary result of the muscle being liberated from this state and left to the operation of the attractive force which is inherent in the physical constitution of the muscular molecules.

Looking forward, the facts which come successively into view may appear at first sight to contradict this conclusion. The contraction which is brought about by mechanical, chemical, and other non-electrical "irritation," may appear to be most readily accounted for upon the supposition that a vital property of irritability has been roused into action by the "irritation." The fatigue following muscular action and necessitating rest, the greater readiness with which dead muscle may be torn asunder, and the diminished degree of shortening when muscle contracts after death, may appear at first sight to point to the power of contraction as a vital endowment. The fact that a muscle wastes in proportion to the number of its contractions may appear to show that contraction is the sign of functional activity in the muscle, for the waste of an

gan is proportionate to its functional activity. The diminishing force of the contraction as the muscle shortens upon itself, and the absence of any change of volume in contraction—the gain in breadth being precisely equal to the loss in length—are also facts which do not seem to fall readily within the scope of any physical explanation. And as for voluntary muscular contraction, how is this phenomenon to be accounted for without a vital power of irritability to sympathise with a power so pre-eminently vital as the will? As in many other instances, however, the impression which remains after more closely regarding the subject is very different from that which is received by looking at it from a distance.

1. The contractions which are brought about by non-electrical "irritation" are of a very varied character. The contraction which attends the touch of a glass rod or other inert body is referred to "mechanical irritation," and so, in a partial sense, is the contraction which is exhibited in the hollow viscera under the action of the contents of these viscera. The contraction of the iris in the light, or of any muscle under the action of heat or cold, is referred to the "irritation" produced by the light or heat or cold. The contraction which depends upon the action of certain chemical or medicinal substances is, in like manner, referred to chemical or medicinal "irritation." In each case there is some peculiarity which it is necessary to consider, in order to know what must be known respecting muscular contraction under these circumstances.

It may be gathered from more than one passage in the preceding pages, that there may be another way of accounting for these contractions than that which refers them to "irritation." In speaking upon the action of an induction coil upon the nerve, it has been argued that the mere *pressure* of the poles of the coil upon the nerve will have an action apart from that of the currents proceeding from

the coil; that this pressure will separate in some degree the electro-motive molecules of the nerve in the part pressed upon; that this separation will interrupt and weaken the nerve-current which is the result of the actions of these electro-motive molecules; that the interruption and weakening of the nerve-current will induce instantaneous currents of high tension in the neighbourhood of the nerve; and that the induced currents thus developed around the muscular branches of the nerve may produce contraction by deranging and disturbing the electro-motive molecules of the muscles—by suspending, that is to say, the muscular current whose office it is to counteract contraction. The contraction was accounted for without any mention being made of mechanical “irritation,” as one effect of the pressure of the poles; and as in this case, so also in the cases where contraction is due to the pressure of a glass rod or other inert body, there appears to be no need to mention mechanical “irritation” as an effect of the pressure. In these cases as in that, the nerve-current and the muscular currents are facts which must not be ignored. In these cases as in that, the nerve-current may be interrupted and weakened by the pressure, and the muscular fibre may be acted upon by the induced currents arising in this interruption and weakening. This, apparently, is what may happen when a nerve in connexion with a muscle is subjected to pressure. This is what may happen also when the pressure is applied directly to the muscular fibres, for it is to be supposed that the muscular current will be similarly affected in the part pressed upon, that the pressure will interrupt and weaken the current in this part, that this interruption and weakening of the current here will lead to the induction of instantaneous currents of high tension in the neighbouring fibres, and that these induced currents will give a shock to these fibres which will, for the moment, suspend their muscular current, and in that way

bring about contraction. And as in the case where contraction is brought about by pressing upon the nerve or muscle, so also in those cases in which the contraction is brought about by cutting, tearing, or otherwise injuring the nerve or muscle, for it is easy to perceive that the explanation which applies to the one case will also apply to the other.

It may be doubted, also, 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 accumulates, and the contraction is deferred until the distension is an occasion of discomfort. Up to this point the urine seemed to favour dilatation rather than contraction in the bladder, and at this point it is not a whit more necessary to suppose that the contraction is due to the urine having acted as a stimulus to the irritability of the coats of the bladder. When the bladder is full, there is a feeling of uneasiness, which shows that the distension is acting upon the afferent nerves and producing a change in them, which, reflected through the efferent nerves belonging to the same nervous arcs, may act upon the bladder; but it does not follow from this feeling of uneasiness that the irritability of the afferent and efferent nerves concerned in this process is stimulated into action under the "irritation" arising from the bladder being distended with urine. On the contrary, this uncomfortable sensation may show that the afferent nerves are becoming exhausted,—for each sensation implies an equivalent expenditure of nervous influence; and this exhaustion of the afferent nerve may imply a similar state of things in the efferent nerve corresponding thereto,—for the nerves belonging to the same reflex arc are so bound up together that it is difficult to imagine a change in one which does not extend to the other; and thus it may be that the bladder contracts under these circumstances because its

nerves are so exhausted as not to supply sufficient nervous influence to antagonise contraction. The uncomfortable sensation arising from the bladder being distended with urine, may also show that changes are being produced in the nerve-current of the afferent nerves of the bladder, and, through them, in the nerve-current of the efferent nerves belonging thereto, which changes will give rise to induced currents among the muscular fibres of this viscus. There is an experiment of M. du Bois-Reymond,* which shows that the nerve-current is weakened in sensation in the same way as that in which it is weakened in muscular contraction. In this experiment, a rheoscopic limb, from which the skin has not been removed, is placed in one of the arms of a V-shaped glass tube, and the nerve is included in the circuit of the galvanometer. Then, after having waited until the needle has taken up the position to which it diverges under the nerve-current passing through the coil, a boiling solution of salt is poured into the mouth of the other arm of the tube. This is the experiment; the result is, that the needle moves towards zero as the solution rises in the arm of the tube which contains the limb. The nerve-current, that is to say, is weakened by treating the limb in a way which would cause exceeding torture if the nerves were in communication with the sensorium of the animal. In a word, the nerve-current would seem to be weakened during sensation. When, therefore, the bladder has become so full as to give rise to uncomfortable sensations, it is fair to suppose that these sensations imply weakening of the nerve-current of the afferent nerves belonging to the bladder, that this weakening of the nerve-current necessitates the development of induced currents in the neighbourhood of the nerves, and that the induced currents thus developed in the neighbourhood of the afferent nerves within the senso-

* 'Untersuchungen,' &c., vol. ii, p. 520.

rium may, perhaps, be the occasion of the *sensation*. It is also fair to suppose that the afferent nerves of the bladder are so connected with the efferent nerves corresponding thereto, that the nerve-current cannot be weakened in the one without being weakened in the other, and hence it may follow, as a consequence of the weakening of the current in the afferent nerve, that there may be weakening of the current in the efferent nerve—a change which, as already explained, may lead to contraction by the development of induced currents within the muscle. What may happen with respect to the bladder, may happen also with respect to every other hollow viscus, for upon a moment's reflection it will be apparent that there are the same reasons for concluding that the hypothesis of "irritation" on the part of the contents of the viscus, is not necessary in order to explain the contraction of the viscus.

It may be questioned, also, whether contraction is ever due to any "irritation" on the part of light, or heat, or cold.

It is in the darkness, and not in the light, that contraction takes place in the irritable cushions of the sensitive plant; and it may be held, with Bichât, that the pupil enlarges in the dark, not because there is then an end to the "irritation" which had kept certain sphincter fibres in a state of contraction so long as they are acted upon by the light, but because the *iris* has passed out of a state of expansion, which was determined by the action of the light. In this way the movements of the iris in the light and the movement of the cushions of the sensitive plant become kindred phenomena, and the difficulty is at an end which arises from the more than doubtful existence of the sphincter fibres, which are supposed to close the pupil when their irritability is roused into action by the light.

How heat acts in producing contraction may be gathered

from an interesting experiment,* in which M. Eckhard immerses the nerve of a rheoscopic limb in water, and afterwards tests the influence of varying degrees of heat upon the nervous incitability by inciting the nerve with a needle, as the temperature of the water is progressively raised by additions of hot water. At 70° Fahr.—about the natural temperature of the frog—this incitability is not appreciably affected by the heat; from 70° Fahr. to 140° Fahr., it is sensibly impaired by every fresh addition of hot water; at 140° Fahr., or thereabouts, the muscles refuse to respond any longer to the action of the needle upon the nerve; at a temperature higher than this, they contract in obedience to the action of the hot water upon the nerve. In other words, the contraction arising from heat is not produced until the temperature is sufficiently high to have rendered the nerve incapable of responding to the incitation of the needle. I find, also, upon putting a piece of ice into the water in which the nerve is immersed, that the current of the nerve fails in like manner as the temperature of the water falls, and that the contraction arising from cold is not produced until the temperature is sufficiently low to have rendered the nerve incapable of responding to the incitation of the needle. It would seem, in fact, that the muscles do not contract in obedience to the action of heat or cold upon the nerve unless the temperature is high enough or low enough to suspend the nerve-current,—for it is a fact that this current is suspended when the incitability is suspended. At any rate, it is plain that the contraction can scarcely be referred to any “irritation” on the part of the heat or cold, for the “irritability” of the nerve has departed before the contraction is brought about.

How little the doctrine of “irritation” will serve to ex-

* ‘Grundzüge der Physiologie der Nervensystems,’ 8vo, Giessen, 1854, p. 81.

plain satisfactorily the action of strychnia or brucia in producing contraction, may be seen in the experiments of Dr. Harley, to which reference has been made in former pages (pp. 20 and 26),—experiments which show very plainly that these poisons have the power of destroying the incitability of muscle and nerve, and of hastening the occurrence of rigor mortis. How little the doctrine of “irritation” will serve to explain satisfactorily the action of a chemical substance in producing contraction, may be seen in the remarks upon the action of salt, as exhibited in one of M. Eckhard’s experiments, (p. 64,) for these remarks show that the salt may act upon the electricity of the nerve, weakening the nerve-current, and producing contraction by the action upon the muscle of the induced currents consequent upon this weakening. There are also other experiments by the same physiologist, which point to the same conclusion respecting the action of chemical substances in producing contraction. In one of these, after immersing the nerve of a rheoscopic limb in an acid solution, of which the strength is progressively increased by additions of acid, its irritability is tested at intervals by a needle. The experiment is the precise counterpart of the one related in the last paragraph, with this difference, that the additions to the fluid in which the nerve is immersed were of hot water in that case, of acid in this. The result is also the same, for the incitability fails as the acid acting upon the nerve becomes stronger, and the muscle contracts in obedience to the action of the acid upon the nerve, after the nerve has ceased to reply to the incitation of the needle. M. Eckhard has also other experiments of the same kind,* in which various agents are substituted for the acid. The agents themselves acted differently—some by abstracting water from the nerve, some by altering the normal albuminous constituents of the

* ‘Grundzüge,’ &c., p. 82.

nerve, and some in a more recondite manner—but all agreed in destroying the incitability of the nerve as their strength became increased, and in not producing contraction of themselves until this incitability was so far impaired that the muscles of the rheoscopic limb refused to respond any longer to the action of the needle upon the nerve.

—In no one of these cases, then, is it necessary to refer the contraction to “irritation,” and in some of them this mode of explanation is set aside by the fact that the “irritability” upon which the “irritation” must act has died out before the contraction makes its appearance. It would seem, in fact, as if there is nothing in any of these cases which is inconsistent with the view of muscular motion to which the previous facts and considerations appear to lead.

2. The fatigue following muscular action and necessitating rest, the wasting of muscle in proportion to the number of its contractions, the diminished shortening during contraction, and the more ready lacerability of muscle after death, are also facts which may be explained without supposing that the contractile power is a vital endowment.

It may be assumed that each contraction involves a corresponding loss of nerve-current; it may be assumed also that strength of the instantaneous currents of high tension which are induced within the muscles when there is any weakening of the nerve-current, will fail *pari passu* with the nerve-current,—for the strength of the induced current will be in direct proportion to the degree of variation in the strength of the nerve-current; and, if so, then it is possible that a muscle may cease to contract after a time, not because the irritability of the nerve is too fatigued to allow of further contraction until it is revived by rest, but because the nerve-current is too enfeebled to furnish induced currents of sufficient strength to throw the muscles into con-

traction until it is restored by rest. Nor does it follow that the muscles should remain contracted until the nerve-current is restored by rest, for the muscular current, which is less speedily exhausted than the nerve-current, is supposed to keep the incitable muscle in a relaxed state, provided the muscle be left to itself.

It is, no doubt, a fact that the waste of an organ is proportionate to the functional activity of an organ, and that the waste of a muscle is proportionate to the number of its contractions; but it is no necessary consequence of this fact that contraction is the sign of functional activity in a vital property of contractility. On the contrary, this waste may have been incurred in restoring the state of *relaxation*, and, after what has been said, who can say that this is not the case? After what has been said, indeed, the natural inference is that the muscle cannot return to the relaxed state without the aid of the muscular current, and that the muscular current cannot be restored without a corresponding chemical change, that is, waste, in the tissues concerned.

It is very possible, also, that diminished shortening in contraction may be the natural result of the physical circumstances in which a muscle is placed after death. When a muscle contracts during life, the antagonist muscle either relaxes or else opposes no resistance to the contraction. The blood, also, is fluid, and the intra-muscular vessels are easily emptied when pressed upon by the contracting fibres. After death, on the other hand, the contraction of any set of muscles may be resisted by the incipient stiffening of the antagonist muscles, as well as by the coagulation which is taking place, or has taken place, in the contents of the intra-muscular vessels. At any rate, it is certain that there is a greatly increased degree of shortening when, as in the process of crimping under the hand of the fisherman, the muscles are gashed in all directions, so as at once

to put an end to muscular antagonism, and to empty the intra-muscular vessels of blood.

The more ready lacerability of muscle after death may also be nothing more than the natural consequence of the physical circumstances in which the muscle is then placed. After death the muscle may be weakened by the action of those solvent juices which are present in muscle, and which are more or less analogous in their properties to gastric juice. After death the muscle is yielded up to the process of decomposition, and the affinities of the compound *muscular* molecules being weakened by the incipient resolution of these molecules into their component elements, the molecular cohesiveness of the muscle may suffer correspondingly. During life, moreover, any attempt at laceration has the effect of throwing the muscle into a state of spasmodic contraction,—a state in which the greater proximity of the muscular molecules to each other will allow the attractive force which is inherent in the physical constitution of these molecules to operate more effectively than it can operate in a living or dead relaxed muscle, or than it can operate in a muscle which is contracted in the less perfect contraction of rigor mortis.

3. The diminishing force of the contraction as the muscle shortens upon itself, and the absence of any change of volume in contraction—the gain in breadth being precisely equal to the loss in length—are also explicable upon the same principles of interpretation.

It is no doubt true, as M. Schwann pointed out, that the force of muscular contraction decreases as the muscle contracts upon itself; but herein is surely no ground for asserting that the law of muscular contraction is essentially different from the law of any known physical attractive force. Indeed, a piece of india rubber, in shrinking after elongation, behaves in every respect as a contracting muscle behaves in M. Schwann's experiment, and here un-

doubtedly the shrinking is nothing more than a simple physical process.

Nor is an argument in favour of the non-physical character of the law of muscular contraction to be based upon the fact that there is no change of volume in muscular contraction. On the contrary, there are certain experiments by Mr. Joule, of Manchester,* which show that the contraction which takes place in a bar of iron, upon the abstraction of magnetism, is equally unaccompanied by any change of volume.

In one of these experiments a rectangular bar of iron, one fourth of an inch broad by one eighth of an inch thick, is placed in the axis of a coil of insulated copper wire. One end of this bar is fixed, the other end is attached to a system of levers, by which any change of length in the bar is multiplied 3000 times. On magnetizing the bar to saturation, by passing a strong galvanic current through the coil, the index of the multiplying apparatus springs from its position and vibrates about a point one tenth of an inch in advance, a distance giving $\frac{1}{3000}$ th of an inch for the actual elongation of the bar; on interrupting the passage of the current through the coil, and demagnetizing the bar, the index springs back to its original position. Upon closing the galvanic circuit a second time, and leaving it closed, in addition to the sudden springing in advance as it responds to the elongation of the bar under the action of the magnetism, the index advances slowly as the bar expands under the action of the heat radiating from the coil; but this sudden and slow elongation of the bar are not to be confounded, for on demagnetizing the bar at any moment, by breaking the galvanic circuit, the index immediately springs back and vibrates about a point exactly one tenth of an inch lower than that to which it had previously attained.

* 'The Philosophical Magazine,' Feb. and April, 1847.

In another experiment a bar of annealed iron, one yard long and half an inch square, is placed in a glass tube, forty inches long and one inch and a half in diameter, around which is coiled a conductor consisting of ten insulated copper wires, each one twentieth of an inch in diameter, and 110 yards in length. One extremity of this tube is closed, the other is fitted with a stopper, the centre of which is pierced with a graduated capillary tube, of which each division is equal to the $\frac{1}{450000}$ th part of the iron bar. Having filled the principal tube with water, and having adjusted the stopper so as to force this water to a convenient height in the capillary tube, the coil is alternately connected and disconnected with a Daniell's battery of five or six cells,—an apparatus of sufficient power to magnetize the iron bar to the full. This is the experiment; the result is, that no perceptible change in the level of the fluid in the capillary tube is produced by closing or opening the galvanic circuit. If the circuit be kept closed for a sufficient length of time, the level of the water in the capillary tube rises steadily as the bar expands under the action of the heat radiating to it from the coil, but, whether rising from this cause or stationary, the level of the fluid in the capillary tube is equally unaffected by the closing or opening of the galvanic circuit. The experiment, indeed, is one which affords most conclusive proof that the bar has undergone no change of volume on being magnetized or demagnetized, for if it were otherwise, the lengthening and shortening of which there is evidence in the former experiment, would cause the level of the water in the capillary tube to rise twenty degrees when the bar was magnetized by closing the galvanic circuit, and to fall as many degrees when the bar was demagnetized by opening the circuit.

Under the influence of magnetism, therefore, there are changes in a bar of iron which are in some degree pa-

rallel to the changes in muscle—changes of which the parallelism does not fail in that point which is so characteristic of muscle, namely, the *suddenness* with which the contracted and elongated states may alternate the one upon the other, for in the first experiment the bar is seen and heard and felt to jump suddenly from the elongated to the contracted state, and from the contracted back again to the elongated state, as the magnetism is withdrawn or communicated.

4. It is impossible to dogmatize upon the mode in which the will acts in voluntary muscular contraction; but this, at any rate, is plain, that there is nothing in the case to render it necessary to suppose that the will is, so to speak, infused into the contracting muscle, and that this most vital of all vital energies acts as a stimulus to a vital property of contractility inherent in muscle. This much is also plain—that there is nothing in the case to render it impossible that the will may act by bringing about a change in the nerve-current of the musculo-motor nerves, that this change may involve the development of induced currents in the muscles, and that these currents may be the agents through which the will works in producing voluntary muscular contraction. And certainly there is nothing in this view which in any sense compromises the dignity of the will as a vital power.

— And if all this be so—if these facts, which at first seemed to present difficulties, may be explained in this manner—what is the conclusion of the whole matter? Is it not that which was hinted at as probable at the commencement of the present chapter? Is it not this—that *elongation*, rather than contraction, is the chief peculiarity of muscle? Is it not this—that there is a state of polarity in incitable muscle during relaxation which produces relaxation, and that contraction is nothing more than the necessary result of the muscle

being liberated from this state, and left to the operation of the attractive force which is inherent in the physical constitution of the muscular molecules? Is it not this — that the transitory contractions, which are said to belong to that form of contractility which is called *irritability*, occur in transitory lulls in the polar action which antagonises contraction; and that the prolonged contraction of rigor mortis, which is referred to that form of contractility which is called *tonicity*, derives its characteristic persistency from the fact that the polar action which antagonises contraction is then at an end? Such, indeed, and no other, appear to be the necessary conclusions.

It is, no doubt, a difficult matter to abandon old views and to turn round so completely as to regard muscular contraction as a process which may be said to be realised most fully in rigor mortis; but this difficulty is worth mastering, for the theory proposed is one which applies to rigor mortis, as well as to ordinary muscular contraction, and which leads us a step nearer to the realisation of a common law for vital and physical phenomena.

For what is the case with respect to rigor mortis? The case is simply this,—that as long as there is any sign of muscular or nervous incitability, or any trace of nerve-current or muscular current, so long is there no rigor mortis. If these signs and traces die out speedily, as in persons in whom the vitality of the frame has been exhausted by long life, or consumed by chronic disease, such as consumption, the muscles become speedily rigid: if, on the other hand, these signs and traces are slow in dying out, as in persons who have been cut down suddenly in the full glow of health, the muscles are slow in becoming rigid. And as to the rest, the simple fact is this, that, once contracted, the muscles remain contracted until they begin to putrefy—an event which happens most speedily in the case where the muscles retain their physical integrity least perfectly.

The facts, indeed—which are utterly unintelligible upon the theory that contraction depends upon the stimulation of a vital property of contractility inherent in muscle—are precisely what they ought to be according to the premises; for, according to the premises, all that is necessary to the commencement of rigor mortis is the dying out of that action in muscle and nerve of which the electrical current is *one* of the signs; and all that is necessary to the continuance of rigor mortis is the absence of this action, and the physical integrity of the muscular structure. According to the premises, indeed, there is no difficulty in explaining the unexplained and apparently contradictory characteristics of this form of muscular contraction; and this being the case, it would seem that rigor mortis may be accepted as the type of muscular contraction in general, and as a crucial argument in favour of the theory which seems to apply to simple muscular motion.

It is evident, also, that this theory of muscular motion leads us a step nearer to the realization of that law which binds vital and physical phenomena together in one, inasmuch as it brings the phenomena of muscular motion within the category of electrical phenomena. In saying this, however, I do not intend to imply that electricity is more than a dim and one-sided aspect of this common and universal law. In a word, I believe that this law must be looked upon, not as reducing vital phenomena to physical, in the sense of degrading them, but as elevating physical to vital, or rather as elevating both vital and physical to a higher point still; for the law which comprehends vital and physical is more spiritual, more universal, than that which merely comprehends vital phenomena. Upon these matters, however, I have speculated elsewhere; and here I will content myself by saying, that what seems to me the strongest argument of all in favour of the theory of muscular motion enunciated in this

chapter, its very *experimentum crucis*, is to be found in the fact that it leads us a step nearer to the discovery of the common law to which I am alluding—the law to whose substantial existence the instincts and the discoveries of science alike bear testimony.

CHAPTER V.

ON THE APPLICABILITY OF THE FOREGOING THEORY TO RHYTHMICAL AS WELL AS TO SIMPLE MUSCULAR MOTION.

IN the preceding chapters, the object has been to discuss certain physiological questions so as to arrive with as little delay as possible at the pathological problems with which they have to do; and, in order to this, I did not stop to speculate upon many objects of interest which presented themselves in and by the way. For the same reason, I propose to consider the questions belonging to the present chapter in the same manner, concerning myself only with the most salient instances of rhythmical muscular movement—the beating of the heart, the writhing movement of the alimentary canal, the heaving and falling of the chest,—and passing by altogether those rhythmical movements in which, as in vibratile ciliæ and in the lateral leaflets of the *hedysarum gyrans*, muscle is not the motive power. In doing this, however, I omit nothing, which, so far as I know, in any degree invalidates the conclusions which may be drawn from the instances selected for consideration.

In dealing with these three problems, the plan which appears to be most satisfactory is to consider them, first, in relation to the operation of the blood, and, secondly, in relation to the operation of nervous influence; and this, therefore, is the plan which will be adopted in the following remarks.

I.

1. If any one will watch the movements of the heart, he may soon satisfy himself that the systole of the ventricles can have little to do with any stimulation on the part of the blood.

When the ventricles pass into the state of systole, the blood rushes through the coronary arteries into the coats of the heart, and the state of systole is instantly changed for that of diastole. Then comes a pause—a pause occupying the full half of the whole rhythmic period, and allowing time for the blood in the coronary vessels to have lost its arterial properties — and after this, the systole recurs. The facts, indeed, would seem to point to the red blood, not as producing the systole by acting the part of a stimulus to the irritability of the muscular fibre of the ventricle, but as producing the diastole by antagonizing the state of muscular contraction which constitutes the systole.

The behaviour of the heart in the two opposite states of plethora and anæmia would also seem to justify the same conclusion. In plethora the pulse is slow and full; in anæmia it is small and quick. In the one case, that is to say, the ventricle fills to distension with rich blood, and the systole is deferred; in the other case, the ventricle takes in but a small quantity of poor unstimulating blood, and is in haste to expel it. The facts, indeed, are precisely what they ought not to be if the blood stimulate the ventricle to contract, for in that case the pulse would be small and quick in plethora, and full and slow in anæmia: and the facts are precisely what they ought to be if the blood antagonize contraction, for in this case it is sufficiently intelligible that the ventricle should dilate more fully, and the dilatation continue for a longer time, when

the blood is rich and warm, as in plethora, than it can do when the blood is poor and watery, as in anæmia.

Remembering what has been said about the action of the blood in muscular motion, and about muscular motion generally, and reflecting upon the facts which have just been mentioned, it would seem, indeed, as if the natural way of interpreting the rhythm of the ventricles is by supposing that the systolic state of contraction is antagonized by the presence of red blood in the coronary vessels, and that this state occurs when the transformation of red into black blood has destroyed this antagonizing influence. Starting from the moment of systole, it would seem that the red blood, which is then injected through the coronary arteries into the ventricular walls, produces the diastole by antagonizing the state of contraction which constitutes the systole. Arrived at the diastole, it would seem that this state must continue until the red blood contained within the ventricular walls is transformed into black blood, and that then the diastole must give place to the systole, for the simple reason that the state of contraction is no longer antagonized by the red blood. Having returned to the moment of systole, it would seem further that provision is made for the immediate return of the diastole, for the systole which follows the diastole cannot take place without injecting into the coronary arteries a portion of the red blood with which the left ventricle is charged during the diastole. And thus it may be supposed that diastole must follow upon the heels of systole, and that diastole must in due time once more give place to systole, so long as the blood and the walls of the ventricles continue to react in this manner.

The differences in the rhythm of the auricles would also seem to furnish no objection to this view. The diastole of the auricles, which is virtually coincident with the diastole of the ventricles, is easily disposed of; for it may be sup-

posed that this state may be due, partly to the same cause as the diastole of the ventricles, namely, the rush of blood into the coronary vessels, and partly to the current of blood which is continually rushing in from the veins. The systole of the auricles, which is contemporaneous with the diastole of the ventricles, is less readily accounted for ; but, upon examination, there is reason to believe that this state may be partly, if not mainly, due to the falling in of the auricular walls upon the sudden sucking in of blood from the auricles into the ventricles at the ventricular diastole, and that in this way the chief difficulty may be done away with. There is reason for this opinion in the absence of valves at the mouths of the great veins opening into the auricles, and the reason is obvious. For if the auricular systole had to minister to the carrying on of the circulation in the same sense as the ventricular systole, is it not fair to assume that there must have been valves to prevent the reflux of the blood from the auricles into the great veins when the auricles contracted ?

And, certainly, there is nothing in the action of the blood upon the vessels generally which need lead to a different conclusion. If the blood produce contraction in the coats of the vessels by acting as a stimulus to the irritability of these coats, the only conclusion appears to be this, that it will not enter into the vessels until it has done all in its power to prevent its own movement ; but if the blood antagonize contraction, a very different conclusion is necessary. In this case, indeed, the blood will open, so to speak, a way for itself by dilating the vessels. In this case, the blood will also furnish other and important aid in carrying on the circulation ; for the vessels will be left at liberty to contract upon the blood contained in them, after this blood has given up the arterial properties by which they were dilated. In this case, indeed, we gain a glimpse into

the nature of "capillary force," and the mode in which this force may operate in producing "determination of blood" and inflammation. But these are questions which I cannot notice without digressing from the narrow path I have marked out for myself, and therefore I resist the temptation to stop, and hurry on.

Nor is the previous view respecting the operation of the blood in the rhythmical movements of the heart to be set aside by the fact that these movements continue during asphyxia, and after the heart has been cut out from the body.

In the case of asphyxia it may be enough to say that the blood is not all at once deprived of its oxygen, and that the blood entering into the coronary vessels may for some time be relatively arterial, when contrasted with the blood escaping from these vessels. It is very possible, also, that the heart may continue to beat after it has been cut out from the body, because the oxygen of the air takes upon itself the work which was previously discharged by the oxygen of the blood. There is no difficulty in supposing that the air may penetrate deeply into the substance of the cardiac muscle, partly through the open mouths of cut vessels, and partly through the pores and interspaces of the tissues themselves; that the state of relaxation may be determined by the oxygen of the air, in the same way as that in which it is determined by the oxygen of the blood; and that the state of contraction may follow when the oxygenating properties of the air are exhausted, just in the same way as that in which it followed when the oxygenating properties of the blood were exhausted. There is also no difficulty in supposing that the contraction may displace the used-up air, and restore the state of relaxation, by bringing the heart into relation with fresh air. And thus, starting from the same point, and returning to it again by the same path, relaxation may end in contraction, and con-

traction be followed by relaxation, so long as the cardiac tissues and the air are capable of reacting in this manner. All this is very possible ; all, this, moreover, is rendered in some degree probable by the fact that a heart ceases to beat when it is placed in a vacuum, or when it is immersed in nitrogen, hydrogen, or carbonic acid, and that it resumes its beatings when air is admitted into the vacuum or substituted for the gases which have just been mentioned. It is also an argument to the same effect, that a heart which has ceased to beat in common air will begin to beat again, and go on beating for some time, when removed from common air into oxygen gas.

2. It is also somewhat difficult to believe that the blood plays the part of a stimulus in producing the peristaltic movements of the intestines. For what is the fact? The fact is, as it is seen to be in one of M. Spiegelberg's experiments (p. 19),—that these movements are increased when the descending aorta is pressed upon so as to prevent the admission of blood into the intestinal vessels, and diminished when the pressure is removed and the blood allowed to return to the vessels. The fact is—that these movements are increased, though not in the same degree, when the vena cava or vena porta is pressed upon so as to keep the intestinal vessels full of venous blood, and diminished when this pressure is removed, and the vessels allowed at once to get rid of black blood and to receive red blood. This experiment, moreover, is in keeping with all the rest:—and thus it is not too much to say that the facts would appear to be altogether opposed to the idea which connects the peristaltic movements of the alimentary canal with any stimulation on the part of the blood.

3. Upon watching the movements of respiration, it would seem, at first sight, as if these movements were connected with changes in the air passing in and out of the air-passages, rather than with changes in the blood passing into

the pulmonary artery and through the lungs. The case, at first sight, would seem to be somewhat similar to that of a heart, or a fragment of a heart, when beating in the air after removal from the body. It would seem, indeed, as if the dilatation of the air-passages in inspiration is partly owing to the relaxation of the muscles of these passages under the action of the oxygenated air; it would seem as if the contraction of the air-passages in expiration is partly owing to the return of these muscles from the relaxed to the contracted state in consequence of the oxygen being used-up which had determined the state of relaxation. At the same time, it is evident that the chief cause of inspiration and expiration is to be found in certain changes in the form of the chest, which changes are brought about by alternate movements of contraction and relaxation in the diaphragm and other thoracic muscles—the inspiratory increase of capacity in the chest being chiefly due to the diaphragm and other thoracic muscles having entered into a state of contraction, the expiratory decrease of capacity being chiefly due to the passive return of the chest to its former dimensions upon the diaphragm and other thoracic muscles having passed out of the state of contraction into that of relaxation. It is evident, indeed, that the instrumentality of the nervous system is indispensable to the carrying on of the respiratory movements, for without this instrumentality these distant muscles could not be thrown into action or out of action; and, this being the case, it may be well to defer the consideration of these movements until they can be viewed in relation to the operation of the nervous system. It may be well to do this, also, because it would be not a little difficult to form any accurate conception of the operation of the blood in these movements apart from the operation of the nervous system.

II.

1. Mr. Paget* has connected the rhythmical movements of the heart with the action of certain nerves and nervous centres which have been detected by MM. Bidder and Rosenberger within the substance of the heart, and, as the link of connexion, he has used the fact that these movements are only manifested in those parts of the heart in which these nerves and nervous centres are to be met with in considerable number—parts which border closely upon the lines of junction between the auricles and the ventricles, and between the great veins and the auricles. In one of the experiments in which this important fact is made apparent, the heart of a tortoise, cut out from the body, is divided into two pieces, the one comprising the auricles and the base of the ventricles, the other being the remainder of the ventricles, and the result is that rhythmical movements are found to continue in the former piece, but not in the latter. In another experiment, the heart of a frog, from which all traces of the auricles have been removed, is set upright in a small pool of blood, and after this the upper border of the ventricle is slowly snipped away, bit by bit; and here the result is that the pulsations are reduced in frequency by every snip, and that they cease altogether after a zone of a certain depth—nearly the upper third of the ventricle—has been removed. In other experiments ligatures are tied tightly around the line of junction between the great veins and the auricles—lines rich in nerves and nervous centres—and, upon doing this, the action of the heart is found, first, to cease for a time, and, afterwards, to commence and continue in the ventricle alone. It was also noticed in these experiments (which were upon the hearts of tortoises and frogs), that there

* "On the Cause of the Rhythmic Motion of the Heart," 'Proc. of Royal Society,' 28th May, 1857.

was a rhythmical movement in the great veins behind the ligatures, and that the rhythm of this movement did not correspond with that of the movement going on in the ventricle. In other experiments, in which ligatures were placed around the great veins, at some distance from the auricles, the action of the heart went on with little or no change after the operation. It was also ascertained, on more than one occasion, that every fragment of the parts bordering upon the line of junction between the auricles and the ventricles had the power of contracting rhythmically, and that the parts which had not this power were capable, like ordinary muscle, of contracting vigorously whenever they were touched by a foreign body. In a word, the evidence is amply sufficient to show that rhythmical movements are only manifested in those parts of the heart in which the nerves of MM. Bidder* and Rosenberger† are to be met with in considerable number, and, in this way, to justify the conclusion to which Mr. Paget has arrived—that these movements are connected with the action of these nerves and nervous centres.

Evidence, moreover, is not wanting to show that the rhythmical movements of the heart are under the control of the great nervous centres. Thus, for example, the action of the heart may be immediately brought to a standstill by passing a series of induced currents of a certain strength through the medulla oblongata or pneumogastrics of a frog. While listening with his ear upon the chest of a dog, whose pneumogastrics were being galvanized by induced currents, Professor Claude Bernard ‡ had already ascertained that the sounds of the heart were not to be heard during the passage of the currents; but the fact of the action of the heart being arrested under these circumstances

* Müller's 'Archiv,' 1852, p. 163.

† 'Du centris motuum cordis.' 8vo, Dorpat, 1850.

‡ 'Thèse de M. le docteur Lafèvre,' Paris, 1848.

was first clearly enunciated by the brothers MM. Ernest and Henri Weber.* It is not right, however, to suppose that the action of the heart is always arrested under these circumstances. If the currents be of a certain strength, this, without doubt, is the result; but if the currents be less powerful, instead of arresting the action of the heart, they are found to have an opposite effect. In one of the experiments by which Mr. Joseph Lister,† of Edinburgh, has demonstrated this important fact, a portion of the left vagus of a rabbit is subjected, first of all, to the action of induced currents, which have been made doubly feeble by charging the galvanic cell with very weak acid, and by removing the core of iron wire from the heart of the coil. In the next place, the currents acting upon the nerve are increased in strength by pushing the core of iron wire home within the coil. In the third place (after the lapse of an interval of fifteen or twenty minutes), the currents acting upon the nerve are still further increased in strength, by adding a little more acid to the galvanic cell. The result, so far as the heart is concerned, is quickening of the rhythmical movements under the weak currents employed in the first instance, arrest of these movements under the stronger currents employed in the second instance, renewal of these movements under the still stronger currents employed in the third instance.

Viewing these facts with the intention of ascertaining their bearing upon the interpretation of nervous action in the rhythmical movement of the heart, the conclusion is not different from the one already arrived at, when speaking

* 'Handwörterbuch der Physiologie,' art. Muskelbewegung, vol. iii, p. 42, 1846.

† "Preliminary Inquiry into the Functions of the Visceral Nerves, with special reference to the so-called 'Inhibitory System,'" 'Proc. of Royal Society,' August 13th, 1858.

of the operation of the nervous system in ordinary muscular motion.

It is not necessary to suppose, with Mr. Paget, that the rhythm of the heart is due to "time-regulated discharges of nerve-force in certain of the ganglia in and near the substance of the heart, by which discharges the muscular walls are excited to contraction," and that these discharges are themselves due to the nutrition of the ganglia and contractile tissues "being, in certain periods, by nutritive changes of composition, raised, with regulated process, to a state of irritability of composition, in their decline from which they discharge nerve-force, or change their shape in contracting." On the contrary, it may be supposed—and the supposition arises necessarily out of the premises—that the intra-cardiac ganglia are affected by the blood in the same way as that in which the brain is affected in the experiments of Sir Astley Cooper, and MM. Tenner and Kussmaul (p. 28). In the case of the heart, indeed, nature may be said to be continually repeating these experiments on a small scale, the state of diastolic relaxation following the injection of blood into the coronary arteries at the ventricular systole being the counterpart of the state of general muscular relaxation which attends the unstopping of the cervical vessels; the state of systolic contraction which happens when the flow of blood into the coronary arteries is suspended by the passing of the ventricles into the state of diastole being the counterpart of the convulsive contraction of the whole muscular system, which attends the stopping of the cervical vessels. Or it may be supposed—and the supposition is perhaps more applicable to the case—that the intra-cardiac ganglia are affected by arterial and venous blood in the same way as that in which the brain is affected; that the state of systolic contraction is the counterpart, on a small scale, of the general convulsive contraction which happens when the blood supplied to the brain is de-

prived of its arterial character ; and that the state of diastolic relaxation is the corresponding counterpart of the state of general muscular relaxation which follows the re-admission of red blood to the brain. Viewed in relation to what has gone before, it would seem indeed as if the systolic state of the heart is produced, not by discharges of nerve-force from the cardiac ganglia to the cardiac muscles, but by the momentary interruption of the supply of nerve-force which proceeds musclewards so long as these ganglia are bathed with arterial blood or (if the heart is cut out and exposed to the action of the air) with oxygenated air.

In order to explain the stoppage of the action of the heart which takes place when induced currents of a certain strength are passed through the medulla oblongata or pneumogastrics of a frog, it is not necessary to assume, with M. Pflüger,* the existence of a special class of nerves, whose office is to diminish, arrest, or *inhibit* action — a Hemmungs-Nerven-system ; for the experiments of Mr. Lister, of which one has been cited already, afford most conclusive evidence that the same nerves may arrest or accelerate muscular contraction, according as they are acted upon by strong or feeble induced currents. “The phenomena observed in this and similar experiments appear to me,” says Mr. Paget,† “very similar to those of *shocks*. As a violent shock of any kind may exhaust the power, or suspend the action, of the brain or spinal cord, so may a shock by violence or galvanic force similarly affect the power of the rhythmic nervous centres for the heart. And the general explanation of all may be, that the nutrition of a nervous centre, and thereby the maintenance of its power, requires

* ‘Ueber das Hemmungs-Nerven-system für die peristaltischen Bewegungen der Gedärme,’ Berlin, 1856.

† Op. cit., p. 479.

rest, and that this rest cannot exist while nerves in relation with it are under irritation." And, certainly, this idea of shock would seem to meet the case more satisfactorily than the idea of Mr. Lister, which is, that the "inhibitory" influence which produces arrest of cardiac motion is the natural influence of the nerve in an exaggerated form. There is, indeed, no difficulty in supposing that the weakening of the nerve-current in the part where the medulla oblongata or pneumogastric nerve is subjected to the action of the induction coil, may necessitate a corresponding weakening of the nerve-current in the neighbouring nerves, and among others in the nerves connecting the cardiac ganglia with the pneumogastric; that the weakening of the nerve-current in these connecting nerves may necessitate the development of induced currents in the cardiac ganglia, and elsewhere in the neighbourhood; and that these induced currents may give such a mechanical shock to the cardiac ganglia as may derange the relations of their electro-motive molecules, and suspend their nerve-currents. As, in concussion of the brain, the action of this organ is suspended, and the muscles of the body are left in a relaxed condition, so in this case the action of the cardiac ganglia may be suspended, and the cardiac muscles left in a relaxed state from a similar cause, the mechanical commotion attending the passage of the induced currents having produced, so to speak, a state of concussion in the small brains of the heart. It may be supposed, also, that the effect of acting upon the medulla oblongata or pneumogastrics with an induction coil, of which the action is weaker than that which arrests the movements of the heart, will accelerate these movements; for if this weaker action fail to arrest, it will not fail to weaken the operation of the cardiac ganglia, and in that way to accelerate the cardiac movements. It will not fail to weaken the operation of the

cardiac ganglia, for it may be supposed that the induced currents developed in these ganglia, in consequence of the changes produced in the nerve-current of the cardiac nerves by the action of the induction coil upon the medulla oblongata or pneumogastric, have a direct relation in point of strength to the degree of this action, and that the operation of the cardiac ganglia is weakened in a similar direct relation to the strength of the induced currents acting upon them. It will not fail to accelerate the cardiac movements, for it has been seen that the tendency to muscular contraction becomes greater in proportion as the operation of the nervous centres upon the muscles related to them becomes diminished. Nay, there is no difficulty in understanding how it is that the action of an induction coil, which at first is more than strong enough to suspend the movements of the heart, may, after a time, cease to have this effect; for when this change is brought about, the incitability of the nerve or nervous centre is so impaired, that the weaker action of the coil fails to tell upon the movements of the heart, and the stronger action is only capable of producing the results which were at first produced by the weaker action.

It would seem, also, as if an additional argument in favour of the view which would connect the contracted state of the ventricle with the absence rather than with the presence of nervous influence, may be deduced from the effects of fear upon the action of the heart. In a state of fear the heart beats quickly, and yet little blood is propelled into the vessels. The beats are perhaps doubled, and yet the skin is cold and pale. Now, under ordinary circumstances, the double number of beats would make the skin red and hot, by propelling a double amount of blood into the vessels; and hence a fair inference from this anomalous state of a rapid pulse and a pale and cold skin would seem to be this—that the ventricular diastole is less

complete while the system is under the influence of fear, and that, on this account, less blood than usual is taken into and pumped out of the heart. In other words, the ventricle would seem to have contracted coincidentally with the withdrawal of nervous influence, for this influence may be supposed to be less freely generated while the system is depressed by fear.

Nor is there anything in what is known of the influence of the sympathetic nerves upon ordinary vessels which need contradict any of these conclusions respecting the operation of the nervous system in the movements of the heart.

Among the most remarkable of the many remarkable disclosures of modern physiological research are the results which follow certain experiments upon the cervical portion of the sympathetic nerve. After removing the superior cervical ganglion, for example, or after dividing the cervical filament of the sympathetic nerve in a rabbit, there is a rapid and unmistakeable increase in the warmth, and vascularity, and sensibility, and muscular incitability of the corresponding side of the head and face, the temperature augmenting by several degrees, the conjunctiva and lining membrane of the nostril and ear becoming bloodshot, the pulse beating with greater fulness and force than its fellow-pulse on the other side, the skin being positively tender in many places, the eye and ear seeming to be more sensitive to light and sound, the muscles responding more readily to incitation of various kinds. The increase of temperature, moreover, is a change which is found to extend deep below the surface; for on burying a thermometer within the substance of the brain, or on immersing the bulb of the instrument in the stream of blood which passes downward in the internal jugular vein, the mercury is seen to rise to a higher point on the side corresponding to the operation than that to which it rises on the other side. These

several strange effects continue with little or no change for weeks, perhaps for months; but—what is almost still more strange—they may at any time be made to disappear for the time, by exposing the cephalic end of the divided nerve to the action of induced currents, or by inciting it in any other manner. There are also several other curious phenomena which are brought about by excising the superior cervical ganglion, or by dividing the cervical filament of the sympathetic, and which pass off in the same manner upon inciting the cephalic extremity of the divided nerve; such, for example, are contraction of the pupil, constriction of the opening of the eyelid and nostril, puckering of the angle of the mouth, and partial pricking of the ear. It must be noticed, also, as a fact of especial interest, that the incitability of the muscles continues for a longer time after death, and that rigor mortis comes on later and lasts longer upon the side of the head and face corresponding to that upon which the nerve has been divided.

The discovery of these remarkable facts is due to the labours of more than one person; but Professor Claude Bernard* and Dr. Brown-Séquard† have between them the lion's share of the honour, the former for being the first to point out the greater number of the effects attending the division of the nerve, the latter for being the first to indicate the greater number of the effects which follow the galvanization of the cephalic end of the divided nerve, and which happen after death. It is, however, only just to say,

* 'Comptes Rendus de la Soc. de Biologie,' Dec., 1851; 'Gaz. Méd. de Paris,' 1852, p. 72; 'Comptes Rendus de l'Académie des Sciences,' March 29th, 1852; 'Leçons sur la physiologie et la pathologie du système nerveux,' 8vo, Paris, vol. ii, Leçons 15 and 16, 1858.

† 'Philadelphia Medical Examiner,' August, 1852; 'Experimental Researches applied to Physiology and Pathology,' New York, 1853; 'Lancet,' October 30th, 1858.

that Parfour du Petit* had noticed several of the effects attending the division of the cervical sympathetic a century ago or more, and that the two physiologists, whose names have been mentioned in the last sentence, may share the honour belonging to discovery in more than one instance, inasmuch as the discovery, and its enunciation by each, took place almost simultaneously.

Another fact, which must be mentioned here, as tending not a little to illustrate the action of the nervous system in the movements of the coats of vessels, is brought to light by M. Claude Bernard in his investigations into the changes of colour in venous blood †—the changes in question being exhibited in the vein proceeding from the submaxillary gland of a dog. This gland, it must be observed, is supplied with nerves from two distinct sources. It is supplied by a twig from the lingual nerve; it is supplied by filaments from the grand sympathetic (chiefly from the superior cervical ganglion), which filaments accompany the arteries of the gland. These nerves may easily be distinguished; they may be acted upon separately, without difficulty; and the results of this separate action are strangely dissimilar. The twig from the lingual remaining intact, and the filaments of the sympathetic being divided, the venous blood proceeding from the gland, not only loses its dark colour and becomes permanently red, but it escapes from a wound in the vein as it would from a wound in an artery, that is, in jets. On the other hand, the filaments of the sympathetic being let alone, and the twig of the lingual divided, the blood in the vein proceeding from the gland remains black, and the shrunken appearance of the vessel shows that the venous stream is diminished in quantity. Corresponding changes are also produced by inciting these nerves without previously dividing them,

* 'Mémoires de l'Académie des Sciences,' 1727.

† 'Journal de la Physiologie,' No. 4, 1858.

or by inciting their peripheral portion after having divided them; the blood in the vein proceeding from the gland acquiring arterial colour and motion, if the twig of the lingual be acted upon; the blood in the vein remaining black, and running in a diminished stream, if the filaments of the sympathetic be the scene of the incitation.

It must be remembered, also, as a fact which may have some bearing upon the successful prosecution of the present inquiry, that the removal of the thoracic ganglia and solar plexus is apt to produce a state of vascular congestion in the neighbourhood, which easily passes into violent inflammation; for it is possible that this change may be less a consequence of the severity of the operation, than of that state of vascular turgescence which the two previous experiments have shown to follow the removal of the influence of the sympathetic nerves from the vessels.

Here, then, are certain facts which seem to afford some insight into the operation of vaso-motor nerves in the movements of vessels, and which must on that account be of no small use in illustrating the operation of these nerves in the rhythmical movements of the heart. And what do they show? They show, as it seems, that the vessels dilate when they are deprived of the influence of the vaso-motor nerves, and that they contract when this influence is duly supplied, or when the due supply is imitated by incitation. They show that the vessels dilate when they are deprived of the influence of *the vaso-motor nerves, not only because the arterial colour and motion of the blood in the vein proceeding from the submaxillary gland, after dividing the filaments of the sympathetic, would seem to imply a dilatation of the vessels by which the blood can get from the artery into the vein without losing its proper arterial colour and motion, but also

because the increased warmth, and vascularity, and sensibility, and muscular incitability of the corresponding side of the face and head, which follow the removal of the superior cervical ganglion, or the division of the cervical filament of the sympathetic, can only be explained on the supposition that dilated vessels allow the parts to be bathed in a more plentiful supply of blood. They show, further, that the vessels contract when the influence of the vaso-motor nerves is supplied in due course, or when this influence is called into action by incitation; for all the phenomena which are noticed under these circumstances are the direct opposites of those which are noticed when the influence of the vaso-motor nerves is withheld from the vessels. It would seem, at first sight, as if the influence of the vaso-motor nerves is one which determined a state of contraction in the coats of the vessels, by rousing into action the contractility of these coats, and that the vessels became paralysed and dilated when this influence was withheld; but this is not the only way in which the facts may be interpreted. On the contrary, it is a fair inference from the premises, that the reaction between the blood and the vessels may necessitate certain changes in the nerve-current of the vaso-motor nerves, that these changes in the nerve-current may give rise to the development of induced currents along the vaso-motor nervous arcs, and that these induced currents, thus developed among the muscular fibres of the vascular coats, may bring about contraction in the way which has been explained on more than one occasion previously. And it is also a fair inference from the same premises, that the muscular elements of the vascular coats will be relaxed by the operation of their own muscular currents, when these muscular elements are no longer subjected to the influence of their vaso-motor nerves.

How to explain the operation of the twig of the lingual

nerve upon the colour of the blood contained in the vein proceeding from the submaxillary gland is another and more difficult matter. At first sight it seems, as it seemed to M. Claude Bernard, that the influence of the nerve is to produce active *dilatation* of the vessel; at a second glance it seems, as it also seemed to the same distinguished physiologist, that the twig of the lingual acts upon the coats of the vessels through the instrumentality of the filaments of the sympathetic, the action being one which paralyses these latter filaments. In accepting this view, however, it is not necessary to seek for any new principle of interpretation to explain the result, for this is already done if it be supposed that the treatment to which the twig of the lingual is subjected produces some movement in the nerve-current of the nerve, that this movement of the nerve-current necessitates the development of induced currents among the filaments of the sympathetic which lie in the neighbourhood of the lingual, and that these induced currents suspend the polar action of these filaments by causing, as it were, a state of concussion—a change which will leave the vessel in a relaxed state; for, absolved from the control of the nerves, the muscular elements of the vascular coats will be kept in a relaxed state by their own muscular currents. It would seem, indeed, as if the twig of the lingual suspended contraction and produced dilatation of the vessel in the same way as that in which the vagus has been seen to banish the state of systolic contraction and leave the heart in the state of diastolic dilatation, or in which, as will be seen in the next section, the spinal cord or the grand sympathetic may banish the state of contraction and leave the intestine in the state of dilatation. The fact, moreover, that the blood in the veins proceeding from the kidney is red while the urinary secretion is in progress may also be of use in affording some insight into the cause of the red

colour of the blood in the vein proceeding from the submaxillary gland when the twig of the lingual nerve is incited, and this, it may be, not only because the submaxillary secretion is in progress when the blood in the vein proceeding from the submaxillary gland is red, but also because the distribution of the twig is to the ducts rather than to the blood-vessels of the gland.

And thus the movements of ordinary vessels under the operation of the nervous system cannot be looked upon as necessitating a different conclusion to that which was arrived at when speaking of the rhythmical movements of the heart in relation to nervous influence.

2. The part which is played by the nervous system in the peristaltic movements of the alimentary canal appears to be one and the same with that which is played by this system in the rhythmical movements of the heart.

As in the case of the heart the beatings are suspended by passing induced currents of a certain strength through the medulla oblongata or pneumogastric nerve, so in the case of the alimentary canal, M. Pflüger* has shown that the peristaltic movements may be arrested by passing induced currents of a certain strength through the spinal cord or the grand sympathetic. The two cases also correspond in this—that the muscular movement is suspended in the state of *relaxation*, the heart pausing in the state of diastole, the intestine remaining in or passing into the state which is the opposite of contraction. Nor does the correspondence fail when the experiment is made with feebler induced currents, for in the alimentary canal, not less than in the heart, Mr. Lister has shown† that the effect of these feebler currents is the very opposite of that which is produced by the stronger currents, namely, increased movement, not arrested movement. In one of

* 'Ueber das Hemmungs Nerven-system,' &c.

† 'Proc. of Royal Society,' August 13th, 1858.

the experiments by which this important fact is demonstrated, the intestines of a rabbit are brought partially into view by removing the skin and the subjacent muscles from a portion of the abdomen. After this, the poles of an induction coil are applied to the spinous processes, so as to include between them the dorsal portion of the spinal cord. The experiment itself is divided into three stages. In the first and second stages the inducing current is derived from a galvanic cell which is charged with a very small amount of acid, and the only difference between the two stages is this—that the core of iron wire is within the helix in the first, and out of the helix in the second. In both these stages, that is to say, the induced currents passing along the spinal cord are comparatively feeble; but in consequence of the iron core being within the coil in the first, and out of the coil in the second, they are considerably stronger in the first than in the second. In the third stage (the two previous stages have been prolonged for a period of twenty or thirty minutes before this stage is commenced) the induced currents passing along the spinal cord are increased in strength by again pushing the iron core home within the helix, and by charging the galvanic cell with an additional quantity of acid. This is the experiment: the result is, that the peristaltic movements are arrested in the first, and quickened in the second stage—quickened, that is to say, in the stage in which the currents passing along the spine are feeblest. In the third stage the result is almost negative, for the circuit of the induction coil may now be opened or closed without producing any very marked or certain impression upon the rhythm of the peristaltic movements. In the first stage the action of the induced currents upon the muscles of animal life is in marked contrast to the action of these currents upon the intestinal muscles, the former muscles being convulsed when the latter muscles were quiescent,

and *vice versâ*. In the second and third stages the action of the currents upon the muscles of animal life is not noted. It is noted, however, as occurring on more than one occasion during the progress of the experiment, that a violent struggle on the part of the animal, when the intestines were in free movement, was followed for some moments by absolute and universal cessation of this movement. In an experiment upon another rabbit, in which, after M. Pflüger's original plan, the intestines were allowed to protrude through an opening in the abdominal parietes, the same results were observable, with this addition, that the protruded intestines were seen to be capable of exhibiting strong local contraction under the prick of a knife, while lying relaxed and motionless under the action of the induced currents upon the spinal cord—a fact noticed by M. Pflüger, and showing that the "inhibitory" influence of the induced currents is exercised upon the nerves, and not upon the muscular fibres of the intestines.

It may be supposed, also, that the peristaltic movements of the alimentary canal are connected with a special system of nerves and nervous centres; for after what has been said of the action of the nerves and nervous centres of MM. Bidder and Rosenberger in the rhythmical movements of the heart, it is to be expected that the rich ganglionic structures lately detected by M. Meissner, of Bâle,* in the submucous tissue of the intestine will have to discharge a similar office in relation to the peristaltic movements of this viscus.

In so far as concerns their relation to the nervous system, therefore, there is nothing in the peristaltic movements of the intestines which does not fully harmonise with what has been said concerning the relation of the rhythmical movements of the heart to the action of the

* Henle and Pfeufer's *Zeitschrift für Rationelle Medicin*. Second series, vol. viii, p. 364, 1857.

nervous system—nothing which may not readily be explained in the same manner; and this being the case, the subject of the present section may be dismissed without further comment.

3. The connexion of the rhythmical movements of the chest with certain operations in the nervous system, in which the medulla oblongata is the great centre, and the pneumogastric and phrenic nerves respectively the principal afferent and efferent nerves, is a fact which cannot be questioned. The dependence of these movements upon the respiratory interchanges which take place between the air and the blood within the lungs is equally a matter of certainty. But exception may readily be taken to the current notion that the inspiratory contractions of the diaphragm and other thoracic muscles, by which the capacity of the chest is increased, and air drawn into the air-passages, are owing to the carbonic acid in the venous blood having acted the part of a stimulus to the pulmonary filaments of the pneumogastric nerves. Exception may be taken to this notion, not only because it is contrary to the whole tenor of the evidence advanced hitherto, but also because it fails to give any insight into the cause of the rhythm of the respiratory movements. According to this view, indeed, it seems as if the inspiratory contractions ought to occur whenever venous blood is pumped through the pulmonary artery into the lungs—as if, that is to say, the rhythm of the respiratory movements ought to correspond with the rhythm of the cardiac movements. But if this view be unsatisfactory, what other view must be substituted in its place? Is it true, as was hinted in a former section of the present chapter, that the rhythm of the respiratory movements is related to the air in the air-passages rather than to the blood in the pulmonary veins? These are questions which arise naturally out of the premises, and which require, as it seems, affirmative answers.

For what is the case according to this view? The case in *inspiration* is simply this—that the oxygen of the air at the openings of the air-passages acts upon the pneumogastric and other afferent nerves belonging to these passages; that an impression or sensation in these nerves is the result of this action; that this impression or sensation implies an equivalent movement in the nerve-current along the whole nervous arcs, of which the pneumogastric and other afferent nerves are portions; that this movement in the nerve-current involves the development of induced currents in the respiratory muscles, to which the phrenic and other efferent nerves of the respiratory nervous arcs are distributed; that these induced currents produce contraction by suspending the muscular current which keeps these muscles in the state of relaxation; and that this contraction of the respiratory muscles produces that expansion of the chest which is the grand agent in filling the air-passages with air. The case in *expiration* is simply this—that in due time the respiratory muscles must relax, and allow that falling in of the chest which is the grand agent in expelling the used-up air from the air-passages, for the simple reason that in due time the air must have given up the oxygen which was the agent in producing and keeping up that impression or sensation in the pneumogastric and other respiratory afferent nerves which led to the inspiratory contractions. And in favour of this view it may be said that the branches of the pneumogastric which receive the impressions or sensations which lead to the inspiratory contractions are distributed to the lining membrane of the air-passages, where they come into relation to the air breathed, rather than to the coats of the pulmonary capillaries, where they would come into relation with the venous blood to be breathed upon by this air—an arrangement which is the very opposite of that which would be likely to exist if the pneumogastric

had to take direct and immediate cognizance of the presence of carbonic acid in the venous blood. Nor is it to be overlooked that, upon this view, the air-passages themselves may co-operate with the chest in producing the respiratory movements; for after what has been said respecting the *direct* effect of arterial or oxygenated blood in muscular motion, it is to be expected that the *direct* action of the oxygen of the air will facilitate the entrance of the air in inspiration, by producing relaxation of the muscular fibres of these air-passages, and that the cessation of this relaxing action when the oxygen of the air inspired is exhausted will facilitate expiration by allowing the muscular fibres of the air-passages to return to the contracted state.

In this way, then, an insight may be obtained into the secret of the rhythm in the three instances of rhythmical movement which have been under consideration—the beating of the heart, the writhing of the alimentary canal, the heaving and falling of the chest; and as this way is that which opens out naturally from the premises, this fact must be taken as an additional argument in favour of the correctness of these premises. And thus the conclusion of the whole matter is this—that the theory of muscular motion which has been seen to be applicable to simple muscular motion is also applicable to rhythmical muscular motion.

PART II.

PATHOLOGICAL DEDUCTIONS RESPECTING EPILEPTIC
AND OTHER CONVULSIVE AFFECTIONS OF
THE NERVOUS SYSTEM.

PART VI

THE HISTORY OF THE
CITY OF BOSTON
FROM 1630 TO 1800

PATHOLOGICAL DEDUCTIONS.

EPILEPSY is at once the great type of convulsive disorders, and the key to their interpretation. Epilepsy, however, is a name which indicates much less now than it did formerly. Thus, it does not indicate the epileptiform convulsion which is connected with certain positive diseases of the brain, with fever, with certain suppressed excretions, with "irritation" in the gums and elsewhere, or with the moribund state; and it is difficult to say what it does indicate, for as our diagnosis gains in exactness, epilepsy changes more and more from a special malady into a mere symptom or group of symptoms. At the same time, it is, and, in all probability, it always will be, convenient to take an ideal type of epilepsy and regard it as a special malady, for there are numberless cases in which, in their earlier stages at least, it is very difficult, if not impossible, to recognise the disease of which the convulsion is merely a symptom.

Passing from this ideal form of epilepsy to the consideration of the actual disorders in which muscular contraction is in excess, it is found that these disorders may be conveniently divided into three categories, of which the distinctive signs are—tremor, convulsion, and spasm;

but it must not be forgotten that such division is purely arbitrary, and that spasm, convulsion, and tremor, are continually occurring in the same case, and at the same time.

The first category, in which the muscular disturbance takes the form of tremor, is that which includes the tremors of delicate and aged persons, of paralysis agitans, of delirium tremens, the rigors and subsultus of fevers, and the shakings of slow mercurial poisoning.

The second category, in which convulsion is the distinctive feature of the muscular disturbance, may be divided into two sections by the absence or presence of consciousness during the convulsions. Where the consciousness is present the convulsion may be called *simple*; where the consciousness is absent, it is *epileptiform*. Simple convulsion is that which is met with in the state called hysteria, in chorea, and in those strange affections which take an intermediate position between the two, as the dance of St. Vitus and St. John, tarantism, and other affections of the kind. Epileptiform convulsion includes the convulsions connected with certain diseases of the brain — chronic softening, chronic meningitis, tumour, induration, hypertrophy, atrophy, congestion, apoplexy, inflammation, with fever, with certain suppressed excretions, with “irritation” in the gums and elsewhere, and with the moribund state.

The third category, in which prolonged muscular contraction or spasm is the distinctive symptom, includes catalepsy, tetanus, cholera, hydrophobia, ergotism, the rigidity of cerebral paralysis, the spasm connected with certain diseases of the spinal cord, and some other spasms of a minor character.

Ordinary epilepsy, affections marked by tremor, simple convulsion, epileptiform convulsion, and affections marked

by spasm, are, therefore, the five special subjects which have now to be considered, and which will be considered most conveniently, perhaps, by arranging them in five separate chapters; thus—

- Chapter I. On Ordinary Epilepsy.
- Chapter II. On the Convulsive Affections which are characterised by Tremor.
- Chapter III. On the Convulsive Affections which are characterised by Simple Convulsion.
- Chapter IV. On the Convulsive Affections which are characterised by Epileptiform Convulsion.
- Chapter V. On the Convulsive Affections which are characterised by Spasm.

CHAPTER I.

ON ORDINARY EPILEPSY.

IN the present chapter, I propose to speak in succession—of the state between the paroxysms, of the paroxysm itself, of the appearances after death, of the pathology, and of the treatment. I shall endeavour to arrive at a safe conclusion respecting the pathology, by taking into consideration, first, the condition of the circulation and respiration, and, secondly, the condition of the nervous system. I do not propose to say anything about diagnosis, for any remarks upon this subject may be conveniently deferred to the chapters which treat of the different forms of simple and epileptic convulsion; and any reference which I may have to make to the causes of the disorder, and to the chances of recovery, will find a place without difficulty in one or other of the previous sections.

1. *Of the state between the paroxysms in ordinary epilepsy.*

An epileptic will often say—"except for these fits, I am quite well;" and in slight cases he may have a fair right to say so; but in cases of ordinary severity there are always certain signs which are altogether incompatible with true health and strength.

In very many instances there is a want of fire in the countenance, and a dilated and sluggish state of the pupil, which seem to point to the brain as lacking in energy; and, in keeping with these signs, it is found on inquiry that the memory is more or less treacherous, the under-

standing more or less vacant and listless, the temper more or less uncontrollable. It is, no doubt, easy enough to meet with epileptics, who, without any want of candour on their part, will deny the existence of any flaw in their mental faculties, and who have all but an absolute right to do so; but, in cases of ordinary severity, I do not remember a single instance in which this denial was borne out by the testimony of their friends,—and the testimony of friends must be of far greater weight in a matter of this kind than the testimony of the patient himself.

In very many instances, the habitual condition is one of fidgetiness and shakiness; and, in addition to this, there may be a marked disposition to actual trembling and cramp. Thus, in upwards of seventy cases which fell under the notice of my friend, Dr. Reynolds,* trembling or cramp occurred at one time or other, and in one form or other, in more than half of the whole number.

In very many instances, if not in all, the pulse is weak and slow, the hands and feet are cool or cold, and a feeling of chilliness is almost habitual. Indeed, so far as my own experience goes, the circulation is always wanting in true power. For a short time after a fit there may be, it is true, some febrile reaction; but if there be it is soon over, and the state which follows is the very opposite of fever. I know, indeed, of cases presenting satisfactory evidences of vascular activity, in which apoplexy and paralysis were the chief dangers to be apprehended, and in which epileptiform convulsions might attend upon the apoplectic or paralytic state; but these convulsions, as will be seen presently, are not to be confounded with those of ordinary epilepsy, without confounding matters practical as well as theoretical, which ought to be carefully kept apart.

In very many instances, moreover, the respiration may

* 'Lancet,' 4th and 11th August, 1855.

often be shallow and retarded, and occasional sighings may tend to corroborate this fact, by showing that the deficiencies of the common breathings have to be made up by some breaths which are more deeply drawn than usual.

In many confirmed and aggravated cases, a short examination will show that a terrible blight has fallen upon all the faculties which distinguish man from the mere animal. In these cases, indeed, a single glance at the countenance will often serve, at once to detect this blight, and to connect it with epilepsy; for now the fire which ought to illumine the features is dying out or dead, and the minute specks of blood which are effused into the skin of various parts of the face and neck, but particularly into the skin of the eyelids and temples, is a sure sign that the face has been "black and full of blood" in some recent paroxysm of epileptic convulsion. In the health of the mere body there may be but little wrong—nothing beyond a somewhat feeble circulation, and a somewhat insufficient respiration; in the health of the mind the case is altogether different; and therefore it is to be supposed that the obscure traces of mental imperfection which are present in cases where the characteristics of the disease are not fully marked, are in reality not accidental, but essential, and that no account of the interparoxysmal state in these cases would be complete, which did not include them.

In all cases, as might be expected, the evidences of mental imperfection are most apparent after a fit. At this time, indeed, the faculties of the mind may be so blunted that the features of the epileptic may become blended with those of the demented person, or symptoms of intellectual or moral aberration may show themselves, and the epileptic may for the time be transformed into the lunatic. The fits, also, may recur so frequently, that the mind may never have the chance of clearing up in the interval, and in this way the distinctive characters of the

convulsive malady may become confounded with those of dementia and insanity. Not unfrequently, also, there is the very gravest degree of mental infirmity from the very first, and instead of only tending to dementia, the history of the epileptic may begin in sheer idiotcy. Indeed, it cannot be looked upon as a mere accident, that idiotcy and epilepsy should so often go together, and that the head of the epileptic should be so frequently wanting in proper size and proportions, as to suggest to the least imaginative observer its suspicious kinship to the head of the idiot.

2. *Of the paroxysm of ordinary epilepsy.*

The signs of the approaching paroxysm are very various and very variable in the same person. The patient himself will generally say, and very truly, that the fit takes him by surprise, and, unfortunately, the signs of danger are, for the most part, vague, and little likely to arrest the attention of any one.

As the time of the fit draws nigh, the mind may become more vacant and listless, and the temper and feelings more moody and uncontrollable; or the increased mental negativeness may be accompanied by drowsiness; or the nights may be restless, and the sleep disturbed in various ways—by distressing and terrifying dreams, by snoring and snorting, by grinding the teeth, and so on.

In some instances there may be giddiness or headache, —but headache, I am disposed to think, will occur far oftener after the attack, and as a consequence of the attack, than as a warning of danger.

Occasionally, the pupils may be more dilated and sluggish than usual, or one pupil may be more dilated and sluggish than the other, or the eyes, one or both, may be rotated in a peculiar manner.

Occasionally, there may be certain vague and undefinable

sensations or movements, very varying in character, but all comprehended under the term *aura*—sensations of pain, numbness or tingling, a feeling as of a current of cold vapour, movements of a shuddering or spasmodic character, beginning in the pit of the stomach, or in a more distant part, as in a hand or foot, and travelling towards the head. There may be symptoms, that is to say, which, as Dr. Watson thinks, are in some degree analogous to the feeling of *globus* in hysteria, or to the numb and tingling sensations which are the frequent precursors of paralysis and apoplexy.

More rarely still, the fit may be ushered in by certain special premonitions. I remember one patient, for example, whose fit was invariably preceded by an intense feeling of hunger. I remember another, who was warned by the vision of a little blue imp, and who described very vividly the way in which this creature of his imagination grinned and mocked at him as he lost his consciousness. I remember a third, who knew that he was about to fall by hearing a sound as of a guitar roughly grated close to his ear.

Not unfrequently there may be disagreeable feelings of tightness about the throat, with cramps and tinglings in the limbs and elsewhere. Less frequently there may be shudderings of a very uncomfortable and violent character, sometimes affecting the whole body, sometimes confined to one of the limbs, and most generally there will be increased shakiness and fidgetiness, if shakiness and fidgetiness be characters of the interparoxysmal state.

As a general, if not as an universal rule, the fit would seem to be preceded by distinct failure in the power of the circulation, the pulse becoming more feeble and slow, the extremities more wanting in warmth, and the patient often volunteering the complaint that nothing will warm him or keep him warm. At any rate, I have met with no exception to this rule. I think, moreover, that I know of not

a few instances in which the improvement in the circulation which has been brought about by a glass of wine, or by some stimulating antispasmodic draught, has had the effect of warding off the fit for the time.

As a general rule, too, I believe that the respiration will be more insufficient than usual, and the breathings most frequently prolonged into sighs, when the time for the fit is close at hand. At any rate, I have met with many instances in which this was undoubtedly the case; and I have notes of four cases in which, when the fits happened during sleep, the movements of the chest became so imperceptible for some moments before the fit, as to suggest the idea of death. One of these cases was lately under the joint care of a physician in the country and myself, and we can both testify to the fact from personal observation.

Last of all, there is a sign which is very difficult to catch, and this is the death-like pallor which overspreads the countenance immediately before the fall. M. Delasiauve* was the first to notice this phenomenon; M. Trousseau insists upon it as a distinctive mark between true epilepsy and feigned epilepsy; and, since my attention was directed to it by M. Trousseau, I have seen it in all the instances (now amounting to a considerable number) in which I have seen the fit from the very beginning. "Il est une signe," says M. Trousseau "qui se produit du moment de la chute, et qui n'est imitable pour personne; c'est la pâleur très prononcée, cadavérique, qui couvre pour un instant la face l'épileptique. Nous ne le voyons pas, parceque nous arrivons toujours trop tard alors que la face est déjà d'une rouge très prononcé." †

— In the severest and most characteristic form of the

* 'Traité de l'Epilepsie,' Svo, Paris, 1854.

† 'L'Union Médicale,' April 28th, 1855.

paroxysm, the patient utters a peculiar choking noise, or a sudden and startling cry or scream, and at once falls down convulsed and insensible. The convulsions are usually more marked on one side of the body than the other. They drag the mouth towards the side which is most affected, and twist the face in the opposite direction until the chin may be behind the shoulder. They push forward the tongue, and crush it between the teeth. They clasp the thumb upon the palm, and hold it down with giant-like force. They seize the walls of the chest and abdomen, and prevent the possibility of breathing. They stiffen the limbs until it is almost more easy to break the bones than to bend the joints. In some instances, they take hold of the bladder, the bowel, or the seminal vesicles, and expel the contents; in others, they bite off the end of the tongue, or break the teeth, or dislocate a limb. At first, it seems as if they would never relax; afterwards they are separated by intervals, which grow wider and wider as the paroxysm draws to an end. The convulsions, that is to say, are tetanic at first—clonic afterwards.

At the instant of the fall, a corpse-like paleness overspreads the countenance; a few instants later, and the livid, black, and bloated head and neck, and the hissing, gurgling, choking sounds proceeding from the throat, suggest the idea of a person struggling under the bowstring of some invisible executioner. At times, however, the signs of suffocation are absent, and the ghastly pallor of the beginning remains throughout.

When the fit is at its height, a quantity of frothy saliva is usually blown or puffed from the mouth, and this is not unfrequently reddened with the blood which has escaped from a bitten tongue or cheek.

If the eyelids are open, the eye is seen to be projected and distorted, with the pupil dilated to the utmost, and absolutely insensible to light. As a rule, however, the

eyelids would seem to be half-closed; and well it is that they are so, for it requires some nerve to meet the hideous stare of the epileptic eye.

All this while, it is usual for the hands and feet to be cool, and bedewed with clammy perspiration. Except the head and neck, indeed, the whole body is cooler than natural, and any little additional warmth of the head and neck would seem to be simply due to the fact that the vessels in these parts are more distended with venous blood.

The other and less obvious features of the paroxysm are in keeping with these.

At first, it may be difficult, perhaps impossible, to feel the pulse, and the heart acts very feebly; but if the fingers of one hand be kept upon the wrist, and the other hand be placed upon the bosom, it is found that the artery soon pulsates with a force and fulness which is never met with in the intervals between the fits, and that the heart beats more and more tumultuously and violently as the pulse rises. In some instances, however, the pulse may remain almost silent, and the heart may beat with extreme feebleness from the beginning to the end.

From the first all consciousness is happily suspended—this is our only consolation in so sad a spectacle—and the most powerful stimulants fail to evoke any sign of action in the dormant mind. The water which may be poured upon the face (with few exceptions) causes no blinking in the half-open or staring eye; the fire upon which the patient may have fallen may char the flesh without producing a single pang.

After continuing for two or three minutes, which seem drawn out to hours, the convulsions cease, and the patient is left with all his muscles unstrung, like a person dead-drunk, or struck down by apoplexy. The lungs, no longer restrained by the suffocative spasm of the earlier part of

the fit, resume their play with deep inspirations, and then act with loud and stertorous breathings; and as the respiration rights itself, the veins of the head and neck become unloaded, the natural colour returns to the surface, and presently the patient wakes to an obscured and troubled consciousness. "Je suis brise," Calmeil tells us, were very often the first words of the returning epileptic at the Salpêtrière or Charenton. The time during which the patient lies after a fit before awaking is very variable, but (except in a first attack) it is rarely more than half an hour, and it may not be more than two or three minutes.

This is the usual, but by no means the invariable, course of the fit. Often, indeed, the attempts at rallying may be very imperfect, and fit after fit may recur for a long period without any interval of waking; and occasionally all rallying may be prevented by death.

After waking, there are generally some symptoms of reaction in the circulation, but in simple epilepsy these are never marked. They may be enough to give a dull flush to the cheek and a little fulness to the pulse for a short time after the patient wakes; but, as a rule, they cease when the coma ceases, and coma is never much prolonged in simple epilepsy. Usually the patient is head-achy and exhausted, listless and stunned, moody and irritable, until a night's rest has enabled him to recover the balance of his shaken nervous system. The jaded countenance also tells plainly of the past struggle, even though it present none of those numerous and minute dots of ecchymosis about the eyelids and upon the forehead which are such unequivocal signs of a severe attack of epilepsy.

As time goes on, the mental faculties recover more and more imperfectly, and more and more tardily, and at last their habitual state may be one of pitiful fatuity from

which no single ray of the Divine principle beams forth. Or the moodiness and irritability which often follow the fit may become more and more marked, until at last they merge into attacks of downright mania. Or symptoms of paralysis may make their appearance. Or death may happen in a fit, or shortly afterwards. The natural tendency of epilepsy is assuredly towards dementia; and dementia is the frequent doom of the epileptic, if his disorder be unchecked and life prolonged sufficiently; but at the same time it is possible for an epileptic to live many years, and to have many fits, without losing the powers which are necessary to render him an agreeable and serviceable member of society. When death happens, it appears to be, most generally, from exhaustion in the period of prostration immediately following the paroxysm.

But the symptoms of epilepsy are not always so startling as have been represented, and in some instances they may be so softened down as to be recognised with difficulty.

In the slightest form of the malady, the patient pauses suddenly in the midst of anything he may happen to be doing or saying at the time, his countenance becomes pale and blank, his lungs cease to play, and, after a moment of absence or giddiness, he is himself again. His memory has kept no record of this sad passage in his history, and if it had escaped the notice of others he might remain in happy ignorance of it. Or, in addition to these symptoms, a livid flush may succeed to the paleness of the countenance, the veins of the neck and forehead may start out in prominent relief, the face may turn slightly towards one of the shoulders, and there may be some convulsive twitching in the face and neck and arms. In such a case there is no scream or cry, no fall, no bitten tongue, no foam at the mouth, and at most there is only some obscure gurgling

in the throat, some staggering, and some slight moistening of the lips with saliva. In such a case the convulsive movements are very partial, rarely extending beyond the face, neck, or arms, but in some few instances the whole frame may be agitated by one or two violent convulsive shocks. This state of giddiness and absence and partial spasm may be followed by fatigue, loss of memory, confusion of thought, depression of spirits, or irritability of temper, and at times it may end in drowsiness or actual sleep; but usually recovery is almost instantaneous. At the same time there is reason to believe that dementia is a more likely, as well as a more speedy, consequence, in this slight form of the disease (*le petit mal*) than it is in ordinary epilepsy (*le grand mal*).

In some of these cases, moreover, it would seem, not only that the patient does not fall or cry, or suffer from general convulsion, but that the state of intellectual eclipse—the most characteristic symptom of epilepsy—is far from complete. Esquirol says, “il est des accès dans lesquels on n’observe pas la perte de connaissance;”* and M. Herpin† directs particular attention to such cases. These cases, however, are very rare, or at any rate they are very difficult to detect, if other proof be wanted than the mere assurance of the patient. They are common enough in certain chronic diseases of the brain, as meningitis or tumour, but in simple epilepsy they would seem to be extremely rare. Cases, however, are on record, and the following, which fell under my own notice, may perhaps be added to the number.

CASE.—This patient, Mr. P—, æt. 18, was the son of a farmer living near Leicester. Early in the summer of 1855, he suffered from unusual palpita-

* Art. “Epilepsie,” in ‘Dictionnaire des Sciences Médicales,’ 8vo, Paris, 1815.

† ‘Du Prognostic et du Traitement Curatif de l’Epilepsie,’ 8vo, Paris, 1852.

tions upon any exertion, and later in the same year he had frequent fits, such as will be presently described. He had been growing rapidly, and in addition to this he had imprudently exposed himself to the sun while out in the fields during harvest. These attacks occurred at frequent intervals until the beginning of May, 1857, when one night he had a severe fit, in which there was convulsion and bitten tongue.

Mr. P— came to see me on the 21st May, 1857, and at that time I found him to be a tall, over-grown lad, with a dilated and sluggish pupil, a weak, slow pulse (60), and a curiously shaped head, evidently not bright, but without any positive signs of mental deficiency. I had just noticed these points, and my finger was still on his pulse, when he told me that he was going to have a fit. Upon looking up, I saw that his features had become pale and drawn, his eyes fixed, his lips slightly moistened with saliva, and his face already turned round to the right shoulder. I also heard certain choking sounds in the throat. On placing my hands on each side of the chest, I found that the respiratory movements were altogether at an end. These symptoms continued for a sufficient time to allow a livid flush to take the place of the original paleness of the countenance, and then, drawing a deep sigh, he suddenly recovered. His pulse at the beginning of the attack was 60, and very weak; after the attack it rose to 80, became fuller, and the heart throbbed a little. On returning to himself, he stated spontaneously that his vision was troubled in the attack, and that he had heard a remark which I had made to his mother, who was with him. He also told me that such attacks occurred several times a day, and that he never lost his consciousness in them.

I saw Mr. P— again on the 1st of November, 1857, and on this occasion I had another opportunity of witnessing an attack. In this instance, as in the one just described, the face was first pale then dusky, the features were drawn, the eyes fixed, the lips moistened with a little saliva, the face twisted round to the right shoulder, the chest immovable, and choking sounds proceeded from the throat; and in addition to these symptoms, the right arm was rigidly contracted and agitated by slight convulsive shocks, and both hands had seized tight hold of the arms of the chair in which he was sitting. Wishing on this occasion to test the condition of the consciousness, I pinched his hand, and asked him if he felt pain. He moved his head a little, and there was an expression in his eye and a motion in his lip, which appeared to indicate a wish to speak. I then held my watch before his face, and as I did this, he began, as it seemed, to try to overcome the spasm which had twisted his head round, and in a moment or two succeeded. On returning to himself, he said that he had made several voluntary attempts to overcome the spasm which had turned his head round to the side, and that he always did this if the fit continued longer than usual; and on asking what I had done during the attack, he told me, without the least prompting on my part, that

I had pinched his hand and held my watch before his face, or what he supposed to be my watch, for, as in the former instance, his vision was greatly troubled. After the attack his countenance had a flushed and jaded expression.

I have seen Mr. P— since the last date, and witnessed another attack of the same nature, only less marked than the two which have been described. I also learn that he has had a repetition of the severer form of attack on more than one occasion.

3. *Of the appearances after death in ordinary epilepsy.*

The morbid appearances after death from *ordinary epilepsy* are necessarily very obscure, if the case have really been one of simple epilepsy, and not one of epileptiform convulsion connected with some special disease. In cases fatal during the fit the brain has been found to be congested; but this appearance is clearly owing to the mode of death, and it is allowed to be so. In cases, again, where epilepsy has been complicated with insanity, the brain or its membranes may present various signs of inflammation, or of changes more or less akin to inflammation; but these signs are clearly referable to the mental disorder, and for this reason—that they are as common, or more common, in insanity without epilepsy. In other cases there are signs of degeneracy, such as pallor of the grey matter, softening, induration, atrophy, dropsical effusion; but these are the very signs which belong to the demented state. It is this very fact, however, which furnishes some grounds for supposing that signs of this character may have something to do with epilepsy. It does so, because the demented state is intimately connected with convulsive disorder; for if a demented person be not epileptic, he is almost sure to be affected with palsied shakings, or cramps, or spasms, in one form or another. In other cases, again, the skull may be thicker and heavier than usual, and the several internal projections—as the clinoid processes—may be considerably developed, or various

parts of the dura mater may be converted into bone. Indeed, there are no constant changes in the brain proper or in its coverings—not even that change in the pituitary body of which so much has been said by Wenzel;* for, writing of it, M. Rokitansky † says that he has “frequently failed to discover it in those who had notoriously suffered from epilepsy and convulsions,” and that he has “met with it in others who were thoroughly healthy.” It is in the medulla oblongata, indeed, that we alone meet with any appearances after death which can be regarded as constant. In early cases of epilepsy, it is true, we may fail to find anything characteristic even here; but in confirmed cases this organ is harder than natural, from the interstitial deposit of a minutely granular albuminous matter, or else softened, swollen, and presenting evident signs of fatty degeneration. The posterior half is 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 are seen to be dilated to thrice their natural dimensions, and with their walls much thickened. These vessels, moreover, are 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 tongue in a fit, and in the course of the roots of the vagus in epileptics who were not in this habit. These facts, for the knowledge of which we are indebted to Professor Schroeder van der Kolk, ‡ are based upon fifteen post-mortem examinations.

* ‘Beobacht. über den Hernauhang Fallsüchtiger Personen,’ &c., 8vo, Mainz, 1810.

† ‘Manual of Pathological Anatomy,’ translated for the Sydenham Society, by C. H. Moore, vol. iii, p. 434.

‡ ‘On the Proximate Cause and Rational Treatment of Epilepsy,’ translated by Dr. W. D. Moore, for the New Sydenham Society, 8vo, London, 1859.

4. *Of the pathology of ordinary epilepsy.*

In this section I propose to review, in succession, the condition of the circulation and respiration, and the condition of the nervous system; for this course seems to be that by which I may most readily arrive at safe conclusions respecting the pathology of ordinary epilepsy.

I. There is nothing in the interparoxysmal state which is calculated to justify the notion that ordinary epilepsy is connected with a state of vascular fulness or over-activity. Plethora, in the form so often exemplified in the butcher is never met with, and feverish excitement of the circulation is of very rare occurrence, even as an accident. On the contrary, the facts would appear to show that the habitual state between the paroxysms of ordinary epilepsy is one which is marked by a weak and slow pulse, by cold extremities, and by shallow, or retarded, or sighing breathings, and which is most marked in this manner when the danger of an attack is most imminent. There are, no doubt, a few cases of *epileptiform convulsion* in which the circulation and respiration may exhibit a greater degree of activity between the paroxysms than that which characterises the interparoxysmal state in ordinary epilepsy; but, as will be seen in another place, there is nothing to show that the epileptiform convulsion is connected with a state of vascular over-activity, and very much to show the contrary. In a word, there is nothing in the history of epileptiform convulsions to cast a doubt upon the conclusion which seems to be necessitated by the facts—that the interparoxysmal state in ordinary epilepsy is marked by wanting vigour both in the circulation and in the respiration.

In the fit itself the facts are altogether unmistakeable. At the instant of the fall a corpse-like pallor overspreads the countenance, and the pulse at the wrist and elsewhere is almost or altogether imperceptible. A moment or two later, and,—with a few exceptions in which the pallor and pulselessness of the commencement remain throughout the fit,—the black and bloated face, the choking sounds, and the suspension of all respiratory movements show very plainly that the state is one of absolute suffocation. In either case, that is to say, the convulsion is associated with the absence of arterial blood. Nor is any proof to the contrary to be found in the fact that the convulsion is almost always accompanied by a full pulse and a throbbing heart; for, instead of showing, as they have been so often supposed to show, that a larger quantity of *red blood* is being pumped into the arteries at the time, these phenomena may do nothing more than show that these vessels are then labouring under a load of *black blood*, precisely as they have been seen to labour (pp. 15-21) in ordinary suffocation. Indeed, it is very evident that the full pulse and the throbbing heart of the epileptic paroxysm must have this latter significance, for the livid, black, and bloated head and neck, and the complete suspension of all respiratory movements, necessitate the conclusion that the supply of arterial blood is cut off at the fountain head, and that any blood which is then coursing through the vessels is not red, but black.

When the convulsion is over, the respiration is speedily re-established, and the readmission of arterial blood into the system may be attended with some transient and inconsiderable febrile reaction; but this reaction has clearly nothing to do with the convulsion, for when this reaction is present the convulsion is absent; and if the convulsion return, it is not until every trace of the reaction has first taken its departure. When the convulsion is over, indeed,

there is little to notice in the state of the circulation and respiration.

In this point of view, then, the conclusion respecting the convulsion of epilepsy is the same as that which has been already drawn respecting the convulsion which is witnessed in experiments like those of Sir Astley Cooper and MM. Kussmaul and Tenner; and this is—that the convulsion is coincident with the cessation of the circulation of arterial blood. And this is the only conclusion which can be drawn, for, after what has been said in the physiological premises, it is not to be supposed that the convulsion is to be ascribed to the circulation of venous blood in the vessels.

— But, it may be asked, is there no change in the blood itself? Is there not some important truth in the “humoral theory of epilepsy,” as recently advanced by the late Dr. Todd? “I hold,”* said this distinguished physician, “that the peculiar features of an epileptic seizure are due to the gradual accumulation of a morbid material in the blood, until it reaches such an amount that it operates upon the brain in, as it were, an explosive manner; in other words, the influence of this morbid matter, when in sufficient quantity, excites a highly polarized state of the brain, or of certain parts of it, and these discharge their nervous power upon certain other parts of the cerebro-spinal centre in such a way as to give rise to the phenomena of the fit. A very analogous effect is that which results from the administration of strychnia, which is best seen in a cold-blooded animal, like the frog. You may administer the drug in very minute quantities for some time without producing any sensible effect; but when the poison has accumulated in the system up to a certain point, then the smallest increase of dose will immediately

* ‘Med. Times and Gazette,’ 5th and 12th August, 1854.

give rise to the peculiar convulsive phenomena. This is the humoral theory of epilepsy. It assumes that the essential derangement of health consists in the generation of a morbid matter, which infects the blood; and it supposes that this morbid matter has a special affinity for the brain, or for certain parts of it, as the strychnia, in the case just cited, exercises a special affinity for the spinal cord. The source of this morbid matter is probably in the nervous system, it may be in the brain itself. It may owe its origin to a disturbed nutrition—an imperfect secondary assimilation of that organ—and in its turn will create additional disturbance in the functions and nutrition of the brain.” And again:—“According to the humoral theory, the variety in the nature and severity of the fits depends on the quantity of the poisonous or morbid material, and on the part of the brain which it chiefly or primarily affects. If it affect primarily the hemispheres, and spend itself, as it were, on them alone, you have only the epileptic vertigo. If it affect primarily the region of the quadrigeminal bodies, or if the affection of the hemispheres extend to that region, then you have the epileptic fit fully developed.”

This theory is based upon the well-known connexion between the presence of urea (or carbonate of ammonia resulting from the decomposition of urea) in the blood and one form of epileptiform convulsion; and it might also have been based upon the connexion between convulsion and blood overloaded with bile. But if there be any evidence in these facts in favour of the existence of this hypothetical morbid material, there is none in favour of the idea that the *modus operandi* of the material is in exciting a highly polarized state of any part of the nervous system. On the contrary, it is certain (as will be shown in another chapter) that the action of the brain and of the nervous system generally is reduced to the very lowest ebb at the

time when convulsion is brought about by the accumulation of urea and bile in the blood; and it is not less certain that strychnia, instead of acting as Dr. Todd supposes it to act—that is, by exciting a highly polarized state of certain parts of the nervous centres—acts by rendering the blood less capable of becoming arterial, and by diminishing the electrical currents of both nerve and muscle.

There is little doubt, however, that retained excretions may play an important part in the production of epilepsy. A free discharge in the office of excretion, not only in the kidneys and liver, but in every excretory organ, is essential to the preservation of healthy blood; and it may well be believed that an imperfect discharge of the office of excretion, *in one or other of the excretory organs*, may lead to the accumulation of effete matter in the blood, and that this accumulation of effete matter may be a not unimportant cause in bringing about an attack of epilepsy. But there is no reason for supposing that the blood under these circumstances is rendered more stimulating. On the contrary, the conclusion which arises out of the history of the cases where the urine or bile is suppressed is the natural conclusion, and this is, that blood thus altered is less fit to discharge its several offices,—in other words, less stimulating.

Nor does there appear to be any reason for supposing that venous congestion has a more important part to play in the production of epilepsy than that which has been assigned to arterial injection. No doubt the veins of the brain and head generally are congested from a very early moment, but there is a moment antecedent to this in which the death-like pallor of the face is a sufficient proof that the veins were emptier than usual before they became congested. At any rate, the acknowledged anatomical difficulty must be overcome before it can be supposed that Dr. Marshall Hall's hypothesis of *trachelismus*—or the

prevention of the return of blood from the brain by the spasm of certain muscles in the neck—has anything to do with the causation of epilepsy.

It would seem, then, as if there was something utterly uncongenial between the convulsion of epilepsy and arterial excitement. It would seem, indeed, as if the spasms, as well as the loss of consciousness and sensibility, were connected with want of arterial blood—vessels empty of red blood in the first instance, vessels filled with black blood afterwards. It is not improbable, also, that the blood may have been previously rendered less stimulating by the retention of something which ought to have been eliminated by one or other of the organs of excretion. In a word, the phenomena are entirely in harmony with the previous considerations respecting muscular motion; for, according to them, the action of arterial blood is to antagonize contraction, and not to cause it.

2. Interrogating the nervous system, the answer is not different from that which the state of the circulation and respiration would lead us to anticipate.

The facts will scarcely warrant the idea that epilepsy is connected with anything approaching to over-action of the *brain proper*. On the contrary, everything seems to point to a state which is the very opposite of over-activity. Thus, the comparative want of memory, and intelligence, and fancy, and purpose, which marks the interparoxysmal condition—the utter annihilation of everything mental in the fit itself—and the gloom and prostration following the fit, are facts which can have no double meaning.

Nor is a contrary opinion to be drawn from the morbid appearances which are disclosed after death. If these chance to indicate previous inflammation, it does not follow that convulsion had any direct connexion with the inflammation as inflammation; on the contrary, the convulsion may have happened before or after the inflammation, when

the energies of the brain were prostrate or exhausted—an alternative which we shall see to be the correct one when we come to speak of epileptiform disease connected with special disease of the brain. And surely it is not possible to draw any but one conclusion from the appearances which are common to epilepsy and dementia—pallor of the gray substance, atrophy, chronic softening, induration, dropsical effusion, and the rest?

But what of the state of the *medulla oblongata*? What of that organ, which, as Dr. Van der Kolk has rightly argued, is especially connected with epilepsy, not only by the post-mortem appearances, but also by the seat and bilateral character of the spasms?*

The spasms of epilepsy 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. The spasms of the walls of the chest and abdomen, which are the most prominent and marked features in the complete attack of epilepsy, and which may be so fierce and unyielding as to cause fatal suffocation, may also be looked upon as pointing to the same nervous centre; for a similar state of things is brought about by the action of an induction coil upon the great afferent nerve of this centre—the pneumogastric.

The bilateral character of the spasms is another argument that the medulla oblongata is especially affected in epilepsy. The lateral halves of this organ are connected in the most intimate manner by transverse fibres and commissures—much more intimately than the lateral halves of the brain and spinal cord; and hence it is that the corresponding nerves belonging to the two sides of the medulla oblongata are under a stronger physical necessity to act

* Op. cit.

together than that which rules the corresponding nerves belonging to the two sides of the brain and spinal cord. In the case of the two latter centres, the nerves belonging to one side may be paralysed or otherwise affected, without any obvious injury to the nerves of the other side; but not so in the case of the latter centre. Indeed, it is evident that the actions which emanate from the latter centre—the play of the features, the motion of the tongue, the vocal adjustments of the larynx, the respiratory movements, &c.—must at once come to an end unless there be the strictest sympathy and concert in the action of the corresponding nerves of the two sides. Now in epilepsy the spasms are always more or less *bilateral*, and for this reason, therefore, it may be supposed that they have some special connexion with a nervous centre of which one lateral half cannot act without the other.

The appearances after death also point to the medulla oblongata as especially concerned in the production of epilepsy. In an early stage of the disorder, we may fail to find any characteristic changes; but, in confirmed cases, the texture is harder than natural, from the interstitial deposit of a minutely granular albuminous matter, or else softened, swollen, and exhibiting signs of evident fatty degeneration. The posterior half of the medulla oblongata, moreover, is redder and more hyperæmic than it ought to be; and, on examining the blood-vessels in this congested portion, they are found to be of thrice their natural dimensions, and with their walls much thickened and altered—this dilatation and alteration being chiefly in the corpus olivare and in the course of the hypoglossus in the case of epileptics who bite their tongue, and in the course of the roots of the vagus in the case of epileptics who do not bite their tongue.

It is evident, then, that the medulla oblongata is especially affected in epilepsy; but it does not follow, as Dr. Van

der Kolk supposes, that the essential cause of the convulsive affection is to be found in an exalted sensibility and activity of the ganglionic cells of this centre.

In favour of this view,—that epilepsy is dependent upon exalted sensibility and activity of the ganglionic cells,—this physician appeals to the fact of spasm, to the presence of a full, bounding pulse, and to the freedom from attack which is for some time the fruit of an attack, particularly if this has been violent; but it is easy to see that this appeal is one which cannot be allowed. After what has been said about muscular motion, it is not possible to allow that spasm is an argument in favour of exalted sensibility and activity in ganglionic cells. After what has just been said about the phenomena of the circulation in epilepsy, it is not possible to allow that the full and bounding pulse of the epileptic paroxysm is produced by increased injection of arterial blood into the vessel. If this could be allowed, there would be no difficulty in supposing that this pulse must imply exalted action of the medulla oblongata—for the action of any organ must be in direct relation to the amount of arterial blood supplied to it; but, so far from this being the case, the simple fact is that the full and bounding pulse of the epileptic paroxysm is the pulse of suffocation—a pulse which is filled with black blood, not with red. And thus the notion of increased injection of arterial blood during the epileptic paroxysm falls to the ground, and with it the theory of exalted activity of the medulla oblongata, which has been based upon it. Nor can the freedom from attack, which is for some time the fruit of an attack, be appealed to as a certain proof that the attack is the sign of the discharge of some overcharge of excitability previously present. On the contrary, it may be argued with some degree of plausibility, from certain facts which have to be mentioned hereafter, that the attack was preceded by depression of the circulation and

innervation, that the convulsion supervened when this depression had reached a certain point, and that the recurrence of the attack was prevented for a time by the state of reaction in the circulation and innervation, which is a consequence of the convulsion. The case may be one, indeed, of which the history of the rigors of ague may serve as no inapt illustration; for here we have, first, the circulation failing more and more until the bathos of the cold stage is reached, and then a state of reaction which banishes the rigors most effectually so long as it continues.

It would even seem as if appeal might be made to the appearances after death, and to the actual condition of the circulation in the fit, for positive arguments against the idea of anything approaching to exalted action of the medulla oblongata.

The signs of fatty degeneration can have but one significance—under-action, not over-action. The interstitial deposit, also, implies an equivalent absence of healthy nerve structure, and so does the dilated condition of the blood-vessels; and this absence of nerve structure must necessitate a corresponding absence of nervous action. The appearances after death, indeed, if they show anything, would seem to show that the medulla oblongata of the epileptic is *damaged in structure*, and because damaged in structure, *weaker in action* than it ought to be.

The great argument against the idea of anything like over-action of the medulla oblongata in epilepsy, however, is to be found in the state of the circulation; for if, as may safely be assumed, the activity of any organ is in direct relation to the activity of the circulation of *red blood* in that organ, how far from anything like over-action must be the state of things in which, as is the case in the epileptic paroxysm, the vessels are at first comparatively empty of red blood, and afterwards completely filled with black blood?

Nor can the curious discovery of Dr. Brown-Séquard,* that certain injuries of the spinal cord are followed by an epileptiform affection, be construed into an argument that there is anything like a state of exalted action of the *spinal cord* in epilepsy. This curious result, which is brought about by puncturing or dividing more or less completely almost any part of the spinal cord, is developed, not immediately, but in the course of three or four weeks after the injury. The attacks, once developed, occur spontaneously at various intervals, often several times a day; they may also be brought on by pinching or otherwise irritating the portion of the skin which corresponds to the region of the whiskers in man. This excitable spot is supplied by twigs belonging to the sub-orbitary, the auriculo-temporalis, the second, and perhaps the third, cervical nerves; and it is a curious fact, that the irritation which brings on a fit when applied to the skin in which these twigs terminate has no such effect when applied to the twigs themselves. Any other part of the skin may be pinched or irritated with impunity, but this one spot can scarcely ever be touched without at once bringing on a fit.

These facts are very curious, and, in the main, very unintelligible; but this much at least is evident, that they do not countenance the idea of any over-action of the spinal cord in epilepsy. The fact that the epileptiform affection does not make its appearance until four or five weeks after the injury would appear to show very clearly that the fits have nothing to do with that local inflammation in the cord which may be supposed to have been set up in the first instance by the injury. After such a lapse of time, indeed, the natural conclusion would rather seem to be, that any over-action of the cord, arising from the

* 'Researches on Epilepsy; its Artificial Production in Animals, and its Etiology, Nature, and Treatment in Man,' 8vo, Boston, 1857.

inflammation produced by the injury, must have died out, and left the cord damaged, weakened, under-acting. Nor is a contrary conclusion to be drawn from the over-incitable condition of the nerves proceeding from the neighbourhood of the cheek or cheeks—for both sides of the face are thus affected, if both sides of the spinal cord have been injured. What the full significance of this curious fact may be we have yet to learn, but at any rate there is no reason to suppose that this over-incitable condition of the skin implies an over-acting condition of the nerves or nervous centres concerned in the phenomenon. The over-incitable portion of skin is not over-sensitive, for the animal manifests no signs of uneasiness when it is handled immediately after a fit. Over-sensitiveness, moreover, would seem to have nothing to do with the matter. At any rate, pain, and not convulsion, is the consequence of handling those portions of the skin of the animal which may have been rendered highly hyperæsthetic by the injury to the cord which brought on the convulsions. It is certain, also, that a somewhat similar condition of over-incitability is brought on when, as in several experiments related in the second chapter, the skin is cut off from the full influence of the nervous centres; and hence the natural inference would be, that the action of the nervous centres upon the over-incitable portions of skin in the epileptic guinea-pig is *minus* rather than *plus*.

As in the former instances, however, so here, we turn to the condition of the circulation and respiration in order to know what is the actual functional condition of the spinal cord in the epileptic paroxysm; and so turning, we see that the action of the cord under these circumstances must be almost or altogether *nil*. For what action can there be at a time when little or no arterial blood is injected into the vessels, or where these vessels are gorged with black blood?

A similar argument will also dispose of the idea of over-activity of the *ganglia of the sympathetic system* as a cause of epilepsy. It is very possible that the contracted state of the arteries, which is implied by the death-like pallor of the countenance and the comparative pulselessness at the wrist, may show that the coats of the vessels are in a state of spasm; and it is also possible that the cause of this spasm may have to be sought in the sympathetic system; but it does not follow that over-action of this system is this cause. On the contrary, the experiments of Drs. Kussmaul and Tenner, already referred to (p. 30), show most conclusively that strong epileptiform convulsion is possible when the action of the sympathetic ganglia is entirely suspended by arresting the supply of blood to these organs.

And certainly no opposite conclusion is to be drawn from the vague and undefinable sensations or movements, very varying in character, but all comprehended under the term *aura*—sensations of pain, numbness, tingling, a feeling as of a current of cold vapour, movements of shuddering or spasm, beginning in a distant part and travelling towards the head; for the most probable interpretation of these symptoms is that of Dr. Watson*—that they are in some degree analogous to the numb and tingling feelings which are the frequent precursors of paralysis and apoplexy, or to the globus of hysteria—phenomena which by the most perverse process of reasoning can scarcely be supposed to indicate other than a state of defective innervation somewhere.

But, it may be asked, is there nothing else? Is there no peculiar state of the nervous system in epilepsy? Is there no "*morbid irritability*"? In order to answer this question, it is necessary to ask another—What is "*morbid*

* 'Lectures on the Principles and Practice of Medicine,' 4th edit., 8vo, London, 1857, vol. ii.

irritability" ? It is not inflammation ; it is not fever ; it is some indefinable and negative state which occurs frequently in teething, in worm disease, in uterine derangement, and in many other cases—a state in which the patient is unusually depressed by depressing influences, and unusually excited by exciting influences. But what is this state ? Is it anything more than mere exhaustion ? In difficult teething, the strength is worn away by pain and want of sleep ; in worm disease, the parasites help to starve and exhaust a system already ill fed and feeble ; in uterine derangement, the health is undermined, in all probability, by pain and by sanguineous or other discharges. In each case there is unequivocal exhaustion of body and mind, and the signs of "morbid irritability" appear to be nothing more than the signs of such exhaustion. A weak person is more affected by the several agencies which act upon the body from within and from without, and he is so because he is without some of that innate strength which belongs to the strong person ; and the person who is "morbidly irritable" is, in reality, one who, for want of this principle of strength, is inordinately excited or depressed by the several exciting or depressing influences which are continually acting upon the system. In a word, this undue "morbid irritability" may be nothing else than the natural consequence of that general want of power, the signs of which are written so legibly upon the vascular and nervous systems of the epileptic. Thus interpreted, indeed, "morbid irritability" only becomes another name for inefficient innervation.

In these points of view, therefore, *the pathology of ordinary epilepsy* would seem to be altogether unintelligible upon the current theory of muscular motion—a theory according to which the muscles are supposed to contract convulsively, because they are subjected to excessive stimulation. In these points of view, indeed, the pathology

of this disorder would seem to be only intelligible when it is interpreted by the theory of muscular motion which is advanced at the commencement of this volume.

5. *Of the treatment of ordinary epilepsy.*

Arguing from the premises, physiological and pathological, it may be inferred that the fact of convulsion cannot rightly be urged as a plea for the adoption of "lowering measures." It may be inferred, indeed, that the great desiderata in epilepsy are a more active nervous system, and a more vigorous circulation, and that the remedies to be sought after are those which will bring about these results.

I. I know of no facts which show that a low diet is beneficial in ordinary epilepsy. On the contrary, I know of many instances where the patient has been undoubtedly benefited by the abandonment of such a diet, and the substitution of a more liberal allowance of animal food. It is an established fact that an overloaded stomach is a very common cause of the attack, and that it is of primary importance to avoid the risk of such overload. But it is also true that an epileptic ought to fear an empty stomach almost as much as an overloaded stomach, and, therefore, in avoiding one danger he must be careful not to fall into the other. In a word, quantity and time are all-important in the regulation of the meals of an epileptic; and if care be taken to insist upon these points, the quality of the food eaten, is, I take it, of comparatively little moment. Indeed, I have never found it necessary to disturb the arrangements of a household by requiring an epileptic to be fed differently to the rest of the family. At the same time, I am disposed to think that the diet of an epileptic ought to contain more than an average quantity of fatty and oily

matters. What would seem to be especially wanted are the materials for nourishing nervous tissue; and as this tissue contains a very large proportion of fatty and oily materials, it is to be supposed, perhaps, that these materials ought to be supplied in the food. Nay, it would seem to be possible that nervous tissue may itself be proper food in such cases, and that any person suffering from epilepsy, or any other malady in which there is reason to believe that the nervous system is wanting in quantity or energy, may be acting far from wisely in excluding the brains of animals from his dietary. I have had no opportunity as yet of testing the practical value of this idea, and I fear that few such opportunities may present themselves until some ingenious cook has invented some more convenient and palatable modes of cooking brain than those which are to be found in the cookery-books.

As a rule, also, stimulants of one kind or another would seem to act beneficially. In some cases, it is true, malt liquors may be objectionable; in some cases, too, wine may be unnecessary; but in the great majority of cases I believe that unquestionable good will result from the judicious use of sherry, or especially of claret. Indeed, I am satisfied that epileptics and nervous patients generally will have good reason to overlook the shortcomings of the recent treaty, by which, at reasonable rates, they are able to substitute the light wines of France for the strong wines of Spain and Portugal, and for the heavy ales of our own breweries. I believe, too, that in a great number of cases coffee will be a more suitable beverage than its less stimulating companion, tea, particularly at the commencement of the day, and that in many instances it will be expedient to abstain from both coffee and tea—at all times.

It is, no doubt, of great importance to prevent the accumulation of effete matters in the bowels, and to remove such accumulation when it exists; but whether purgatives

are the proper remedies in this case is another question. If the bowels do not act with sufficient regularity, there is, in all probability, some error in the diet, some excess of animal food, some deficiency of fatty and oily matters, or of culinary vegetables and fruit; and the first thing to be done is, obviously, to correct this error. And this is often all that is wanted, if care be taken to explain to the patient that his bowels can act without purgatives, and that he need not—particularly if advanced or advancing in life—be altogether cast down, if, now and then, they do not act every day. Indeed, if the diet be properly regulated, and this explanation made, the patient will generally have the satisfaction of finding his tongue clean, when he remembers to look at it, and of forgetting his stomach and bowels altogether. Or if the result be not quite so satisfactory, an occasional injection of cold water or salt and water, upon getting up in the morning, will rarely fail to set matters right, without disturbing the digestion in any way, or giving rise to disagreeable feelings of depression or irritability.

As a rule also, it would appear to be advisable to order the habits in such a way as to save the strength as much as possible. Proper exercise is, of course, necessary; gymnastic exercises by which the chest is expanded, and the respiratory capacity increased, are, without doubt, most valuable adjuvants; but it is no less certain that fatigue is a common cause of the epileptic attack, and that this fact must be borne in mind in regulating the amount of exercise. As a rule, too, it would seem that epileptics require more than the average amount of sleep to enable them to recover from the multifarious fatigues of the day and night.

II. The more strictly medicinal part of the treatment of ordinary epilepsy is a subject of no small difficulty. The treatment of the present day is very different from what it

was when almost all disorders were referred to inflammation or over-action of one kind or other. Practically, the lancet is now rusty from long disuse, and leeches are in a fair way of being left undisturbed in their swampy homes; practically, also, it has ceased to be the habit to distress the stomach and bowels by the use of strong purgatives or emetics; and this change may be appealed to as an argument that "lowering measures" had disappointed the hopes of those who had tried them so long and so patiently, and who gave them up so unwillingly.

In a great many cases the appearance of an epileptic is one which is well calculated to suggest the idea that a course of ordinary tonics, as steel, or quinine, or cod-liver oil, would be likely to do good; and there is every reason to believe that good is often done by adopting the suggestion.

Dr. Watson, to whose judgment all will willingly bow, says,* "Of all the metallic remedies, I prefer some preparation of zinc or iron;" and in so saying he expresses what is, I believe, the opinion of the great majority of medical practitioners in this country. At the same time, I think that there are signs of a change of opinion, and that many are disposed to give the preference to iron, and to place it before zinc, not after zinc. At any rate, I have no hesitation in saying that I have often found unequivocal improvement in the general health, and as unequivocal amelioration in the fits, under a course of iron, and that in several cases where this improvement and amelioration was most marked and most rapid the patient had been taking zinc or copper previously.

The voice of experience is less loud and peremptory in favour of quinine than it is in favour of iron, and, as a general rule, there is reason to believe that quinine will be less serviceable than iron. Quinine has been used by more than one writer, by MM. Rostan and Piorry among

*Lectures,' 4th edit., vol. ii, p. 659.

the rest, and several cases of epilepsy are on record which are supposed to have been cured by its aid. I may say, also, that I have often used it with advantage for a time. I remember, in particular, several epileptics who were sent from marshy parts of the country, and who made rapid progress under a course of quinine; but I also remember other cases, apparently suitable, in which harm rather than good was the result of such a course, and, upon the whole, I am obliged to think less favorably of quinine as a remedy for the majority of cases than I was once disposed to think.

I do not know that I can appeal to the experience of others for evidence in favour of cod-liver oil as a remedy in epilepsy; and, until very recently, I did not know that I had evidence in my own experience which is calculated to support this appeal. I find, however, on looking over a considerable number of cases in which I had tried the effect of a course of common tonics for a while, that the weight of evidence is less in favour of steel or quinine than I expected, and much more in favour of cod-liver oil than I expected; and I am now disposed to think that the result might have been still more satisfactory if I had recognised this fact before, and given the oil more liberally and for a longer time. I can readily admit that cod-liver oil is "floating fuel" for the fire of respiration, that it burns vicariously in this fire in place of the tissues of the body, and that, so burning, it may act as a tonic by giving these tissues additional time to become properly nourished. But at the same time I cannot forget the facts which show that the circulation of the blood, and the generation of animal heat, are under the direct control of the nervous system, and that the action of a tonic in improving the circulation, and in increasing the natural warmth, cannot be wholly explained without referring to this system. I cannot forget, for example, that the circulation and calorification are depressed when the action of the nervous sys-

tem is depressed, as during fear, and that they are exalted when the action of the nervous system is exalted, as in joyous excitement. I cannot forget facts which may be said to show that the activity of the circulation and calorification are in direct relation to the activity of the innervation, and, this being the case, I am disposed to think that the power of cod-liver oil in increasing the activity of the circulation and calorification is in part due to the fact that it increases the activity of the brain and nervous system by supplying oily matter which may be necessary to the proper nutrition of the oily parts of the nervous tissue. This being the case, indeed, I am disposed to think that cod-liver and other oils may have some claim to be regarded as of special use, not only in cases of epilepsy, but also in all other cases in which the brain and nervous system are in need of a tonic. But, be this as it may, I have no doubt whatever that cod-liver oil did good in many cases of epilepsy in which I gave it a fair trial, and that it contrasted favorably in this respect, not only with quinine, but also with steel.

In other cases of epilepsy—in all cases, perhaps, at some time or other—the state of the patient suggests the necessity of some stimulating agent; and here again there is good reason to suppose that good is done by yielding to this suggestion.

“If,” says Dr. Watson,* “I were called upon to name any single drug from which, in ordinary cases of epilepsy, I should most hope for relief, I should say it was the oil of turpentine. And I find that other physicians have come to the same conclusion. Dr. Latham, the elder, was, I believe, the first person who made known its efficacy in this disorder. Foville states that he has seen excellent effects from it. It is highly spoken of by Dr. Perceval in the ‘Dublin Hospital Reports.’ It is not

* ‘Lectures,’ &c., 4th edit., vol. ii, p. 622.

given in large doses, but in smaller ones frequently repeated—from half a drachm to a drachm every six hours.” And that turpentine, thus given, is a very valuable remedy when the patient is unusually prostrate, and particularly when this prostration is kept up and aggravated by frequent fits, will, I am sure, be doubted by no one who has been called to treat a number of such cases.

Another remedy which puts in its claim for approval in this place is valerian. This is a favorite remedy, both in this country and elsewhere, and its claims, though not equal to those of turpentine, appear to be in every way deserving of attention. Recommended by Aretæus and Dioscorides, and in use ever since, it was never other than a favorite remedy. Now the prominent action of valerian is that of a stimulant—an action depending upon the presence of a volatile oil, of which one portion is a volatile acid, capable of forming a salt with bases, and known under the name of valerianic acid; and it is a natural question, after what we know of the action of turpentine and the depressed state of the circulation and innervation in epilepsy, whether this stimulating action does not show that this drug may be efficacious, and explain the secret of its efficacy.

Under these circumstances, therefore, believing that the state in epilepsy is one which will often suggest the use of stimulants, and having these practical arguments in favour of turpentine and valerian, the question which arises without any prompting is whether camphor, or naphtha, or ether in its various forms, or ammonia, or one of the stimulant gum-resins, or musk, or castor, or any other stimulant, will be of use in epilepsy? And so far as I can judge, the answer to this question is not different from what might be expected.

That camphor is often a very valuable remedy I have no doubt whatever. In doses of about three grains twice or

thrice a day, I have often seen such results as to justify me in ascribing to camphor the virtues belonging to turpentine, with this addition in its favour, that it is not unpleasant to the taste, and that it exercises, or seems to exercise, a directly quieting influence over the generative and urinary organs. And this is no small addition in its favour, for there is reason to fear that turpentine may occasionally aggravate that erotic tendency which seems to exist in many cases of epilepsy, and which prompts to acts which must, without doubt, be reckoned as no unfrequent causes of the fit.

Naphtha would also seem to have the advantages, without the disadvantages, of turpentine. In doses of from half a drachm to a drachm, and taken for some time, I have often seen what seemed to be unequivocal evidence of the beneficial action of this remedy. I find, also, that the patient soon becomes indifferent to its disagreeable taste, particularly if it has been redistilled more than once. The taste is also a good deal masked by the addition of tincture of valerian or tincture of hop, one or both, and at the same time the combination is one which may be supposed to increase the efficacy of the medicine.

Of the stimulant gum-resins my experience is not very ample; but I think I have seen enough to enable me to say this, that they are occasionally of considerable value. Of the action of musk and castor I have no experience.

With respect to the different forms of ether—Hoffmann's anodyne, chloric ether, spirits of nitric ether, and so on, there can, I think, be no doubt as to their great value as occasional remedies; and the same may be said of ammonia. In the majority of instances, indeed, it is only to this class of remedies that we can trust for warding off a fit. In severe cases, also, ammonia would seem to be of use, not only as a stimulant, but also as an alkali—by helping, that is to say, as an alkali will do, to rid the blood

of uric acid, or some analogous impurity which has increased the danger of a fit by rendering the blood less stimulating.

But however beneficial these stimulants may be in many cases, and in all cases at one time or other, it is necessary to confess that they will not do all that is wanted. They will contribute important aid in bringing about the cure, but, like the ordinary tonics which have been already considered—steel, quinine, and cod-liver oil—they seem to have no special claim to be considered as specific remedies for epilepsy.

— But, it may be asked, what is to be said of the thousand and one remedies which have been recommended from time to time, as having a special influence? What, among others, must be said of bromide of potassium, and of certain preparations of zinc, copper, and silver? What of strychnia, belladonna, conium, digitalis, indigo, cotyledon umbilicus, selinum palustre, poudre de Neufchâtel, compression of the carotids, tracheotomy, cauterization of the larynx and other parts?

“About fourteen months ago,” wrote Sir Charles Locock in 1853,* “I was applied to by the parents of a lady who had hysterical epilepsy for nine years, and had tried *all* the remedies that could be thought of by various medical men (myself among the number) without effect. This patient began to take *bromide of potassium* last March twelvemonth, having just passed one of her menstrual periods, in which she had two attacks. She took ten grains three times a day for three months; then the same dose for a fortnight previous to each menstrual period; and for the last three or four months, she has taken them for only a week before menstruation. The result has been that she has not had an attack during the whole of this period. I have only tried the remedy in fourteen or

* ‘Med. Times and Gaz.,’ 23d May, 1853.

fifteen cases, and it has only failed in one, and in that one the patient had fits, not only at the time of menstruation, but also in the intervals." In using bromide of potassium in these cases, Sir Charles Locock's object was to calm an erotic disposition, which attended and aggravated the epileptic symptoms, and this end may have been answered—in all probability, this end was answered. But this is not the only way in which this remedy acts beneficially; on the contrary, I can testify, after repeated trials, that the bromide is often a very valuable remedy in cases where there is not the slightest sign of an erotic disposition. I can testify, indeed, that this remedy has proved more or less serviceable in cases the most dissimilar in character—so serviceable that the name of Sir Charles Locock ought to be remembered with gratitude by every epileptic, and by many suffering from other forms of convulsive disorder. How to explain the *modus operandi* of this medicine is no very easy matter; but I am inclined to think that this, in part at least, is, by an alterative action upon the blood, analogous to that of iodide of potassium and common salt—an action by which, possibly, the blood may be kept free from compounds analogous to uric acid. And this I do, because for a long time I have found decided benefit from occasional doses of a draught containing bicarbonate of potass, with or without a couple of grains of iodide of potassium, and a drop or two of tincture of colchicum, or wine of white hellebore. At any rate, the alkaline character of the compound would seem to be necessary in some cases; for on looking over about thirty cases in which I tried bromide of iron and bromide of potassium, month by month alternately, I find that the latter preparation seemed to exert a more beneficial influence than the former.

The present fancy for *oxide of zinc*, as a remedy for epilepsy, has been caught from the work of M. Herpin,*

* 'Du Prognostic et du Traitement curatif de l'Epilepsie,' 8vo, Paris, 1852.

a work which is chiefly devoted to the purpose of showing that many cases of epilepsy may be cured by the vigorous and persevering use of this remedy.*

In this work, M. Herpin relates thirty-eight cases of epilepsy and epileptiform disease, in nearly all of which he gave oxide of zinc; but, as I have shown at length in the second edition of the present work (pp. 183-190), the favorable opinion of this physician as to the power of this medicine is by no means borne out by an analysis of these cases. It would appear, moreover, that M. Herpin himself has become less confident than he was when he wrote the work in question; for he has written subsequently, "que l'oxyde de zinc, ne cessant point d'être

* The doses used by M. Herpin are very large, or very soon they become so. In the case of an adult, 3 grammes of the oxide are mixed with 4 grammes of powdered sugar, and divided into twenty doses, of which one is to be taken three times a day. These twenty doses serve for the first week. After this the quantity of the oxide is increased every week by the addition of 1 gramme (about 15 grains); and in this way, if the patient persevere, he will take 52 grammes in eight weeks, 132 grammes in fourteen weeks, and 327 grammes in about six months. In the case of an infant, the quantity given in the first week is 0.25 gramme, and the addition made for each succeeding week is 0.25 gramme; so that 5.25 grammes will be taken in six weeks, 23 grammes in three months, and 68 grammes in six months. Sometimes, in consequence of the stomach or bowels being a little rebellious, it was found desirable to omit the dose taken in the morning, and once or twice it was necessary to go on for longer than a week before beginning to increase the dose; but these cases were exceptional, and, when they did occur, relief was often obtained at once by giving the oxide in the form of a pill, instead of in the form of a powder. And, lastly, it is a fundamental rule in this plan of treatment to persevere in the use of the remedy, and, in as short a time as possible after the cessation of the fits, to give a larger quantity of the oxide than had been given previously. This is to prevent relapse. Thus, if two months had been spent in the treatment, and 45 grammes of the zinc had been given already, it would be necessary to go on for another month, and, at increased doses, to give at least 100 grammes before giving up the treatment; or, if the zinc had been given for three months, and as much as 125 grammes taken before the attacks yielded, it would be necessary to go on for three months longer, and not to give up the treatment until at least 300 grammes had been taken.

convenable pour les enfans et les vieillards, échoue très-souvent chez les adultes." M. Delasiauve, who quotes these words in his admirable treatise on epilepsy,* tells us that one reason for this change of opinion was the absolute failure of an experiment at the Bicêtre, in which one of the physicians of the establishment, M. Moreau, treated eleven adult epileptics in every particular after M. Herpin's method. M. Delasiauve also tells us that M. Herpin now gives the preference to ammonio-sulphate of copper in the treatment of epilepsy in adults; and I might argue that his faith is somewhat shaken in this remedy, for I have recently had more than one patient, who, under M. Herpin's advice, had been ordered to leave off the copper after a short time, and to take a vegetable simple, of which I shall have to speak presently. Nor can I speak favorably as to the results of the trials, nine in number, in which I gave oxide of zinc, after M. Herpin's method; and my experience in this respect is in accordance with the more extended experience of my friend and colleague, Dr. Marcet. At the same time I am by no means prepared to say that zinc is of no value in epilepsy. On the contrary, I can believe that this medicine may be of considerable value in certain cases, and in moderate doses; and this belief is not a little strengthened by the beneficial results which Dr. Marcet has recently found to attend the use of oxide of zinc in many forms of nervous excitability. Of the other preparations of zinc it is not necessary to speak, for there is every reason to believe that their action for good or evil is analogous to that of the oxide.

It is not easy to obtain any conclusive evidence of the value of the *ammonio-sulphate of copper* in epilepsy. Speaking of the cases recorded in his published work, M.

* 'Traité de l'Epilepsie,' 8vo, Paris, 1854.

Herpin says that, including relapses, he obtained eighteen cures in fourteen patients; but when these cures are fairly analysed, they do not turn out to be a whit more satisfactory than those which he ascribes to oxide of zinc. Nor do I know of any evidence in favour of this salt of copper which is thoroughly satisfactory. I have never given the medicine a fair trial, and therefore I have little right to express an opinion; but this I may say, that I have met with several patients who had taken it previously, and who have said that they felt more nervous while taking it, and that no beneficial change was wrought by it upon the fits.

Nor do I know of any more satisfactory evidence in favour of *nitrate of silver*. I know this, however, that it is no uncommon thing to meet with patients whose skin had been tinged of a dismal gray colour, and whose fits had been worse rather than better during the time they were taking the medicine.

Strychnia was a very favorite remedy with the late Dr. Marshall Hall, but the dose given was attenuated to such a degree as to render it somewhat difficult to believe that much good came of it. Dr. Hall allows, indeed, that harm is done if the dose be sufficient to produce the physiological effects of the drug. And, certainly, it is not very likely that strychnia will prove to be a valuable remedy in epilepsy, if its *modus operandi* be that which Dr. Harley declares it to be, and if the physiology of muscular motion and the pathology of epilepsy have to be read in accordance with the premises.

Belladonna—a remedy recommended by Stoerek, and used extensively by MM. Debreyne and Bretonneau—has been again brought into notice by M. Trousseau during the last few years. M. Trousseau says that he has employed this remedy for twelve years, and that he has always had under treatment from eight to ten patients. He

says, further, that of 150 persons so treated, twenty have been cured, and that M. Blache, who employed belladonna in a large private practice, has met with a like proportion of successes and failures. It is a fair question, however, whether 13 per cent. of successes (which may possibly, in part at least, be explained in a different way) can be regarded as sufficiently conclusive evidence in favour of the remedy; and this the more, as other practitioners, M. Delasiauve among the number,* have been less successful. Judging from what I have seen myself, my impression is that belladonna is of very doubtful value in the majority of cases.

Nor is a more favorable conclusion to be drawn respecting the value of *coniium* in epilepsy. I have tried this remedy in several cases in small and also in full doses; but the result was no more satisfactory than that which had been already arrived at by M. Schroeder van der Kolk.†

Another remedy upon which stress has been laid is *digitalis*,‡ and Dr. C. J. B. Williams has added the weight of his authority in its favour. Dr. Williams looks upon palpitation or over-action of the heart as a very important cause of the fit, and *digitalis* is one of the remedies (hydrocyanic acid is another) to which he trusts for tranquillising the circulation, though at the same time he allows that a permanent cure is obtained most readily by the tonic class of medicines. But, if the previous reading of the phenomena of epilepsy be correct, it is not with palpitation or over-action of the heart that we have to do when the paroxysm is imminent, and most assuredly there is nothing in the shape of unequivocal success resulting from the employment either of *digitalis* or of hydrocyanic acid

* *Op. cit.*, p. 370.

† *Op. cit.*, p. 130.

‡ 'Med. Times and Gaz.,' 14th Nov., 1846.

which would lead us to think that this reading has been erroneous.

Indigo has often been given as a specific, particularly in Germany, and if it has not done any good it is not because it has not been given in sufficient doses. In some cases, indeed, an ounce or more has been given in the course of the day, and for many months in succession. But little or nothing, however, is heard of this remedy now, and I believe that there are few of those who have tried it who will be surprised that Dr. Pereira should say, "I have tried it in a considerable number of cases in the London Hospital, but without deriving the least benefit from it.*"

With regard to *cotyledon umbilicus* there would seem to be little in experience, and less in the simple itself, to warrant any hope, except that which may arise from the exercise of the imagination of the patient. It is true that this remedy was tried by the late Dr. Graves, of Dublin, and that this most trustworthy physician speaks somewhat in its favour;† but it is also true that it failed in three out of the six cases in which it was tried, that it did doubtful service in the fourth, and that a part of the good which seems to have been done in the remaining two cases may be fairly ascribed to the aid of imagination and to the leaving off of some less innocent drug.

Selinum palustre—a simple without a place in Pereira, but with a certain reputation in Switzerland—is one of the four principal remedies to which M. Herpin has pinned his faith. Nay, it is one of the four to which precedence is given in the following "ordre de mérite," namely, selinum palustre, ammonio-sulphate of copper, oxide of zinc, valerian. Now, selinum palustre is an umbelliferous plant, of which several grammes may be taken

* 'Materia Medica,' 4th edition, vol. iii, p. 331.

† 'Dublin Quart. Journ. of Med.,' Nov., 1852.

at once; and in questioning three or four patients who have taken it, the answer was that they were warmed and comforted by it. In other words, its action seems to have been that of a feeble stimulant. It is difficult, however, to understand the grounds upon which M. Herpin attaches so much value to this medicine. He says that five of his "cures" were due to its use, but of these very five the amendment was more nominal than real in three, the fits recurring in a short time, and in the remaining two the only approach to a cure was in the fits being separated by a little wider interval. Indeed, this very loose way of speaking of these cases as *cures* must make us very cautious in accepting what M. Herpin says respecting the *cures*, which he believes to have been brought about by the use of zinc or copper, or other remedies of less equivocal activity.

Poudre de Neufchâtel is a remedy which has some credit in Switzerland, and which has lately been brought prominently into notice by having been given in some of the cases recorded by M. Herpin. I had lately under my care a lady who took this preparation under M. Herpin's directions for several months. And what is this *Poudre de Neufchâtel*? It is nothing but the powder of *taupe grillée*, or, in plain English, fried mole. It is, indeed, a relic of the days when animal remains of a more objectionable character, fried or otherwise, were offered to the unhappy epileptic. In justice to M. Herpin, however, it must be said that he does not *believe* in this out of the way remedy, and that he only tries it when other remedies have failed.

Compression of the carotids has been recommended on the supposition that a main cause of epilepsy is an afflux of blood to the head. Among recent writers Dr. Romberg is one who regards it as an effectual prophylactic where there is warning, and sufficient time to profit by the warning. Dr. Sieveking, also, in his able work on

epilepsy and epileptiform seizures,* says that "it certainly deserves an extensive trial . . . and that it is probable that the cephalalgia and somnolency, which the patients so frequently complain of as distressing symptoms following the attacks, may be entirely prevented by it." As yet, however, the evidence in favour of this measure is both scanty and inconclusive, and it might be easily frittered away by any one who is at all disposed to be sceptical.

And, certainly, it must be allowed that *tracheotomy* does not realise all the original hopes of Dr. Marshall Hall. It does not prevent convulsion. It does not, with certainty, make the convulsion slighter. It does not prevent danger, for (as I have shown elsewhere)† three of the few patients upon whom the operation has been performed have died either in the fit or in connexion with the fit, and of the three the artificial opening was free from all obstruction, at least in one. The first two cases, indeed, were calculated to damp the zeal of any one less sanguine than Dr. Marshall Hall.

CASE I.—The patient in this case was a boatman, æt. 24, who had been epileptic for seven or eight years, and whose fits were frequent and severe. The operation was performed by the late Mr. Cane, of Uxbridge, during a fit of "asphyxial-coma" that had lasted nineteen hours. The relief was immediate, and for some months afterwards no fit had happened; but, unfortunately for the credit of the operation, the patient, not liking the gurgling noises and the voicelessness consequent upon breathing through the wound, *had chosen to wear the tube with its opening carefully corked-up*. This information I had from Mr. Cane himself. What the end was is not known, for soon afterwards the patient was discharged from his employment for drunkenness, and lost sight of.

CASE II.—The patient in this case was a stout, thickset, muscular female, æt. 36, the daughter of an epileptic father, and herself epileptic for twenty-four years. Her complexion was ruined by the former use of nitrate of silver.

* 'Epilepsy and Epileptiform Seizures,' post 8vo, London, p. 195, 1858.

† 'Lancet,' 14th May, 1853.

The operation was performed by Mr. Anderson, of York Place, Baker Street, and the tube was worn until her death, *which happened in a fit*, about twenty months afterwards. After the operation the fits continued as before—possibly a little less frequently and severely, but decidedly of the same character. Her health and spirits are also said to have undergone some slight improvement, and she lost a numbness in the right arm which had previously distressed her, but those who knew her best doubt the existence of any appreciable change of this kind until about two or three months before her death—until, that is to say, sixteen months after the operation. The following notes of the final seizure were given me by Mr. Anderson. “8 a.m.—Had been up and dressed; heard to fall heavily. A woman removed the inner tube from the trachea, as she was in a fit apparently more severe than usual. She ‘snorted loudly;’ nails of a deeper colour. She was placed on the bed, as the woman thought she would recover as usual.” The woman here referred to tells me that the patient was black in the face and violently convulsed, and that death must have taken place within ten minutes.

In a word, it may be questioned whether the supposed benefits of tracheotomy are sufficient to counterbalance the associated inconveniences and dangers of the operation, even where (what rarely happens) the asphyxial symptoms are in any important degree dependent upon spasmodic closure of the glottis.

As to the value of *cauterizing the larynx*, it is less easy to come to a conclusion. Dr. Brown-Séquard says* that a third of his epileptic guinea pigs were cured by this mode of treatment, and that all the rest, with the exception of two or three, were benefited by it; and he suggests its adoption in the treatment of epilepsy. A little later, Dr. E. Watson, of Glasgow,† recommended the same mode of treatment, and related three cases—two by himself, and one by Dr. Horace Green, of New York—in which good would seem to have resulted from it. Dr. Brown-Séquard also lays stress upon the importance of using *cauterization in other parts*, as in the nape of the neck, and especially in the neighbourhood in which the

* ‘Philadelphia Medical Examiner,’ April, 1853.

† ‘On the Topical Medication of the Larynx,’ 8vo, London, 1854.

aura originates, and he prefers the moxa or hot iron to milder measures. This practice, he tells us, proved very beneficial in the case of his poor guinea pigs. In a word, the last-named physician, furnishes us with some additional facts in favour of the value of *counter-irritants* as a means of cure in epilepsy; and not only so, but he gives a hint which may prove to be of some practical value, by pointing out the larynx, and the locality in which the aura originates, as sites in which counter-irritation may be especially serviceable. Now, the verdict of past experience is very much in favour of counter-irritants, and this verdict may recommend itself to the judgment, though on different grounds to those on which it has been given. After what has been said, indeed, it is not easy to believe that these measures do good by withdrawing some morbid irritability from a vital organ; but it is possible that they may do good by the inflammation which they excite. It is possible that they may do this, because the fits of epilepsy are not unfrequently suspended for the time by inflammation arising from injury inflicted during the fit, or in some other way, as well as by idiopathic inflammation, and because all tremulous, convulsive, and spasmodic disorders are suspended under similar circumstances. Upon the occurrence of true meningitis, for example, there is an end to the trembling of delirium tremens; upon the establishment of the cutaneous inflammation of smallpox, there is an end to the convulsions which may have attended upon the initial period of collapse; and if inflammation of the lungs be established in hooping-cough, there is an end, for the time, to the laryngeal spasm. It is possible, therefore, and it is quite in accordance with the premises, that counter-irritants, so-called, may be beneficial in epilepsy. It is possible, also, that these measures may do good, because the discharge resulting from them may rid the blood of some

impurity. But at the same time I must say that my own experience in this matter is at once scanty and unsatisfactory.

“There is,”* said the late Dr. Marshall Hall, “No royal road to the cure of epilepsy. The idea of a remedy for the disease is unphilosophical, and the treatment should consist in a well-advised plan, embracing every means of good, and avoiding every means of harm.” And, without doubt, there is much truth and wisdom in this remark. At the same time I cannot help thinking that there is a main road to the cure of epilepsy, along which we have yet to travel, and that some remedy has yet to be applied, without which the best-advised general plan of treatment will, at the best, prove unsatisfactory.

Reflecting upon the physiological and pathological premises, the great desideratum in a case of epilepsy would seem to be a more vigorous action of the nervous centres, for, according to these premises, convulsion itself is the consequence of a failure in this action; and hence it may be supposed that the remedy which has yet to be applied is one which will have some special power of rousing and sustaining the action which is deficient. Reflecting upon what has already been said respecting the beneficial influence of cod-liver oil in epilepsy, and upon the possibility that this remedy may do good by furnishing one of the materials, namely, the fat, which is necessary to the full nutrition of the fatty nervous tissue, it may be supposed that the remedy which has yet to be applied is one which will supply this kind of food to the nervous system. But is this all? Is there any material besides fat or oil which may be wanted to secure the healthy nutrition of the nervous system. These questions suggest themselves naturally, for however mysterious the properties of the nervous system, this much is plain enough—that the

* ‘Lancet,’ 30th October, 1848.

nervous tissue cannot exist independently of proper nutrition, and that any means which will merely rouse its action, without at the same time providing for its nutrition, must in the end do harm, and not good. Now, on examining the chemical constitution of nervous tissue, it is evident that phosphorus is a very important ingredient. It is evident, too, that the amount of fat and phosphorus has some relation to the activity of the nervous centres, for both these ingredients increase from infancy to adult age, and decrease afterwards as the influence of advancing age tells upon the system. It is also a most significant fact that the proportions of fat and phosphorus approximate very closely in infants and idiots. The mean of six analyses of the human brain by M. l'Heretier,* who has investigated the subject very carefully, is given in the following table:

	Infants.	Youths.	Adults.	Aged persons.	Idiots.
Fat	3.45	5.30	6.10	4.32	5.00
Phosphorus.....	0.80	1.65	1.80	1.09	0.85
Albumen.....	7.00	10.20	9.40	8.65	8.40
Osmazone and salts. ...	5.96	8.59	10.19	12.18	14.82
Water.....	82.79	74.26	72.51	73.65	70.93

The ingredients of the spinal cord and of the nerves are substantially the same as those of the brain, and there is reason to believe that the proportions of fat and phosphorus vary in the same manner at different periods of life. Here, then, are some very curious and striking facts. Here is nearly 2 per cent. of phosphorus in the brain of

* 'Simon's Animal Chemistry,' by G. E. Day, M.D., Sydenham Soc. edit., 8vo, London, 1846, vol. ii, p. 427.

adults, and in infants and idiots considerably under 1 per cent. Here is, that is to say, little more than half the proper proportion in a state between which and the worst forms of epilepsy there is a somewhat close relationship. The facts, indeed, are well calculated to suggest the question whether phosphorus may not be as necessary as fat to the proper nutrition of a weak nervous system—as necessary as iron where there is a deficiency of red corpuscles in the blood; and this question, once put, would seem to require an answer in the affirmative. “In *small doses*,” says Dr. Pereira, “phosphorus excites the nervous, vascular, and secretory organs. It creates an agreeable feeling of warmth in the epigastrium, increases the fulness and frequency of the pulse, augments the heat of the skin, heightens the mental activity and the muscular powers, and operates as a powerful sudorific and diuretic.”* In large doses, phosphorus, without doubt, is a caustic poison; in proper doses it produces the very changes which are necessary in a case of epilepsy. In proper doses, and under the eye of a medical man, it is quite innocent of harm, and it may be productive of much good.

This inference is that which may be drawn from what has been said; and this inference, so far as I can see, is not contradicted by experience. Given in the large doses in which phosphorus has been given in a few cases already on record, the good resulting may have been doubtful—very doubtful; but this experience is nothing to the point, for there is no reasoning in any case as to the effects of medicinal doses from the effects of poisonous doses. Given in medicinal doses, I have seen enough to know that this remedy may be given, not only without harm, but with the unmistakable promise of real

* ‘Elements of Materia Medica and Therapeutics,’ 4th edition, 8vo, London, vol. i, p. 343.

and substantial good. As yet my experience is very limited, for it is only the other day that the idea of using oil and phosphorus as nervine tonics occurred to me; but, as I have just said, I have already seen enough to justify me in saying what I have just said. I have also seen what leads me to expect that the promise of good from this mode of treatment is not confined to cases of ordinary epilepsy, but that it includes other cases in which the action of the nervous centres, one or more, is defective—namely, two cases of hysterical paraplegia, one case of weakness of the arm and leg on one side following some choreic symptoms, and one case of melancholia. The form in which I have given the phosphorus is the phosphorated oil of the Prussian Pharmacopœia—a preparation which is made by dissolving twelve grains of phosphorus in one ounce of almond oil by the aid of warm water. About four grains of the phosphorus are taken up, and the usual dose is from five to ten minims. I have given this phosphorated oil along with cod-liver oil, in a little orange wine, twice or thrice a day, the formula being—

R Olei Phosphorati (Ph. Borussicæ), ℥v—x;
Olei Morrhuæ, ℥ii—iv. M.

I am also inclined to think that the new electric band of M. Pulvermacher may be an important help towards recovery in the case of some epileptics. The experiments of M. Matteucci, which were related in a former chapter (pp. 75, 76), show that the tetanus of strychnia may be prevented or suspended by passing a continuous galvanic current along the spinal cord, and also that this nervous centre may be “irritated” in any way during the passage of the current without the production of a single convulsive movement in the muscles to which the spinal nerves are distributed; and, showing this, they suggest the idea that a continuous current along the spinal

cord or medulla oblongata, especially the latter, may have some power in preventing convulsion. I have had this idea in my mind for some time, but I did not know how to apply it. I did not know how to get a *continuous* current of sufficient strength, lasting for a sufficient time, and being at once perfectly portable and easily manageable. M. Pulvermacher, however, has convinced me that I may get all I want from the electric band which he has just invented, and he has also promised to adapt this band to the purpose in view. There are, I doubt not, many difficulties in the way of carrying out this idea practically, and these may not be overcome at once, but I am sanguine enough to believe that they will be overcome in the end, and that then the continuous current of electricity will prove to be a real help to recovery—a help not to be used on all occasions and indiscriminately, but to be resorted to now and then, with a view to tide over known times of danger. There is also some reason to think that, in some instances, the placing of one of M. Pulvermacher's electric bands along the course of an aura might be of some use in preventing the fit.

— With regard to the treatment of the fit itself little need be said. As a rule, it will only be necessary to take care that the patient does not injure himself; that the head is not allowed to hang too low; and that any neck-lace or neckerchief be loosened. The latter precaution is of greater importance than it may seem to some, for the neck will often swell in the most remarkable manner from the accumulation of blood in the veins, and thus any ligature around the neck may increase the risk of cerebral hæmorrhage by preventing the return of blood from the brain. If salt be at hand, a little may be put in the mouth; if water be within reach, a little may be sprinkled on the face—though the advantages of the latter expedient are

scarcely sufficient to compensate for the disadvantages and possible risks arising from wetted garments. In ordinary epilepsy it can scarcely ever be necessary to have recourse to chloroform, as it may be in some prolonged epileptiform affections; but if the convulsive stage should happen to be unusually prolonged, no remedy would seem to be more appropriate and effectual than chloroform.

CHAPTER II.

ON CONVULSIVE AFFECTIONS WHICH ARE CHARACTERISED BY TREMOR.

THE first category of convulsive affections, in which the muscular disturbance takes the form of *tremor*, includes the tremors of delicate and aged persons, of paralysis agitans, of delirium tremens, the rigors and subsultus of fevers, and the shakings of slow mercurial poisoning.

1. *The general history of convulsive affections which are characterised by tremor.*

The subjects of common trembling have a certain delicacy of constitution which cannot be overlooked; and if not women, they may be said to have a feminine habit of body and mind. It is evident, also, that they are altogether *unnerved* during the paroxysm, and that their thoughts and words are as little under the control of the will as the muscles. At this time, also, the circulation is greatly depressed, and the pulse does not recover until the paroxysm is over.

Those who tremble from old age present unequivocal marks of decrepitude—the listless wish, the blanched locks, the fireless countenance, the shrunken limb, the feeble pulse, the frigid hand. Every faculty, mental and bodily, has given way under the wear* and tear of life; and during the actual attack of trembling, the pulse fails, and the mind loses the small amount of power which had remained in it.

In paralysis agitans, the occasional and partial tremors of old age have become permanent and general, and in

extreme cases they scarcely cease during sleep. If walking continue to be practicable, it is performed by stepping hastily and tremblingly upon the toes and forepart of the foot, and there is constant danger of falling from the way in which the head and body are bowed forward. And in the end, the hands, feet, and tongue fail to fulfil their proper offices, the outlets of the body are uncontrolled by the will, and the patient has to be fed and treated as a child. The paleness and chilliness, and the decided relief afforded by wine, reveal the real state of the circulation in paralysis agitans.

The trembling of delirium tremens, which is only the aggravated form of that trembling from which drunkards habitually suffer when sober, is primarily confined to the hands and limbs. It is associated with extreme fidgetiness and restlessness, and in some instances with convulsive startings. The accompanying phenomena are very marked. The hands and feet are cold, the pulse is quick and weak, the respiration is disturbed by sighs and pauses, the tongue is moist and creamy, and every movement is attended by profuse perspiration. The mind is confused, irritable, despondent, anxious, and tortured with gloomy forebodings or spectral delusions. Everything and everybody is an object of mistrust, or fear, or dread. Sleep has vanished, but the state of wakefulness is not attended by headache. In some instances there are fits of unruliness, and sometimes of fierceness, but these are easily subdued by ordinary firmness on the part of the friends and attendants. As the malady progresses, the tremors acquire the character of subsultus, the convulsive startings become more common, the coldness of the hands and feet extends to the rest of the limbs, or even to the trunk and head, the skin is more than ever drenched in perspiration, the delirium becomes low and muttering, and by quick degrees the patient sinks into a state of

mortal collapse. Such is the usual course where the disease ends fatally. In some instances, however, the ordinary symptoms may change into, or alternate with, those belonging to meningitis, in which case the pulse rallies, the skin and tongue become dry, and, as the headache and other symptoms of inflammation make their appearance, *the tremor ceases*.

The tremors of fever and their accompaniments are familiar to all. The rigors or shudderings at the commencement are accompanied by chilliness and paleness of the skin, by blueness of the nails and *cutis anserina*, by feebleness and frequency of the pulse, by sickness and vomiting, by indescribable feelings of languor, feebleness, oppression, by aching pains in the head, back, and limbs. The subsultus of the final stage is attended by a dusky and cool skin, a fluttering pulse, a fainting and almost silent heart, a short and sighing respiration, and by the utmost exhaustion and prostration both of body and mind.

In slow mercurial poisoning the muscles are very disobedient to the will, and all co-ordinated movements are clumsily performed. The skin is gray and dry, the pulse weak and subject to great fluctuation upon change of posture, the appetite capricious or wanting, and the whole mental and bodily strength greatly impaired. Moreover, paralysis and premature old age is the end of the unchecked disorder.

2. *The pathology of the convulsive affections which are characterised by tremor.*

§ I.

The state of the circulation and respiration in these several affections is sufficiently obvious. In an attack of common trembling there is no doubt that both these functions are

much depressed, for this is evident, as well in the paleness and chilliness of the person trembling as in the decided relief afforded by wine.

In delirium tremens, the perspiring skin, the cold hand, the quick and compressible or fluttering pulse, the treatment demanded, are all significant and unmistakeable facts. It is evident, also, that the trembling is actually connected with this state of things, for if the dry skin and excited pulse of true meningitis make their appearance, the trembling is at an end. On the other hand, an argument to the same effect is to be found in the fact that tremor is exaggerated into subsultus, or even into convulsion, as the heart and pulse fail in the course of the disorder.

Rigor, moreover, is coincident with a sense of coldness, a feeble pulse, a sunken countenance, a corrugated skin, and subsultus with a circulation which is faltering on the very verge of stagnation. Nor is this coincidence accidental. It is not—because the rigor disappears as the pulse and warmth return; it is not—because the subsultus may be checked for the time by the use of wine.

And in mercurial tremor an inference as to the real state of the circulation may be drawn from the general practice prevailing among the subjects of this disorder of resorting to gin and similar stimulants to make themselves steady.

— In a word, the state of the circulation in the different forms of tremor is one of under-action, and not one of over-action; and in this respect, therefore, the pathology of tremor agrees with the pathology of epilepsy, and with the physiology of muscular motion, as set forth in the premises.

§ II.

In any attack of trembling the mental faculties are all

unstrung; and in the extremest forms of this trouble, as in paralysis agitans, they have altogether succumbed before the inroads of age or disease, and the patient lives only to fear and eat.

In delirium tremens the mental state is passive in every point of view. The patient is in a helpless state of fear and dread, and at every new impulse his thoughts course timidly from one object of fear to another. He lies unmanned, as it were, before some dim phantom of evil, and when a fierce delirium takes the place of the delirium tremens, and when other symptoms betoken the existence of active determination or inflammation within the skull, then the affection ceases to be delirium *tremens*, for the trembling has disappeared.

In the initial rigors of fever, the mental state is one of dejection, languor, stupor; in subsultus, it is one of wandering silliness or of drowsiness not far remote from coma.

In slow mercurial poisoning the failure of the mental power keeps pace with the failure of the bodily strength, and the condition is one of premature old age.

— In these several forms of tremor, therefore, this condition of the nervous system, as reflected in the state of the mind, is one of comparative inactivity. Nor is it easy to suppose that the condition of the cerebral hemispheres is different from that of any other part of the nervous system, for if a due supply of red blood be, as it undoubtedly is, necessary to the exercise of the different nervous functions, then it follows that the medulla oblongata, the spinal cord, and every other nervous centre, as well as the cerebral hemispheres, must be in a state of comparative inaction during the time of the trembling. With this state of circulation, indeed, it is impossible to suppose that there can be any increased supply of nervous influence to the muscles during trembling.

Nor is it an objection to this view of the matter that trembling ceases during sleep. If inactivity of the brain be a cause, it might seem that trembling should not cease during sleep; but this is by no means a necessary consequence. On the contrary, it would rather seem that the muscles should be quiet at this time because the brain is quiet. It may be supposed that each thought and feeling involves an equivalent expenditure of cerebral nervous influence, and that there is a cessation of this expenditure during sleep. In other words, it may be supposed that each thought and feeling involves an equivalent expenditure of the *nerve-currents* which go to form so large a part of nervous influence, and that there is a cessation of this expenditure of cerebral nerve-current during sleep. It may be supposed, too, that the cessation of the expenditure of cerebral nerve-current during sleep will put a stop to those movements in the nerve-current of the motor nerves which are necessary to re-establish the electric equilibrium which has been disturbed by the expenditure of cerebral nerve-current. And, lastly, it may be supposed that this cessation of movement in the nerve-current of the motor nerve will suspend that development of induced currents within the muscle which attends upon any movement in the nerve-current in the motor nerve, and which, according to the premises, is the cause of muscular contraction. In a word, there is almost the same necessity that trembling should cease during sleep, as that it should cease after decapitation; for, absolved from the promptings of a waking brain, and left to themselves, how can the muscles be otherwise than quiet? It may be supposed, also, that the warmth of the bed will tend to check the disposition to tremble by increasing the activity of the circulation, and that the recumbent posture may help towards the same end by putting an end to those constant movements

in the nerve-current of the motor nerves which are necessary to that continual development of induced currents by which the muscles are made to contract, as they have to contract when the body is kept in the erect position.

3. The treatment of the convulsive affections which are characterised by tremor.

The few remarks which have to be made under this head are altogether in harmony with what has been already said upon the treatment of ordinary epilepsy.

It is evident, in the first place, that abstinence does not form a part of this treatment. A person who trembles habitually, whether young or old, trembles more before a good meal than after it. Food, moreover, is of more importance than medicine in the treatment of ordinary delirium tremens* and subsultus, and in mercurial trembling the muscular disturbance is always worse during fasting.

It is evident, also, that a glass of wine will tranquillise an ordinary attack of trembling. Wine, moreover, will steady the hand in delirium tremens, and, if the symptoms are not beyond the reach of remedies, it will calm the

* Within the last few months I have had four cases of delirium tremens in the Westminster Hospital, in which the treatment was by hot beef-tea and belladonna. A teacupful of very hot beef-tea, with bread sopped in it, was given regularly every hour, except during sleep; and every two or three hours, for the first couple of days, a draught containing ten drops of the tincture of belladonna. The hot beef-tea was the only stimulant given. In each case the patient slept a good deal during the first night, and the appetite for meat had returned on the fourth day. In each case the belladonna produced dilatation of the pupil and dryness of the throat. This drug was given chiefly on the supposition that it might tend to counteract the dismal character of the thoughts, (for, in full doses, as is well known, belladonna produces a gay and cheerful delirium,) and this end may have been somewhat answered; but I am disposed to think that the part played by the drug was subordinate to that which was played by the hot beef-tea.

fainting heart and still the twitching wrist of the last stage of fever. In like manner, coffee rather than tea would seem to be a desirable remedy in all these cases.

In the case in which the trembling arises from slow mercurial poisoning, it may be necessary to put in practice the elegant treatment recommended by M. Melsens,* and endeavour to liberate the metal from the poisoned tissues by favouring the formation of a new and soluble compound between it and iodide of potassium, which compound is afterwards eliminated from the blood by the kidneys: in the other cases of trembling the remedies which would seem to be most admissible are of a stimulating and tonic character.

— As in epilepsy, therefore, so in the different forms of tremor, the rational treatment would seem to be that which avoids every cause of depression and exhaustion, which seeks after every means of increasing and establishing the strength, and which trusts to stimulants in any emergency.

* 'British and Foreign Medico-Chirurgical Review,' Jan. and April, 1853.

CHAPTER III.

ON THE CONVULSIVE AFFECTIONS WHICH ARE CHARACTERISED BY SIMPLE CONVULSION.

THE second category of convulsive affections, or that in which *convulsion* is the distinctive feature of the muscular disturbance, may be divided (as I have already said) into two sections, by the absence or presence of consciousness during the convulsion. Where the consciousness is present, the convulsion may be called *simple*; where the consciousness is absent, it is called *epileptiform*. *Simple convulsion*, with which I am now concerned, is that which is met with in the state called hysteria, in chorea, and in those strange affections which are more or less akin to chorea, as the dance of St. Vitus and St. John, tarantism, and other affections in some degree analogous to these.

I. *The general history of convulsive affections which are characterised by simple convulsion.*

1. They who suffer from what is ordinarily called *hysteric convulsion*, belong almost exclusively to the female sex, and not only so, but they possess, in an aggravated degree, the peculiar weaknesses of this sex. They are never *strong*, and, in the majority of instances, a delicate constitution has been enfeebled by disease or by some faulty habit of life.

For the most part, they are undecided, irresolute, fickle, purposeless, yielding easily and almost helplessly to every impulse either from within or from without, and scarcely ever capable of anything like sustained action. They do what they ought not to do, and they leave undone what

they ought to do; and their excuse is that they cannot help it. With them *will* is little more than an empty name, and so much are they the creatures of feeling that a small matter serves to make them melt into tears or burst into laughter. The temper, also, is as little under control as the feelings, and impatience, perverseness, obstinacy, and anger, are no uncommon symptoms. There is no lack of ideas; but, as a rule, these are allowed to take their own course with little check from the reason; and hence fancies and whims of all kinds in endless succession, or, what is worse, some one whim or fancy in possession of the mind, and the reason unable to eject it. Not unfrequently, also, there is a disposition to exaggeration and deceit which must betoken some bluntness in the moral sense if some allowance have not to be made on the ground of an imagination which cannot always stoop low enough to perceive the line which separates facts from fancies.

These mental peculiarities are all written with a certain degree of plainness upon the countenance. There is no want of brightness in the eye, no sluggishness of the pupil, no marks of "slowness" as in many epileptics. On the contrary, there is a brisk, unsteady expression, which shows that the mental error is on the side, not of dulness, but of sensitiveness.

Among other evidences of an undue sensitiveness, pain and fidgetiness take a prominent place. Pain is a very common occurrence, or, at any rate, a very common complaint. It is pain in the head, pain under the left mamma, pain anywhere or everywhere in turn, seldom nowhere. Fidgetiness, also, is a common occurrence, particularly in the legs, and, in some instances, this is sufficient to make it impossible to remain in one place for any length of time. Or this want of control over the muscular system may be seen in the way a sudden noise will cause the patient to tremble or start. As a rule, also, the bodily

strength is far from perfect. Sometimes, it may be, there is more strength than would appear at first sight, for any kind of exertion may be shunned from sheer idleness; but very generally there is an actual want of strength—a want which is shown both in the inability for prolonged exertion, and in the slowness with which the system recovers from fatigue.

The pulse is generally soft, quick, and much affected by changes of posture, becoming slower and falling, it may be, to a natural rate of frequency in the recumbent position, and resuming its former quickness in the erect position. The heart is readily thrown into a state of annoying and distressing palpitation, especially by any agitation of the feelings. The circulation, indeed, is wanting in healthy vigour, and as additional evidences of this fact may be mentioned as of almost constant occurrence, paleness of the skin, coldness of the hands and feet, and a disposition to chilblains even when the weather is not very cold.

The digestive functions, moreover, are carried on in a very unsatisfactory manner. Little is eaten, and the food least liked is meat; taste is capricious, or actually depraved; digestion is tedious, and often accompanied by distressing flatulence; and obstinate constipation is a frequent trouble.

And, lastly, there are most generally unmistakable signs of uterine derangement, more particularly excess, or suppression, or alteration of the monthly discharge.

Such are the principal features of the persons who suffer from hysteric convulsion.

2. The persons who suffer from *chorea* agree in many particulars with those who suffer from hysteric convulsion. They exhibit, often in an exaggerated degree, the same signs of wanting will, of halting reason, of inordinate sensitiveness. They suffer from the same timidity, the

same fretfulness, the same uncertainty and irritability of disposition and temper. If there be any marked difference, it is that they have less vivacity.

In many cases this want of vivacity is written upon the countenance in an expression of languor and vacancy; in some cases this expression may be so marked that a person suffering from chorea, if seen in a moment of quiet, may almost be mistaken for an idiot. Indeed, it may be necessary in these latter cases to make the patient get up and move about before the true nature of his malady can be detected.

In the moments of comparative quiet the muscles in chorea are far less under control than they were in hysteria, and instead of mere fidgetiness of the legs, there is a fidgety state of every muscle in the body. The features, for instance, are distorted by frequent grimaces; the tongue, always in a hurry, trips or stammers in its words, and if put out, it is in again before there has been time to look at it; the hand is with difficulty kept from tossing and jerking in a very inconvenient manner; and the foot will only consent to move in a starting, jumping kind of gait. The muscles, also, are very readily fatigued, and very slow to recover their lost power, and, in addition, they may be soft, flaccid, and wasted. There is, indeed, far more disturbance in the muscular system than there was in hysteria, and far less disturbance in the sensory nerves. At all events pain is by no means a prominent symptom.

The circulation is subject to considerable fluctuations, being always considerably affected by changes of posture, or by any excitement of the feelings; but any excitement is very slight and transient in comparison with the opposite state of depression. The pulse, most generally, is quick and weak; the heart is readily thrown into a state of palpitation; the hands are far from a comfortable state of warmth. In many cases, also, the weakness of the

circulation is further shown by paleness of the face, lips, gums, and tongue; by pastiness of the skin; by watery effusion into the subcutaneous tissue, or into the serous cavities; by murmurs in the heart and great vessels, with other signs of anæmia; or the signs of anæmia may be deepened into those of actual chlorosis. In this country, indeed, there is a connexion between rheumatism and chorea which cannot be overlooked; but this fact is not to be construed into an argument that the phenomena of fever are mixed up with those of chorea. On the contrary, chorea is essentially a feverless malady, and when it occurs in connexion with rheumatism (this will be seen presently) it is not during the occasional bouts of fever, but during the feverless overspent intervals of inaction which are habitual.

In other respects, the persons suffering from chorea resemble very closely the persons suffering from hysteria. They exhibit the same want of tone in the digestive functions. Their urine is similarly pale and copious. And if they are old enough, and of the proper sex, it will rarely happen that there is no evidence of uterine disorder.

In its ordinary form the time at which chorea is most likely to happen is between the second dentition and puberty, but adults or even old persons may be the sufferers. Statistics have also shown that boys are less liable to this affection than girls.

Among the several causes of chorea, fright is assuredly the most common; indeed, chorea may be said, almost without any figure, to be only the perpetuation of that state of startling from which all suffer for a moment under such circumstances. Blows and falls on the head, and particularly irritation about the teeth, are frequently referred to as causes, and certainly there are not a few cases on record in which the symptoms of chorea have ceased on the removal of offending teeth. On the other hand, there are

very few cases, except perhaps those occurring after the period of youth, in which the symptoms can be referred to any positive mischief in the central organs of the nervous system.

2. *The history of the paroxysm of simple convulsion.*

1. The *hysteric paroxysm* is frequently ushered in by a feeling of uneasiness in some part of the abdomen, generally in the left flank,—by flatulent distension of the bowels, with disagreeable rumblings and eructations,—by copious discharge of limpid urine,—by palpitation,—by a sense of want of breath,—by an attack of actual fainting,—or by a feeling which has received the name of *globus hystericus*. This latter feeling gives the idea of a ball in the lower part of the abdomen, which, after rolling about in that region for a little time, mounts first towards the stomach, and afterwards towards the throat, when it gives rise to repeated attempts at swallowing, accompanied by a distressing sense of choking. Then, after screaming, or laughing, or sobbing, as the case may be, the patient begins to struggle violently and to dash herself about in the most extraordinary manner, springing bolt upright from the recumbent posture, falling back again, rolling round and round, dashing her limbs about in all directions, striking her breast, grasping and pulling at her throat as if to tear away some ligature which prevented the breath from entering, tearing her hair or garments, and sometimes striking violently at the bystanders. She *struggles* violently, and with a degree of power that is not a little surprising; and this fact of struggling is plainly written upon the flushed cheek, the dilated nostril, and the set teeth.

All this time the patient is in a state approaching very closely to unconsciousness; but she certainly knows to some extent what she is about, though as a rule she will

strenuously deny it. Indeed, a threat to drench her with water, will rarely fail to bring the paroxysm to an end. The eye, moreover, is bright and twinkling, and the pupil responds readily to light—which facts are not very compatible with complete loss of consciousness during the fit.

The circulation, for the most part, remains as it was in the intervals between the paroxysms, or if it become a little excited, it is far less than might be expected in such violent struggling. The breathing, on the other hand, is generally slow, embarrassed, and frequently interrupted by sobs and hiccup.

After continuing for a time, which may vary from a few minutes to two or three hours, the paroxysm generally passes off in a burst of laughter, or a flood of tears, or an abundant eructation of air, or a copious discharge of limpid urine. It is rare, however, for the patient at once to recover her small share of equanimity, and generally she will lie for some time panting and trembling, her eyes fixed and wrapt, her limbs catching and jumping, her whole body starting and shuddering at the least noise or the gentlest touch. She will lie, indeed, in a state which is the very reverse of stupor. Sometimes she may suffer for a short time from headache, from pain in the epigastrium, from a feeling of exhaustion or extreme fatigue, or from numbness or loss of motion in one or more limbs; sometimes paroxysm may follow on paroxysm for hours, and at all times it is easy by any slight imprudence to bring on another paroxysm.

This is the common form of hysterical convulsion; but another form is described, and frequently met with, which is not distantly related to simple syncope. In this the patient sinks down suddenly, with slow and embarrassed breathing, but without any struggling or evident convulsion, and after lying for a moment, in which her face

becomes a little flushed, and her neck a little tumid, she recovers, and after a good laugh or cry, is well again.

— When these symptoms are well marked, it is not easy to confound them with those of epilepsy. In hysteric convulsion the muscular disturbance is more that of struggling than convulsion; it is sufficiently violent, but it is quite different from the convulsion of epilepsy; it is also general: in epilepsy, on the contrary, the muscular contractions are more continued, they fix the limbs rather than dash them about, and they are more marked on one side of the body than the other. In hysteric convulsion the colour of the face is natural or only a little heightened, the features are not distorted, the eyelids are closed, the eye is bright and twinkling, and its pupil is sufficiently sensitive to light, the mouth is only a little set, the lips are not covered with saliva, the neck is not twisted; in epilepsy, on the contrary, the colour of the countenance is livid, leaden, or black, the eyelid half open, the eye dull, distorted, projected, the pupil dilated and absolutely disobedient to light, the mouth dragged almost to the ear, the teeth are clenched, the lips are drawn apart and covered with froth, often with bloody froth, and the whole face is twisted round so that the chin rests upon one of the shoulders. In hysteric convulsion the breathing is slow and embarrassed, frequently considerably so, but not more than this; in epilepsy it is absolutely suspended. In hysteric convulsion the paroxysm ends in a burst of laughing or sobbing, and after this the patient is left in an excitable languid state—a state which is the very opposite of anything like stupor: in epilepsy the paroxysm ends in deep coma, or rather the deep coma of the paroxysm is continued for some time after the convulsion is at an end; and after this there is for some time a stunned and stupid state of mind, with a marked disposition to sleep. In

hysteric convulsion the consciousness is only partially suspended; in epilepsy it is absolutely extinct.

When, indeed, the characters of the two paroxysms are fully marked, there is little danger of confounding hysteric convulsion with epilepsy; but at the same time there is no lack of cases in which the characters of the two interblend in a very curious manner. True epileptic convulsion may indeed alternate with hysterical convulsion, and it not unfrequently happens, as the phenomena of epilepsy change for the better, that the loss of consciousness becomes less and less profound, as well as less and less prolonged, until the patient remembers something that may have happened during the paroxysm, and wakes up as soon as the convulsion is over. Indeed, this would seem to be the common mode of change for the better, in many, if not in the majority, of cases.

2. The paroxysm of *chorea* consists in certain tremulous movements, and in certain irregular and often comical or awkward shocks or convulsions. The muscles are rebellious, for they act most irregularly when they are required to carry out the behests of the will; they are also disobedient to that part of the nervous system whose office is to co-ordinate the action of several muscles in a common object, and hence such acts as handling, standing, walking, or speaking are ill performed, or altogether impracticable. The muscles of the face are affected first, then those of the limbs, and, lastly, those of the trunk. The upper limbs are always affected before the lower limbs, and not unfrequently the upper alone are affected; the lower limbs would never seem to be affected exclusively. The movements are more marked on one side of the body, and sometimes they are confined to one side.

In the cases ordinarily met with, the muscular disturbance is partial, often confined to the eyelids or lips, and frequently not going beyond the face and neck, and one of

the upper extremities. The paroxysms, moreover, are separated by wide intervals; they are not very distressing when the patient is left to himself, and when he does not attempt any unusual kind of exertion; and they cease during sleep. But, on the other hand, there are cases of a very different character—cases, happily, not very common—in which the muscular disturbance is so general and severe, that the sufferer cannot be kept in bed without being strapped down, and where no quiet can be got but that of death. Once seen, it is not easy to forget a scene so sad as that presented by a patient thus tossing and struggling, chafed, bruised, perhaps bleeding, and never sleeping until the sleep is one from which he can only wake in another state of being.

As the disorder proceeds there is always more or less wasting in the muscles, and when the movements are confined to one side—a limitation which, according to some tables of Dr. Wicke,* occurred in 59 cases out of 149—the wasting was on the affected side.

As a rule, there is no stupor during the paroxysm, and even in very severe cases the patient is fully sensible of his misfortune; but, in some of the more uncommon cases, there is a dreamy state of abstraction, and the mind is more or less closed to impressions from without. In all cases the will may be said to be in abeyance, and no effort is made to change or arrest the movements. As a rule, also, there is little pain, and the sense of fatigue is far less distressing than might be expected.

During the paroxysm the pulse fluctuates a good deal, and the heart often palpitates considerably, but the circulation is in no sense excited; on the contrary, the skin is rarely otherwise than pale and cool, particularly that of the hands and feet. Under ordinary circumstances the respiration appears to be but little affected; but in the

* Romberg, *op. cit.*, vol. ii, p. 51.

severer cases it is rapid and embarrassed, and there is often a distressing sense of want of breath, which at the end of the paroxysm seeks relief in several long-drawn sonorous inspirations.

— There are also some other affections which have a claim to be looked upon as varieties of chorea. Among these, perhaps, it may be right to mention certain movements which are often considered merely as bad habits or awkward tricks, such as semi-involuntary, and semi-uncontrollable winking, grimacing, giggling, or sneezing. As varieties of chorea, it may also be right to mention (for they do not seem to admit of a more relevant notice elsewhere) those uncommon cases of rhythmical contraction of the muscles of the neck by which a semi-rotatory or oscillatory movement is given to the head. Thus: the sterno-cleido-mastoid and the trapezius muscles of one side—the muscles supplied chiefly by the spinal accessory nerve—may be the seat of the disturbance, and the effect of the contraction may be to rotate the head obliquely, so as to bring the ear and shoulder of that side closely together, to raise the chin into the air in the opposite direction, and to draw the head slightly backwards. These movements take place suddenly or gradually or by a succession of jerks; they continue for a few seconds, and then ceasing, they allow the head to return to its proper position until the time for the next contraction arrives. In an elderly gentleman, a clergyman, who consulted me about twelve months ago, these movements were repeated with tolerable regularity at intervals of a minute. In this case, as in such cases generally, the muscular disturbance ceased during sleep, and it might be suspended for a short time by a strong effort of the will, and by holding the head firmly between the hands; but at other times it went on in monotonous regularity without intermission. Or a somewhat similar semi-ro-

tatory movement of the head may be caused by the rhythmical contraction of the muscles supplied by the superior cervical nerves—the splenii and obliqui capitis; or the head may go on continually bobbing or bowing forwards in consequence of contraction alternating with relaxation in the rectus capitis anticus. Muscular affections such as these are frequently accompanied with muscular contractions elsewhere, particularly in the face and in the calves of the legs; but sometimes they are the sole evidence of disorder until the patient begins to be worn out by annoyance and local pain (for the convulsed muscles are generally very painful, particularly about their insertions) and want of rest. As varieties of chorea, moreover, it may be right to mention those strange affections which are distinguished by leaping, turning, or rushing backwards and forwards—affections, to the proper comprehension of which a few historical details may be of service.

The strange malady called the *Dance of St. John* appeared at Aix-la-Chapelle in the summer of 1374, and spread like wild-fire over the whole of Germany and the countries to the north-west. Hand in hand great multitudes of men and women blockaded the streets and thronged the churches, dancing and leaping, howling and screaming, until they fell down in a state of utter exhaustion. One symptom appears to have been a distressing state of flatulency, and to relieve this, when the paroxysm was over, they would entreat the bystanders to tighten their girdles, or even to leap upon them. Some had their heads filled with ecstatic visions, in which St. John was a prominent object; others were rendered frantic by certain colours and sounds. Sometimes the dancing movements were ushered in by symptoms of an epileptiform character. For nearly two hundred years society was disorganized by persons suffering from this demoniacal

epidemic, and by rogues who simulated it for sinister purposes. Dr. Hecker tells us* that the feast of St. John the Baptist was always held as a day of wild revelry, and that at the time when this strange malady made its appearance the Germans were in the habit of mixing up with the Christian ceremonial an ancient Pagan usage—the kindling of the “Nodfyr.” It was the custom, among other things, to leap through the flames, and to consider that a year’s immunity from the disease was gained by this baptism of fire. In this leaping run mad, Dr. Hecker thinks, we may find the origin of the Dance of St. John.

In 1418, close upon the heels of the Black Death, a second dancing epidemic, the *Dance of St. Vitus*, broke out at Strasburg, and, in a few days, the streets of this large city were filled with swarms of dancers accompanied by musicians playing on bagpipes. So numerous were they who suffered, and they who pretended to suffer, that the city authorities divided them into companies, and appointed persons whose duty was to conduct them to the chapels of St. Vitus, near Zabern and Rotenstein, as well as to protect and restrain them by the way. They were taken to these chapels in consequence of a legend, invented for the occasion, which represented that this St. Vitus, when suffering martyrdom under Diocletian in the year 303, had, in answer to prayer, received power to protect from the dancing mania all those who observed the day of his commemoration and fasted upon its eve. At any rate, to the shrine of St. Vitus the people went, and there priests were ready to sing masses and to perform other services fitted for the occasion. In its main characteristics the Dance of St. Vitus does not appear to have differed from the Dance of St. John.

* Hecker’s ‘Epidemics of the Middle Ages;’ translated for the Sydenham Society by B. G. Babington, M.D., F.R.S. 8vo, London, 1844.

Attention was first prominently directed to these two dances at the times which have been mentioned, but there is good reason to believe that they were known previously.

At the beginning of the sixteenth century, a change had taken place by which these disorders were made to approximate more closely to the chorea of the present day. This is evident from the descriptions given by Paracelsus and other competent observers. At this time these maladies were characterised by frequent fits of hysterical laughing or crying, and by an extravagant disposition to dance about, but without the howling and screaming and mental delusions and distressing flatulency of former days. In some instances, also, the propensity to dance was not irresistible. Still, now and then the disorders appeared in their old form, and Dr. Hecker tells us that so late as 1623 some women were in the habit of paying a yearly visit to the chapel of St. Vitus, in the territory of Ulm, in order that a dance at the altar there might save them from dancing elsewhere until the same time next year.

Almost contemporaneously with the Dance of St. Vitus a dancing malady, called *Tarantism*, appeared at Apuleia, and thence spread with great rapidity over the rest of Italy. It was attributed to the bite of a tarantula or ground-spider common in the country; but it is far more probable that the fears as to certain supposed consequences arising from the bite—fears arising easily in the gloomy and despondent temper of the times—had more to do in causing the malady than the bite itself. Those who were bitten became dejected and stupefied; or else, becoming greatly excited, they went about laughing, singing, or dancing. In any case, they were so acutely sensitive to the influence of music of a certain kind, as to be utterly unable to restrain themselves. A kind of

bacchantic furor was excited by the very first notes, and as the strain proceeded they would dance and leap and shout and scream until they fell down from sheer exhaustion. Some colours appear to have excited them, others to have calmed them. Some had a strong disposition to rush into the sea, and many were carried away by strong sensual passions into deplorable excesses. Some, again, were disturbed by the same flatulent distress as that which occurred in the Dance of St. John. In this case, music was looked upon as the only remedy, and the country everywhere resounded with the merry notes of the Tarentella. The favorite instruments were the shepherd's pipe and the Turkish drum. It was supposed that the poison of the tarantula was diffused over the system by the exercise of the dancing, and expelled along with the perspiration. It was customary for numerous bands of musicians to traverse the length and breadth of the land during the summer months, and the seasons of dancing at the different places were called the women's little carnival, "carnevaletto della donna," for it was the women, more especially, who conducted the arrangements and defrayed the expenses. Tarantism continued long after the Dance of St. Vitus had died out in Germany; indeed, the epidemic was scarcely at its height until the middle of the seventeenth century.

It would seem, also, that the *Tigretier*, or dancing mania of Abyssinia, a malady occurring most frequently in the Tigré country, is, in some respects, not unlike the ancient dances of St. Vitus and St. John. Beginning with violent fever, this malady soon turns to a lingering sickness, in which the patient becomes reduced to the last degree of emaciation and exhaustion. This sickness may continue for months, and end in death if the proper cure be not sought after. The first cure, which is the cheapest, is one in which a priest ministers. It is a kind of water-

cure, with a blessing superadded. If this fail, the aid of music is appealed to, and arrangements are made for a prolonged performance. The place chosen is generally the market-place. As the music begins the patient bestirs herself and rises upon her feet; as the performance proceeds, rapidly acquiring power, she throws herself into the maddest postures conceivable, dancing and leaping more like a deer than a human being. This she continues to do from early morning until the day is nearly, and the musicians altogether, spent, when she starts off and runs until her legs refuse to carry her any further. Then a young man who has followed her fires a gun over her head, and, striking her on the back with the flat of a broad knife, asks her name, when, if cured (she had never uttered this name during her strange illness), she repeats her Christian name. After this she is re-baptized and considered convalescent. The account of this extraordinary disorder is by Mr. Nathaniel Pearce, who lived nine years in Abyssinia, who saw what he describes, and who published his account about thirty years ago.*

A place in this strange category of disorders must also be conceded to those extravagant leapings and dancings which have been met with at different times among various sects of religious enthusiasts—the jumpers in this country and America, the convulsionaires in France, and the leapers from “leaping ague,” who some time ago startled the grave kingdom of Scotland. Those affected with this latter disorder complained of pains in the head and elsewhere, and soon afterwards they began to suffer at certain periods from convulsive fits or fits of dancing. During these periods they acted as if mad, distorting their bodies in various ways, springing to a surprising height, or running with amazing velocity until they fell down exhausted. When confined in cottages, a favorite

* ‘Life and Adventures of Nathaniel Pearce.’ 8vo, London, 1831.

practice was to leap up and swing about among the beams supporting the roof. The effects of music do not appear to have been tested.

The time for a general visitation of maladies such as these would appear to have passed by, at least in this country, but there are still isolated cases which serve to remind us of troubles which were once more general, and which, as I have said, have some claim to be looked upon as remote varieties of chorea. One of these, often quoted before,* is related by Mr. Kinder Wood, and this, with two that have fallen under my own observation, shall serve as illustrations.

CASE.—Mr. Kinder Wood's patient was a young married woman, who had suffered previously from headache, nausea, quick involuntary movements of the eyelids, and various contortions of the limbs and trunk. The paroxysms themselves were not always of the same kind. As the case might be, she would be violently and rapidly hurled from side to side of the chair in which she might happen to be sitting, or else thrown suddenly upon her feet, when she would jump and stamp for some time. Or, starting up, she would rush round and round the room, and tap with her hand each article of furniture that lay in her course. Or she would spring aloft many times in succession, and strike the ceiling with the palm of her hand, so that it became necessary to remove from the ceiling some nails and hooks which had done her an injury. Or she would dance upon one leg, with the foot of the other leg in her hand. These movements always began in the fingers, and the legs were not affected until the arms and trunk had been first seized upon. Noticing a rhythmical order in some of her movements, as if they were obedient to the memory of some tune, a drum and fife were procured, when she immediately danced up to the musicians, as closely as she could get, and continued dancing, until missing the step, she suddenly came to a standstill. On the next occasion, a continuous roll of the drum was tried, when the dancing movements immediately came to an end, and the patient sat down. On subsequent occasions, also, the drum was had recourse to when the dancing movements showed a tendency to begin, and with the same result, so that the dancing movements, which had lasted for about a week, may fairly be said to have been stopped by the drum. Unfortunately, however, for the credit of the music, the drum and fife were found to have lost their power on two subse-

* 'Medico-Chir. Trans.,' vol. vii, p. 237.

quent occasions on which the dancings recurred. These strange paroxysms were generally accompanied by some headache and nausea, and followed by a feeling of great weakness and exhaustion, but the patient was always able to go about her household duties in the intervals.

CASE.—The patient in this case (which fell under my own notice) was a young lady between twelve and thirteen years of age, who had suffered for about three years from a choreic practice of “making faces” and bobbing her head forwards in a very curious manner. About three weeks before the date of my first visit (24th June, 1857), she suddenly began to suffer from the peculiar paroxysms which have now to be described, and a few months previously she had suffered for some weeks in a similar manner. When attacked by one of these paroxysms she would sink or rise into a sitting posture, with the legs folded under her, and then the head would be agitated by a violent alternating semi-rotatory movement, until the hair would stream out horizontally on all sides, like the strands of a mop, when twirled over the side of a vessel. Then followed a movement in which the whole body was thrown round and round by a succession of rapid vaults. In making these vaults the hands were placed upon the floor or bed, and the arms used as a kind of leaping-pole; and except at the instant of swinging round, when the feet and legs were thrown outwards, the half-sitting, half-kneeling posture was never abandoned. The movement of alternating semi-rotation of the head, and of circumvolution of the entire body, each lasting from one to three minutes, continued, with short intervals of rest, for about half an hour or even longer, and then she would fall back, panting and exhausted. Paroxysms such as these occurred several times a day during the first fortnight of my attendance, and then ceased suddenly. After this she rapidly improved in general health, and the choreic twitchings of the muscles of the face and the bobbings of the head became much less frequent. The improvement, however, was only temporary, and at the end of three months the paroxysms returned, though in a modified form and much less frequently. At this time, indeed, the alternating semi-rotatory movement of the head did not return, and the movement of circumvolution was varied with other movements. Thus, instead of turning, she would at times make a succession of leaps in a straight line, so that it was necessary to run in order to prevent her from falling out of bed; and now and then, after falling back exhausted at the end of the paroxysm, she would roll over and over sideways for three or four times. During these strange paroxysms there was not the least trace of stupor, and she would often complain of pain in the head, or of being excessively tired even while the muscular disturbance was at its height. In some instances during the second relapse, however, the mind was in a wrapt or entranced state, and now and then words escaped which showed that she was absorbed by some alarming dream or vision. At these times her eye had a fixed stare, and her cheeks were somewhat flushed. When the paroxysm was over she would lie

for some time in an intensely nervous and excitable state, starting at the slightest noise or the gentlest touch, and now and then bobbing her head with much violence; and if the mind had been at all entranced previously, this state would continue for a short time, and then pass off with a succession of sighs. Under ordinary circumstances, however, the mind was perfectly clear, and the first moment of rest was occupied in complaining of the feeling of headache and fatigue from which she suffered. In the intervals she was nervous and excitable, but in every respect an acute, clever, accomplished, amiable girl. Her principal complaint was about a dull pain across the top of the head; an occasional complaint was about a feeling of tingling in the back and limbs. Her pulse was quick and weak, her hands and feet habitually cold, chilblains were common in only moderately cold weather, and anæmic sounds were audible in the heart and great vessels. Her appetite was very defective, and the digestion sluggish, but there were no worms nor any other evidence of derangement in the alimentary canal, beyond a slight disposition to tympanitic distension of the abdomen. Nor was there the slightest evidence of uterine derangement; indeed, the patient was in this point of view a mere child.

CASE.—This case is that of a young gentleman, Mr. E—, æt. 23, who came up from the country about six years ago to consult me for what he considered to be epileptic attacks. These attacks occurred at night, and the only particulars respecting them were gleaned from his man-servant, who slept in the same room, and who said that he had been awakened more than once by hearing sounds of choking and struggling, that these sounds had ceased before he could get up, and that his master was asleep when he got to his bedside. He had, also, other attacks, for the sake of which I now refer to the case. In the first place, he had a curious pursing up of the mouth, attended with frequent shruggings of the right shoulder, and tossings out of the corresponding leg; in the next place, he had attacks of shuddering, which were so violent as to shake things out of his hand, or even to shake himself out of the chair in which he happened to be sitting; in the third place, he had what he called “a fit of turning.” He had scarcely given me these particulars, when, after two or three electric-like shudders, he got up from the chair on which he was sitting, and standing upon the hearth-rug in an uncertain kind of attitude, he turned slowly round and round upon his heels for about twenty revolutions, and then sat down again. Before doing this he told me not to be surprised at what I saw, and not to attempt to stop him. He said, moreover, that the impulse to move round was not altogether irresistible, but that he felt it more easy to yield, and that he found himself less agitated afterwards if he yielded. This gentleman had gained honours at college, and there was no reason to suppose that his mental powers had deteriorated in any marked degree. He had suffered for some time from vertigo, and occasionally from headache, but neither of these symptoms had

ever been at all urgent. His pulse was 60 and weak, and during the paroxysm I have described it fell to 52 and became weaker. I noticed, also that his respiration was somewhat impeded, and that he drew several long breaths in succession after he had sat down.

3. *The pathology of convulsive affections which are characterised by simple convulsion.*

§ I.

1. The pulse of persons who suffer from hysteric convulsion is generally soft, quick, and much affected by changes of posture. The skin, also, is pale, and the hands and feet are subject to chilblains even when the weather is not very cold. Nor is there, as many imagine, any real excitement of the circulation during the convulsion. Indeed, the mode of breathing, which is generally slow, embarrassed, and accompanied by deep sobs and hiccup, is altogether incompatible with anything like excitement in the circulation. In all who suffer from hysteric convulsion, there is a disposition to irregular distributions of blood, which distributions are sometimes inflammatory in their character. There is, indeed, a want of balance which in one sense must be regarded as another sign of a fundamental weakness in the circulation. At the same time there is some reason to believe that unnecessary stress has been laid upon this disposition to inflammation in hysterical subjects; and there is no reason whatever for supposing that any excitement of the circulation is concerned in producing the symptom with which I am here concerned—the convulsion.

2. As in hysteria, so in chorea, the circulation is subject to considerable fluctuations, but the habitual state is one of marked depression. The pulse, most generally, is quick and weak, and the heart is readily thrown into a state of palpitation. In many cases, also, as additional evidences of a feeble circulation, the face, lips, gums, and tongue

are unnaturally pale, the skin is pasty, and the serous cavities may even be waterlogged. In some instances there may be all the signs of actual chlorosis.

A disposition to rheumatism would also seem to be common in chorea. Thus, in the digest of 309 cases of chorea occurring in Guy's Hospital, Dr. Hughes says,* that "out of 104 cases in which special inquiries were made respecting rheumatic and heart affections, there were only fifteen in which the patients were both free from cardiac murmur and had not suffered from a previous attack of rheumatism." Nor is it possible to get over this statement by supposing that the pain of the supposed rheumatism may have been simply neuralgic, and the cardiac murmur merely anæmic, for in eleven out of fourteen cases of death from chorea which are reported in this very digest, there were vegetations upon the cardiac valves. It is not to be supposed, however, that the chorea and rheumatism were actually concurrent in these cases. On the contrary, the usual statement is that the choreic patients had suffered from a *previous* attack of inflammation. Thus, in three cases that fell under my own notice recently, the chorea supervened in a period varying from seventeen to thirty-one days *after* an attack of slight rheumatic fever; and this was the case, also, in a case referred to by Dr. Romberg. Indeed, this connexion between chorea and rheumatism is altogether accidental, for we find this last-mentioned physician saying, that "the rheumatic predisposition noted by English medical men was rarely traceable in the cases which have presented themselves to my observation."†

This predisposition to rheumatism, therefore, cannot be taken as an objection to the idea, now very generally

* 'Guy's Hospital Reports,' 1846, second series, vol. iv, p. 372.

† Romberg, *op. cit.*, p. 64.

admitted, that chorea is an essentially feverless malady. Indeed, this very predisposition may be taken as an argument that the circulation in chorea is below the normal standard of activity, for, except on the rare occasions when fever is present, the circulation in rheumatism is considerably below this standard.

§ II.

1. The habitually feeble state of brain in persons subject to hysteric convulsion is shown in a variety of ways—indecision, irresoluteness, fickleness, pliability, oversensitiveness, fidgetiness, and so on. And in the fit, the will is altogether in abeyance, and the mental state is one approaching very closely to unconsciousness. Nor is it easy to suppose that any part of the nervous system can be in a more excited state; for, as in the case of epilepsy and the different forms of tremor, the state of the circulation at the time of the convulsion is incompatible with the existence of anything like high functional activity in any organ, nervous or other.

In a word, there is nothing in the history of hysteric convulsion or its antecedents which does not harmonise with the history of epilepsy and tremor, and their antecedents; and there is no necessity to suppose that the uterus has anything to do with the phenomena of the so-called hysteric convulsion beyond this—that many common and important causes of weakness and exhaustion refer more or less directly to this organ. Nor is the hypothesis of “morbid irritability” more necessary to explain the phenomena of hysteric convulsion than it was to explain the phenomena of epileptic convulsion.

2. The part which the nervous system has to play in the production of chorea, and of movements allied to chorea, is one of great obscurity. In some cases, the seat of mischief would seem to be in this place, in others

in that ; and so numerous and varied are these places that the first thing to be done here is to aim at acquiring some definite notion respecting the parts which may and may not be the seat of mischief, by taking a cursory survey of the functional history of the chief centres of nervous power.

— The experiments of M. Flourens show very clearly that the *cerebral hemispheres* may be cut or sliced in any direction without giving rise to a single painful feeling, or to a single choreic or convulsive movement. In the case of a pigeon, whose right cerebral hemisphere was entirely removed, the effect of the operation was to produce a state of stupidity, with blindness of the left eye, and marked, but very transient, feebleness of all parts of the left side of the body. The sight of the right eye was not affected, and the bird heard, felt, stood, walked, flew, and in all other respects behaved as if nothing was the matter. In another pigeon, in which both cerebral hemispheres were removed, all the signs of mental life were immediately swallowed up in profound sleep. At first, there was some evident loss of power on both sides of the body, but this soon passed off. Left to itself, the bird remained motionless in the posture of roosting ; being pushed, it walked ; thrown into the air, it flew ; left to itself again, it immediately recovered its equilibrium and fell sound asleep. Fed by putting food into its mouth, it swallowed without difficulty, and digested what it swallowed without delay. It was absolutely without the faculty of seeing and hearing and swallowing and tasting ; but it still retained some power of feeling and directing its movements, for on being pricked with a knife it behaved as an animal behaves when waking from sleep, fluttering, crying out, probably moving away, perhaps flying for a few yards. Similar results were also brought about by similar experiments upon mammals and other animals. The evidence of ex-

periment, indeed, is fully calculated to show that the seat of mental life must be sought for in the cerebral hemispheres, and this evidence is in harmony with what remains to be mentioned. It is in harmony with the fact that this part of the encephalon is imperfectly developed in idiocy, and that its development keeps pace with the manifestation of mental life in the ascent from the lower vertebrata towards man. It is also in harmony with this fact, that the same state of mental negation is produced when the proper action of the cerebral hemispheres is interfered with in other ways, as by want of blood, by pressure, or by concussion; and that the opposite state of mental excitement is produced, when an excited circulation or any other cause has determined a state of increased action in these parts. With respect to the special functions of special parts of the cerebral hemispheres or of their commissures little is known, but that little tends to show that the mental powers are one and indivisible rather than many, mapped out in as many distinct localities. There is no reason even for supposing that the anterior lobes are specially connected with speech, and the posterior lobes with sensation. It has been said that the brain may be cut and sliced without giving rise to pain, or convulsive movement, or paralysis, and this is true of the healthy organ; but it is not less true that tremblings, convulsion, spasm, paralysis, pain, and other symptoms may make their appearance when this part of the encephalon is in a state of inflammation or morbid irritability. It is to be supposed, however, that these latter symptoms must be referred to a kind of reflex implication of other parts of the encephalon, and for this simple reason—that they are not produced by injuries to the healthy cerebral lobes, and that they are produced by injuries to certain portions of the healthy encephalon, whose functional history has yet to be considered.

Experiments upon the *corpora striata* give little more than negative results. Those of MM. Majendie and Schiff show that these portions of the encephalon may be punctured or sliced in various ways without giving rise to pain or convulsive movements or paralysis, and that they may be removed without material damage to the powers of sensation and movement. In some experiments upon rabbits, however, the removal of these organs seemed to diminish the power of controlling and directing movement; for, after a while, the animals would begin to move, at first slowly, and afterwards with increasing speed and longer leaps, until they were brought to a standstill by striking against some obstacle. But there is nothing in these experiments, or in the records of pathological anatomy, to warrant the notion, entertained by some writers, that these organs are the special centres of voluntary movement.

Experiments upon the *thalami optici* produce many strange results. If, as the recent experiments of M. Schiff have shown, one of these organs be punctured, the animal at once begins to turn or roll round and round—towards the same side if the puncture be at the anterior part, towards the opposite side if the puncture be at the posterior part. Attention had been previously directed to these rotatory movements by other observers, and particularly by M. Longet; and it had also been shown that deep incisions into one optic thalamus produced pain and convulsive agitation, particularly on the opposite side of the body, with loss of vision in the opposite eye; and that after complete division or removal, the animal falls down blind in the opposite eye, and with the opposite half of its body paralysed both as to sensation and voluntary movement—a result which does not happen after complete division or removal of one of the *corpora striata*. There are, also, some clinical records which tend to show that

an apoplectic effusion or other pathological lesion is more likely to be in the optic thalamus than in the corpus striatum, if there be much diminution of *sensation* in the paralysed limb; but such evidence affords no ground for the notion that the optic thalami are special centres for *sensation*,—and yet there is no better ground to be found elsewhere.

Turning and rolling movements are also produced by puncturing different parts of one of the *crura cerebri*. If the puncture be in some parts near the optic thalamus, the movement, as Dr. Brown-Séquard shows, is towards the opposite side; if it be more posteriorly, the movement, as M. Schiff shows, is towards the same side. If the crus be entirely divided, there may still be rotation; but, be this as it may, there is, at the moment, pain and convulsive movement in the trunk and limbs on the opposite side, and afterwards paralysis of sensation and voluntary movement in the same region. And if the incision trench upon the roots of the third pair of nerves, the injury will also tell upon the parts to which these nerves are distributed—on the same side of the head, or on the opposite side, or on both sides, accordingly as the line of incision is below, or above, or across the line in which the nerves of the two sides effect their decussation.

The *corpora quadrigemina* may be removed without giving rise to either pain or paralysis, and the only marked result of the operation is total blindness—a result which is not unintelligible when it is remembered that these nervous centres are the homologues of the optic lobes in the lower animals, and that they receive the greater number of the terminal fibres of the optic nerves.

The experiments of M. Flourens have also shown that injury to one of the *corpora quadrigemina* is followed by blindness in the opposite eye, and by a state of choreic agitation, or of actual turning or rolling towards the side of the injury.

The *cerebellum* may be cut and sliced without giving rise either to pain or stupor, and without damage either to common or special sensations, but not without producing great disturbance in the movements of the muscles. After slicing away half its cerebellum, a pigeon is rendered restless, and all its movements become violent and irregular; after removing the other half, all power of flying, springing, walking, or even standing, is at an end, and the animal is left lying upon its back, or breast, or side, without any power of changing its position, but wide awake in all its senses, except perhaps the muscular. The power of executing the various movements of locomotion is altogether lost. M. Rolando, who anticipated M. Flourens in calling attention to several of these facts, has also noticed that an animal which is deprived of half its cerebellum falls upon the same side, from inability to support itself upon the leg of that side; and M. Majendie has found that an animal went backwards, instead of forwards, whenever it attempted to move, when the cerebellum was deeply wounded; and that it turned or rolled towards the side of the injury, when the posterior part of the *processus cerebelli ad pontem* was pricked or cut. M. Lafargue has also shown, that the animal turned or rolled towards the opposite side when the anterior part of the last-named process was injured. If the restiform bodies be injured, the animal cries out with pain, and frequently it turns at the same time; if the *crura* be injured, it turns without fail, and suffers pain, but by no means to the same degree as when the restiform bodies are the seat of injury. It must not be forgotten, however, that there are not a few cases in the records of pathology, where the cerebellum was destroyed more or less entirely by disease, or where this organ has been absent, in which such a state of things was not made known by any particular disorder of the muscular movements during life. And certainly there is

nothing in these records, and as little in the evidence of experimental physiology and comparative anatomy to justify the notion, which was so strenuously upheld at one time, that the cerebellum has some special connection with the sexual function.

The inquiries of MM. Longet and Flourens have shown that a rabbit or puppy may be deprived of the whole of the encephalon above the *pons Varolii* without extinguishing all traces of mental life. A pigeon so treated is at once plunged into the profoundest sleep; it is perfectly motionless if left to itself; it breathes, but more slowly than natural; it swallows, if fed; it stands, but it has no longer the power of flying, or leaping, or walking; it is absolutely blind and deaf, but it still cries out when its tail is pinched, and puts up its foot to push away the bottle when ammonia is held to the nose. If the pons be cut or injured in the anterior and superior parts, the animal, as M. Majendie pointed out, turns or rolls towards the opposite side; if the injury be near the insertion of the fifth pair of nerves, the experiments of Drs. Brown-Séquard and Martin-Magran show that there is still rotating movement, but in a different direction to that which was noticed by M. Majendie—that is, towards the same side. According to its position, also, the injury may tell upon the parts which are in more immediate relation to the pons through the cranial nerves connected therewith, the effects being on the same side of the head, on the opposite side, or on both sides, according as the injury implicates these nerves below, or above, or through their line of decussation in the pons.

A rabbit or puppy which has been deprived of the whole of its encephalon above the *medulla oblongata* continues to breathe and swallow, and various other reflex movements go on in the trunk and limbs; but it no longer cries when its tail is pinched, or puts up its foot to push away

the bottle when ammonia is held to its nose, as it did while it retained its pons Varolii. The removal of this last-named centre has, in fact, extinguished the last faint trace of mental life. There are also many experiments which show that very different effects are produced by mutilating different parts of the medulla oblongata in animals which still retain the superior parts of the encephalon. On dividing one of the anterior pyramids above the points of decussation, the animal immediately loses all power of voluntary movement in the opposite half of the body below the neck, but there is no perceptible lesion in the sensibility of the paralysed parts. And this result is not unintelligible, for it is an ascertained fact that nearly all, if not all, the fibres which carry the orders of the will to the muscles pass in the anterior pyramids, and in the layer of gray matter in contact with them, and that these fibres change sides at the points where the anterior pyramids decussate, that is at the lower border of the medulla oblongata, and at this place singly, and not all along the median line of the encephalon, as was once supposed. It is also a fact that the muscles which are paralysed as to voluntary movement by dividing the anterior pyramids are not readily thrown into contraction by puncturing this portion of the medulla oblongata. On dividing one of the corpora olivaria (which body is the true continuation of the anterior column of the spinal cord on that side, and in which are very few, if any, of the fibres which bring the muscles under the orders of the will), there is, on the contrary, little or no paralysis anywhere, and as little pain, but the injury at once produces a state of spasm in the muscles of the side, especially of the neck, and the contracted muscles may remain contracted, sometimes for hours, sometimes for days and weeks. This result (which is not peculiar to this part, for permanent contraction is present in every case of rotatory movement—indeed, the

rotation is chiefly due to the inability to move in a straight line in consequence of the muscles of one side being in some degree in a state of permanent contraction) may happen, also, after removal of all the encephalon above the medulla oblongata. Dr. Brown-Séquard, who has noticed this fact, has also found that the animal will turn or roll towards the same side if the medulla oblongata be punctured near the insertion of the lower roots of the par vagum, and towards the opposite side if the puncture trench upon the insertion of the glosso-pharyngeal nerve. M. Schroeder Van der Kolk, moreover, looks upon the olivary bodies as the special ganglia of the hypoglossal nerve, and as having the regulation of the movements concerned in speech, but the facts in support of this opinion are as yet not altogether conclusive. On carrying the incision more deeply, the breathing stops, and instant death is very likely to be produced; and this fact led M. Flourens to think that the medulla oblongata contained a point in which life was centred—the *nœud vital*, and that the medulla itself was the centre of the respiratory movements; but Dr. Brown-Séquard has exposed the fallacy of this opinion by showing that instant death does not happen if the pneumogastriacs are divided before dividing the medulla, and that irregular breathing will continue for a short time after the entire removal of this centre in animals whose spinal cord is rich in gray matter, as in alligators, birds, puppies, kittens. He has shown, indeed, that the cause of sudden death in extensive injury to the medulla oblongata is the stoppage of the movements of the heart by the shock transmitted along the pneumogastriacs. There are also other experiments by various observers which show that one side of the medulla oblongata may be completely divided without causing death, and that the effect of the operation is to leave the animal paralysed as to sensation, as well as voluntary movements, on the opposite side of the

body, below the incision—a result which is explained, for the voluntary motor fibres, by the decussation of the anterior pyramids at the lower portion of the medulla oblongata, and, for the sensitive fibres, by their decussation in the medulla itself, as in the spinal cord, at the level of their insertion or thereabouts. It is noticed, also, that the sensibility and warmth are likely to be increased in the non-paralysed side of the body—a result which may be due to paralysis of the vaso-motor nerves on this side having brought with it an ampler supply of blood by leaving the vessels in a dilated state.

Dr. Brown-Séquard has shown that a rabbit or any other animal turns or rolls towards the opposite side, if one half of the *spinal cord* be punctured or otherwise injured in the neighbourhood of the medulla oblongata. He has shown that the effect of dividing one half of the spinal cord in any place below the decussation of the anterior pyramids is to produce a crossed paralysis so far as concerns sensation and the power of voluntary movement below the level of the incision, the paralysis of sensation being on the opposite side of the body, the paralysis of motion being on the same side. He has also shown that, in many instances, the sensibility and natural warmth are augmented on the same side as that of the section of the cord, and that augmented sensibility is a constant result of injury to the posterior part of the cerebro-spinal axis from the tubercula quadrigemina to the lower end of the spinal cord. Nor has he left these curious facts without explaining them. He has explained how it is that the division of a lateral half of the spinal cord produces paralysis of sensation on the opposite side, below the section, by showing that the sensitive fibres and cells (the transmission of sensitive impression in the spinal cord takes place chiefly through the gray matter, partly through the anterior columns, and to some extent, before reaching the gray matter, through

fibres belonging to the posterior columns) cross in the spinal cord at or near the level of entrance, and then pass upwards along the opposite side of the cord. He has explained how it is that the division of a lateral half of the spinal cord produces paralysis of voluntary motion below the section on the same side, by showing that these voluntary motor fibres, after decussating in the anterior pyramids, pass down each side of the cord, (along the lateral columns, in the gray matter below them, and also in the anterior column) without decussating in their course along the cord. He also affords some clue to the explanation of the augmented sensibility and warmth which may be present on the side which is paralysed as to voluntary movement, by hinting, as has been already mentioned, that both phenomena may be due to paralysis of vaso-motor nerves on this side, and to the admission of an ampler supply of blood into the paralysed vessels.

All that need be said respecting the *ganglia of the sympathetic system* has been said already (p. 113). It is very possible that these centres may be the source of reflex disorder in other centres, and that impulsive or vertiginous movements may originate in this way, but there is no reason for supposing that such movements may be the direct result of disorder in these centres.

Looking back at these facts, then, it may be supposed that choreic movements of a rotatory character may originate in various parts of the nervous system—in the optic thalami, corpora quadrigemina, crura cerebri, pons Varolii, crura cerebelli, in certain parts of the medulla oblongata, and also in the upper portion of the spinal cord; that common choreic agitation may be caused by slicing away the cerebellum, and by puncturing one of the corpora quadrigemina; that the removal of the encephalon before the optic thalami *may* be attended with an impulse to go forward; and that a deep wound in the cere-

bellum *may* be attended by an impulse to go backward. The parts injured, says Dr. Brown-Séquard, "seem to be quite different from those employed in the transmission of sensitive impressions or of the orders of the will to the muscles, at least in the medulla oblongata and pons Varolii. They constitute a very large proportion of these two organs, and perhaps the three fourths of the first one; they are placed chiefly in the lateral and posterior columns of these organs; many of their fibres do not decussate, and produce spasms on the corresponding side of the body; they seem to contain most of the vaso-motor nerves, by which, directly or through a reflex action, they may act on other parts of the nervous system."* It is possible, also, to look upon the disordered movements of the facial muscles and tongue—which are such characteristic features of chorea—as indications of disorder in the same parts of the nervous system, for the nerves which are at fault here are intimately connected with these parts—the facial arising near the pons Varolii, from the upper part of the groove between the olivary and restiform bodies, the hypoglossal nerve having a still more intimate relation to the olivary bodies.

There are, moreover, one or two facts which must not be passed over—facts which seem to show that rotatory movements may have their starting-point in a *nerve* at a distance from the nervous centres. Thus, Dr. Brown-Séquard has shown that a rabbit may be made to turn or roll towards the injured side, by injuring the expansion of the auditory nerve within the ear; and M. Flourens has witnessed similar movements in a pigeon, after simply tying a bandage over one of the eyes. It would seem, indeed, as if the parts of the nervous centres which are concerned in the production of these movements may be affected from a distance by *reflex* action. Nor is this to be wondered at;

* 'Lectures on the Physiology and Pathology of the Central Nervous System.' 8vo, Philadelphia, 1860, p. 196.

for it is a fact which cannot be questioned, that distant parts of the nervous system are continually being affected in this manner and that the varied consequences of a particular injury are only to be accounted for by supposing many of them to be of a reflex character. It is a fact, for example, that a guinea pig becomes subject to a peculiar epileptiform disease if its spinal cord be injured. It is a fact that paralysis may make its appearance on the *same* side of the body, when the superficial fibres of the crus cerebelli in the neighbourhood of the insertion of the fifth pair of nerves are injured. It is a fact that epileptiform seizures may result from a diseased condition of a portion of the upper surface of a cerebral hemisphere. It is a fact that tetanus may arise from a punctured nerve in the thumb. Indeed, there are no lack of instances to show that any part of the nervous system may act upon any other part, and that the exact diagnosis of many disorders of this system is a difficult, if not hopeless, task, for the simple reason that any given lesion in any part may be attended by a wide range of symptoms depending upon sympathetic disorder in other parts.

— Reverting to what has been already said respecting the general history of persons suffering from chorea, it is not easy to find any reason for supposing that the choreic movements are dependent upon over-action of the nervous system. The vacillation, irrationality, inordinate sensitiveness, timidity, fretfulness, and irritability of these persons shew very plainly that the cerebral hemispheres are acting very feebly, and the depressed state of the circulation would seem to imply a corresponding depression in the functional activity of all the nervous centres, without exception, in common with every other organ in the body. It would seem, in fact, as if the disordered muscular movements were connected with a deficient supply of nervous influence to the muscles.

Nor are the disclosure of pathological anatomy at variance with this conclusion. In the fourteen cases of deaths from chorea contained in Dr. Hughes' digest (which cases may be said to constitute more unexceptional evidence on the subject than any other on record) the brain was quite healthy in four, and only congested in three others, so that we may conclude that there was nothing the matter with the brain in half the cases. And of the remaining seven cases, the particulars are as follows:—in the first, serous effusion beneath the arachnoid and into the ventricles, slight effusion of blood beneath the right cerebral hemisphere, softened brain;—in the second, arachnoid opaque, brain dark and soft;—in the third, pia mater watery, cineritious matter red, soft, and partially adherent;—in the fourth, brain soft and vascular, much fluid in ventricles;—in the fifth, arachnoid opaque in parts, cerebrum vascular, left thalamus rather soft;—in the sixth, dura mater adherent very firmly to calvarium, and more opaque than natural, cerebral vessels turgid;—in the seventh, blood effused into arachnoid, fornix and edge of third ventricle soft, red, and tumid, brain softened. In the same fourteen cases the spinal column was not opened in six. Of the remaining eight, the cord and its membranes were quite healthy in three, and only a little congested in one, so that there was nothing the matter with the spinal cord or its membranes in half the number examined. Of the remaining four, the particulars are these:—in the first, soft adhesions of the arachnoid, gray matter dark;—in the second, vessels rather large and numerous, serous surfaces opaque, old adhesions of the membranes, especially posteriorly;—in the third, medulla slightly softened, rachidian fluid opaque, yellow, and densely coagulable by heat;—in the fourth, softening of the cord opposite the fourth and fifth dorsal vertebræ. In half the number of cases, therefore, there are signs which show more or less clearly the presence of inflamma-

tory changes during life, but it is not possible to assume that these changes have any essential connection with the chorea, and this for no other reason than that they were wanting in the other half of these very cases. It may be, indeed, that the inflammation preceded the chorea, and left the cord damaged, and to that extent weakened, and this opinion would not seem to be improbable where the signs of mischief were evidently of no recent date; or it may be that the inflammation had been a consequence rather than a cause of the chorea,—the cord, like the muscles, breaking up, as it were, from sheer fatigue. Or, possibly, the appearances as of inflammation, if they had been more carefully noted, might have been explained without the hypothesis of inflammation of all.

And, certainly, there is very little in the history of the artificial production of rotatory movements to make it necessary to suppose that the injury has acted in the sense of a stimulus to the irritability of the injured nervous centre. The fact is simply this, that the rotation commences immediately upon the infliction of the injury. The part is punctured, and the animal turns or rolls without the loss of a single moment. There is no time for the setting up of inflammation. There is a mechanical lesion; there may be, nay there must be, some disturbance in the electrical equilibrium of the part. After what has been said, indeed (pp. 66—69), there is no difficulty in understanding that the effect of the injury will be to produce a momentary disturbance in the electrical equilibrium of the part by weakening its nerve-current; and that this disturbance of equilibrium, propagated along the musculo-motor nerve and producing movement in its nerve-current, may produce momentary muscular contraction by giving rise to the induction of momentary induced currents in the muscles to which the musculo-motor nerves are distributed. The difficulty is in understanding how the disturbance in the elec-

tric equilibrium of the nervous centre should be perpetuated so as to produce continuing contractions in the muscles; and this difficulty is one with which it is not very easy to deal. It is possible, however, that the injury which produces the turning or rolling may act with different degrees of force upon different parts of the injured nervous centre, and that the electrical activity of the parts at a distance will be less disturbed than that of the parts immediately acted upon. It is possible that the recovery of the normal electrical activity of the injured parts, may be retarded in proportion to the degree of the injury, and that this recovery may occupy some considerable time in the parts most injured. It is possible, too, that the electrical equilibrium of the injured nervous centre may not be re-established until the normal electrical activity of the several injured parts is recovered, and that the nerve-currents of the centre will be in a state of unrest until this equilibrium is established. And if so, then it is possible to perceive in the distance why it is that the muscles under these circumstances may be kept in a state of continuing contraction; for so long as the nerve-currents of the nerve-centre are in a state of unrest, the nerve-current of the musculo-motor nerves will not be at rest; and so long as there is any movement in the nerve-current of the musculo-motor nerve, this movement will produce muscular contraction, by giving rise to the development of induced currents in the muscles to which the musculo-motor nerves are distributed. It is possible, indeed, to imagine an electrical condition of the nervous centres which will involve a continual state of muscular contraction; and this being the case, the problem of turning or rolling is half solved, for Dr. Brown-Séguard has shown that turning or rolling is caused by an attempt to move when the muscles of the two sides of the body contract with unequal degrees of force, in consequence of the muscles of one side being more or less in a state of continuing contraction.

— In the pathology of chorea and the cognate affections there are, no doubt, many points upon which it does not do to dogmatise in any manner; but the tenor of the evidence, so far as it may be apprehended at present, is in favour of the previous conclusions respecting hysteric convulsion and tremor and ordinary epilepsy. And thus it may be said that the pathology of simple convulsion is in harmony with the theory of muscular motion which has been advanced in the physiological premises, rather than with the current theory of muscular motion.

4. *The treatment of the convulsive affections which are characterised by simple convulsion.*

1. In the treatment of hysteric convulsion it is manifestly of primary importance to correct, as far as may be, those erroneous habits which have led to the disorder, and because this is no easy task, the results of treatment are by no means so certain and satisfactory as might be expected. Exercise, early hours, useful occupations, other reading besides novels, and strict temperance in all matters relating to the emotions and appetites—a rule of life, indeed, rigorously in accordance with the dictates of common-sense—all this must be insisted on and realized before much good can be done. In matters of eating and drinking there seem to be no sound reasons for supposing that a different rule is necessary to that which is ordinarily observed in this country, but if there be, it is not a rule requiring less wine and stimulants than usual. Indeed, there are many cases, especially those in which the strength is continually kept down by profuse menstruation or leucorrhœa, in which stimulants, in no stinted quantity, must be regarded as necessaries of life.

It may be questioned, moreover, whether the practice of giving purgatives is so beneficial as it is supposed to be by some. Aperients may be very necessary, and

active purges may now and then do no great amount of harm, but neither aperients nor active purges are in much request if care be taken to prevent the diet from being too unstimulating, and the habits from being too sedentary. At any rate, according to my own experience, the bowels under these circumstances will act well enough with little aid from aperients or purges; the stomach will do its work all the better for being allowed to do it in peace; and hysteric convulsion would seem to be a less frequent occurrence.

It may also be questioned whether the use of the shower-bath is called for in the majority of cases. If there be no objection to it on the part of the patient, and if reaction be established at once, it may do good; but where the patient shrinks from the shock and is slow to recover her warmth, the benefit would seem to be very problematical. I think, indeed, that I have seen several cases in which positive good has resulted from its discontinuance.

It is no doubt a matter of extremest moment to correct any uterine derangement, and in order to this, as it seems to me, we cannot be too chary in having recourse to measures which may serve to give an undue prominence to uterine matters in the imagination of the patient. Common measures used in the common old-fashioned way, particularly if the diet be not too unstimulating, will rarely fail to set matters right.

The other symptoms are such as to indicate the necessity of tonics, and few are now deterred from the use of these remedies by any scruples about plethora and irritation. In some cases, iron seems to be very serviceable; in others, perhaps in the majority, cod-liver oil seems to be more serviceable; in a few cases in which I have had an opportunity of trying it, the addition of phosphoretted oil to the cod-liver oil would seem to have done positive

good. If pain be a prominent symptom, anodynes may be required, as opium in one or other of its forms, henbane, &c., and these remedies will be found to agree much better than they do in epilepsy; but, as a rule, the pain would appear to be best quieted by the remedies which are had recourse to for removing the spasmodic symptoms—ether, ammonia, valerian, assafœtida, or it may be, wine. Indeed, the principles of treatment are in no essential particular different from those which have been advocated when speaking of epilepsy, except that more may depend upon a radical reformation of the habits of the patient and less upon the use of medicine.

In the convulsion itself little need be done beyond loosening the stays and sprinkling cold water in the face; but if more be required, as when fit succeeds upon fit in spite of all ordinary measures, there is nothing better than the plan recommended by Dr. Copland, which is to throw up an enema, consisting of half an ounce or an ounce of turpentine in a little gruel, or milk, or broth.

2. "When," says Dr. Watson, in his chapter on chorea,* "a vast number of different drugs are recommended as specifics in any given disease, we may sometimes infer from that very circumstance that the disease is difficult of cure, and generally untractable under all plans of management. But there is another class of diseases which a variety of drugs are supposed capable of curing—those which tend to terminate in health. I believe, also, that many of the boasted specifics have been quite innocent of any share in the recovery of the patients to whom they were administered: at the same time, I am quite certain that treatment has a great influence over the disease."

Where there is a fixed pain in the head, Dr. Watson recommends the local abstraction of blood, but, with this exception, he considers that "bloodletting is neither

* Watson, op. cit., vol. i, p. 675.

useful nor even satisfactory." Indeed, he allows that there is "a deficiency rather than a redundancy of red blood in the system." He says further that, setting aside this complication with headache, most of the cases of chorea may be dealt with successfully if we have at our command purgative medicines, the cold shower-bath, preparations of iron and arsenic, and the oil of turpentine. "The instrument," as Dr. Watson says in another place, "is not broken anywhere, but slackened, jangling, and out of tune; and (to pursue the metaphor) we often can restore its harmony by bracing it up again."

It may be a question, however, whether leeches are required even in exceptional cases, whether purgatives given by the mouth do not derange the stomach to a degree for which there is no compensating advantage, and whether the shower-bath is altogether innocuous; for an opposite plan of treatment would seem to answer the end as well,—if, as Dr. Watson believes, "there are *cures* in the disease as well as recoveries." At least, I have been in the habit of placing most confidence in stimulants for some time back, and apparently this confidence has not been misplaced. The following case is the first of seven cases, treated in the same manner under the same roof, and with similar results:

CASE.—S. T—, æt. 17, a clerk to a coal merchant, was admitted under my care into Burdett Ward, Westminster Hospital, on the 6th of January, 1858.

About two months previously he began to complain of pain and weakness in the ankles, and for these symptoms he went to the Orthopædic Hospital, and was recommended to wear strong boots with irons up the legs. A few days later his speech became hesitating, and the limbs were jerked about in a ludicrous and awkward manner. Ten days ago these latter symptoms were considerably aggravated, and at the advice of a physician of eminence he was put under a treatment consisting of daily purges of croton oil, with cod-liver oil and digitalis. It appears, further, that he had suffered for three or four years from frequent headache and epistaxis.

On admission, the expression of the countenance was so vacant as to

suggest the idea of idiotcy—an idea not a little confirmed by the fact that the saliva was allowed to dribble from his mouth without any effort to wipe it away. His grimaces were extraordinary, and the tossings of his head and limbs and body were such as to make it difficult for him to sit in a chair while the bed was being got ready. If spoken to, his apprehension appeared to be very slow, and his speech was so "thick" as to be unintelligible. The tongue could not be held out for a single instant. The hands and feet were very cold; the pulse was 72, weak; the heart's sounds normal; the appetite good. The treatment recommended was the following draught:

℞ Naphthæ purificatæ naturalis,* f ʒj;
 Ferri ammonio-citr., gr. xv;
 Mucil. Acaciæ, q. s.;
 Aquæ menthæ pip., f ʒj;

every four hours, a turpentine enema every night, with wine and full diet.

January 7th.—The sleep has been sound and refreshing throughout the night. The enema operated well. Pulse 88, stronger. Speech more intelligible. Agitation of the arms and head less marked.

8th.—Another good night's rest. The tongue can be kept out without much difficulty. The arm can be held out with comparatively little jerking. Pulse 88, stronger. Enema brought away more fæces of a dirty mud colour. None of the evacuations following the enema have been copious, and the patient says that the effect of the enema is to make him warm for two or three hours. On inquiring as to the effects of the purgatives given to him before he came into the hospital, he said that they had made him feel sick, and that they had increased the agitation and taken away his appetite.

13th.—This afternoon he is sitting up in a chair, and on being told to get up and walk along the ward, he did so with very little assistance. The tongue is now protruded naturally, and there is scarcely anything wrong with his speech. The enema to be discontinued.

19th.—The choreic symptoms have altogether disappeared, the speech is quite distinct and clear, the countenance has lost altogether the vacant expression it had upon his admission. Convalescent. The naphtha and steel draught to be changed for another containing quinine and steel.

* Three liquids are known under the name of naphtha:—(1.) Pyroxylic spirit, or wood naphtha; (2.) pyroacetic spirit, or acetone; and (3.) natural, mineral, or coal naphtha. This last liquid, which is closely akin to petroleum, and to which the name naphtha ought to belong exclusively, is the liquid here given under the name of naphtha naturalis.

As to the value of iron in chorea it is not very easy to come to a conclusion. There are many cases, without doubt, where this remedy does not seem to agree; and I am disposed to think that, in the end, the oily tonics will be more suitable in the majority of cases than the ferruginous. I base this conjecture, partly upon the marked benefit which I have found to result from the use of these tonics in a more serious convulsive affection, namely epilepsy, and partly upon the fact that cod-liver oil is known to be a very valuable remedy in a state of chronic and subacute rheumatism—a state with which chorea would seem to have some not very distant affinities. I may mention also, as a feeble argument in support of the same conjecture (for little more can be proved by one case), that I have at present under treatment a case of slight chorea, in which, after giving naphtha and steel for a fortnight with little benefit, convalescence became fully established within the week under the use of cod-liver oil and phosphoretted oil. Of the good arising from zinc or arsenic I am not able to speak very confidently, from the little I have seen in my own practice.

Now and then the stimulating purgatives (as castor oil and turpentine) may be rendered necessary by the disturbance arising from worms. Now and then, too, the aid of surgery may be required to remove carious teeth, or for the more serious operation of dividing offending nerves and muscles. The former need will often arise, for a carious tooth is frequently found to play an important part in keeping up a state of chorea; the latter need can but seldom occur, but it may occur in cases of local choreic disturbance, as in that in which the head is being continually twisted half round in consequence of recurring contractions in the sterno-cleido-mastoid and trapezius muscles from a disordered state of the spinal accessory nerve.

And not unfrequently generous living, gymnastic exercises, abundance of sleep, and a merely nominal treatment, have served to bring about a speedy "cure," upon leaving off the cold shower-bath, and ceasing to "regulate the secretions" by a too assiduous devotion to purgatives.

CHAPTER IV.

ON CONVULSIVE AFFECTIONS WHICH ARE CHARACTERISED BY EPILEPTIFORM CONVULSION.

EPILEPTIFORM convulsion, as I have already said, agrees with ordinary epilepsy, and differs from simple convulsion, in that it is attended with loss of consciousness. It may occur in connexion with certain diseases of the brain—chronic softening, chronic meningitis, tumour, induration, hypertrophy, atrophy, congestion, apoplexy, inflammation—with fever, with certain retained excretions, with “irritation” in the gums and elsewhere, and with the moribund state; and a clear view respecting it is only to be obtained by considering it in these several connexions.

1. *The history of the convulsive affections which are characterised by epileptiform convulsion.*

1. The persons in whom epileptiform convulsion is a sign of *chronic softening of the brain* are never young. If not advanced in years they are prematurely old. Under all circumstances there is unquestionable, often very marked, impairment of the intellectual faculties, and, in extreme cases, the mind may be a complete wreck. Fire and energy have died out, and dulness and drowsiness point to the coming coma of which they are the forecast shadows.

Paralysis, generally of a hemiplegic character, is the usual accompaniment of this mental blight—paralysis which, as a rule, differs from the paralysis of hæmorrhage

in being more slowly developed, as well as in being less complete and less uniformly unchanging. At one time or another, also, and generally, but not always, on the paralysed side, there is more or less permanent spasm; but this symptom is not always present, and, most assuredly, it is not that diagnostic sign of softening which it was once supposed to be. Trembling and shuddering are also occasional symptoms.

Pains in the head are rarely absent, and pains in the limbs and paralysed muscles are common troubles. In the head, the pain is rarely more than a dull aching diffused over a considerable portion of the scalp, and sometimes it does not amount to more than a sensation of weight. It is often spoken of as rheumatic, and so also are the pains in the limbs. In other respects the nerves of sensation do not appear to be much affected. There is often an arcus senilis in the eye, but vision is not often seriously damaged, and hearing and feeling are less affected than might be expected from the extent of the paralysis.

Of the other appearances which may be noticed during life, the principal are those which indicate a feeble circulation—extremities habitually cold, cord-like arteries from atheromatous changes in their coats, a heart weak and it may be fatty, and so on.

In this condition the brain is pallid, whiter than it ought to be, deficient in red spots, and in parts softer than natural, sometimes even diffuent. These parts are more commonly met with in the medullary or least vascular portions of the cerebral tissue, and their locality is not at all determinate. Examined microscopically, the softened substance is found to consist of broken-down brain-tissue, with a greater or less number of cells containing oil, and sometimes with few or many blood-corpuscles (for hæmorrhage is a common consequence of softening), *but*

without the characteristic products of inflammation, such as exudation-corpuses and pus-corpuses. The colour of the softened portions will be pale or red in various shades, according to the quantities of blood mixed in them. Atheromatous and calcareous deposits are also common in the vessels of the brain.

— The epileptiform convulsion which occurs in chronic softening of the brain is usually less violent than that of simple epilepsy, but not always. Consciousness is completely lost, and the subsequent stupor passes off very slowly. Recovery, indeed, is imperfect as well as tardy, and the mental confusion or paralysis which existed before the fit is generally aggravated by the fit. In a word, the effects of the storm do not pass off so speedily and (in a sense) so completely as they do in ordinary epilepsy, though it often happens that the symptoms of suffocation and cerebral congestion during the fit itself are less urgent.

2. *Chronic meningitis* is generally met with in children or in young persons, and not in persons past middle age, as is the case with chronic softening of the brain. It is usually accompanied by evidences of a scrofulous habit, and not unfrequently it would appear to have originated in some local mischief, such as disease in the bones of the ear.

As in chronic softening of the brain the most prominent symptom is progressive impairment of the mental faculties, but in chronic meningitis this impairment would seem to be attended by peevishness, impatience, fidgetiness, and a disposition to delirium, rather than by dulness. At one time or another mental wandering in the evening or at night is a common symptom, and not unfrequently this wandering may be exaggerated into actual insanity. It would seem, also, that convulsion occurs more commonly than it did in chronic softening of the brain, and that

spasm and paralysis occur less frequently. At an early stage of the disease there will often be twitchings or contortions in the features, or squinting, or some tripping of the tongue, and at an advanced stage there may be a palsied limb—this being, in all probability, the extent of the mischief in this direction. It is no unusual thing to find the flexor muscles of the paralysed limb permanently contracted, and the whole of these muscles unduly sensitive to percussion; but spasm, either permanent or transitory, is not a prominent phenomenon. Headache is rarely absent, and the pain is very much of the same character as it was in chronic softening—dull, and not confined to any one spot in particular. Vertigo is a frequent phenomenon.

The pulse, for the most part, is quick, weak, and greatly affected by changes of posture, as indeed is to be expected from the state of weakness and exhaustion which is invariably present. In the evening faint hectic reaction is a not uncommon condition, when the cheeks become flushed, the eyes brilliant, and the aching head hotter than it was before; and this faint hectic reaction is one of the points in which chronic meningitis differs from chronic softening. Moreover, nausea, vomiting, and constipation are not uncommon symptoms.

— The epileptiform convulsion which occurs in chronic meningitis may be general and accompanied with complete loss of consciousness, but in many instances it is confined to a part of the body, and some degree of consciousness is retained throughout the paroxysm. In a case recently under my care, the convulsion never went beyond the most extraordinary agitation of one arm, and though utterly unnerved and wholly unable to control herself, the patient remembered everything that had happened during the fit. In a case like this, or in any case where the state of coma is absent or incomplete, there is no consecutive

stupor; and it would always seem to be a marked difference between the convulsion arising from chronic meningitis and the convulsion arising from chronic softening, that the consecutive stupor is not a marked phenomenon in the former instance.

Chronic hydrocephalus is, in every sense, a passive rather than an active affection. Its affinities, indeed, connect it with atrophy, and not with any inflammatory changes, for the brain is, for the most part, wasted, the membranes unchanged, or only slightly opalescent, and the effused fluid is simple serum highly diluted with water. Or if there are evidences of structural change in the membranes or elsewhere, the microscope shows that the change is of a tuberculous character. If the affection be congenital and life prolonged, the child is in all probability blind, deaf, dull, idiotic perhaps, paralysed and wasted; and it would seem to be a rule that the frequency of convulsion is directly related to the degree of the wasting.

3. *Tumour of the brain* may be of several different kinds. It may consist, for example, of tubercle or cancer; it may be formed by an hydatid or aneurismal growth. Tubercle, which is by far the commonest kind of tumour, is generally met with in children and young persons, and where it is met with, it is rarely a solitary manifestation of the scrofulous habit. Cancer, which ranks next to tubercle in order of frequency, is oftener met with in persons advancing or advanced in life, and, as was the case with tubercle, the deposit in the brain is rarely the only evidence of the constitutional disease. The other kinds of tumour are comparatively uncommon, and when they do occur their character is only to be guessed at inferentially. Thus, aneurism may be suspected if the vessels elsewhere furnish evidence of similar enlargement.

The symptoms of tumour of the brain are far from

characteristic when clearest; and in cases where the development of the morbid growth has been gradual, they may be obscure to the last degree.

Epileptiform convulsion is a very common symptom, as common as in chronic meningitis, and much more common than in chronic softening. Scarcely ever absent, it is difficult to associate it with any particular position of the tumour. "Upon analysing a considerable number of cases," says Dr. Reynolds in his excellent work on the 'Diagnosis of Diseases of the Brain,' I find that convulsions are most frequent in tumours of the cerebellum, and that they diminish in frequency as the seat of the tumour advances forwards—that is, through the posterior and middle to the anterior lobes of the cerebrum;" but, unfortunately, there are too many exceptions to make this conclusion of much practical value. There are, also, quite as many exceptions to contradict the notion that convulsion is a much more common symptom in tumours of the meninges than in tumours of the substance of the brain. And this is not to be wondered at, for, as has been seen already, there is such an admixture of direct and reflex symptoms, arising from the intimate sympathies which exist between the different parts of the encephalon, as to make it, in many instances, almost impossible to decide upon the actual starting-point of the mischief.

Headache is another very common symptom in tumour of the brain, and the pain is of a particular character. It is not dull, diffused aching, as in chronic softening or meningitis; it is severe, and confined to one spot, with frequent pangs of a violent and almost intolerable character. These pangs are readily brought on by any effort, mental or corporeal, by emotional excitement, by a glare of light, or by a sudden noise. The pain is generally referred to the neighbourhood of the tumour, but not always; thus, the pain connected with a tumour in the

cerebellum has been confined to a limited spot in the forehead. M. Romberg says that the pain is increased by forced *expiration* when the tumour affects the upper surface of the brain, and by forced *inspiration* when it affects the under surface, and many think that tumours of the meninges are more characterised by pain than tumours in the substance of the brain. But here, again, the evidence is not very conclusive, and the probability is that the symptom of pain is of no great service in fixing the position of the tumour.

Vertigo and vertiginous movements are often met with in cases of tumour of the brain, but not so often, perhaps, as is supposed. It would seem, too, that these symptoms are of as little use in fixing the position of the tumour as pain or convulsion, for there is no lack of cases in which they were present where the incito-motory tract was not directly implicated.

Amaurosis is another symptom which is often met with in tumour of the brain. "Amaurosis," says Dr. Reynolds, "is most common in tumour of the anterior cerebral lobes; and it becomes relatively less frequent as the seat of the tumour retrogrades;" but it is not less true that this symptom is very often met with in cases where the tumour has been seated in the cerebellum. When the amaurosis is of reflex origin it varies considerably, coming and going alternately, and varying in degree even within a few minutes; and this will be of some use in diagnosis, for there is a greater probability of the optic nerve not being directly implicated if the amaurosis be of this character; but there may be some doubt as to the practical value of this rule.

In some cases other cranial nerves may be concerned, and the disorder of sensation or motion in the localities to which these nerves are distributed may show that this is the case. If, for example, the articulation be impaired, it

is possible that the tumour may press upon the hypoglossal nerve; if the facial muscles are paralysed, the pressure may bear upon the facial nerve. But here, again, there is the same difficulty to be contended with, for it is quite possible to find cases in which each particular cranial nerve has been affected, not directly, but indirectly, that is, by a reflex action arriving at it from a distance.

In some cases, also, there may be paralysis of motion or sensation, one or both, in the limbs on one side, or even to a greater or less degree on both sides of the body; or there may be rigidity in some of the muscles of the arm or leg—sometimes on the opposite side to that of the paralysis; and these paralytic or spasmodic symptoms, interpreted by the functional history of the nervous centres, will help to fix the position of the tumour. As a rule, however, the arms and legs would seem to be free from either paralysis or spasm in tumour of the brain.

As a rule, too, the intelligence does not suffer in any marked degree in these cases, and when it is otherwise the difference would seem to be owing to the accidental presence of chronic meningitis. And, certainly, there is little reason for supposing that disturbance in the mental faculties is in itself a sufficient reason for supposing that the position of the tumour is in one part of the brain rather than in another.

In a word, there are many cases in which it is necessary to doubt the possibility of diagnosing the presence of a tumour in the brain, and many more in which, at present, it is altogether useless to attempt any minute diagnosis as to the position of the tumour. This much, however, is certain—that the person suffering from tumour of the brain is generally weak and exhausted from the pain, or want of sleep, or mental depression, one or all. The pulse is quick, weak, and irritable, fluctuating considerably in the twenty-four hours, and occasionally exhibiting a slight hectic

quickenings in the evening, particularly where the tumour has given rise to a state of chronic meningitis. Want of appetite, nausea, vomiting, constipation, may also accompany the hectic quickening of the pulse, but all these symptoms are connected with collateral conditions rather than with the tumour itself.

— Epileptiform convulsion is a common occurrence in tumour of the brain, particularly in the advanced stages of the disease. In the few days preceding death, indeed, convulsion may succeed convulsion, with scarcely any interval. The paroxysm may be violent and general, as in ordinary epilepsy, or it may be irregular and confined to the head and upper part of the body. In the general attacks the loss of consciousness is complete, and the after-stupor profound; but in the slighter and more partial forms, the consciousness is often partially retained, much to the distress of the patient, who greatly dreads those attacks in which there is consciousness of the sad struggle of which he is the subject. It is, indeed, the rule for the after-stupor to be much less marked in tumour than in simple epilepsy and chronic softening, except perhaps for a day or two preceding death, when the patient is lying, in all probability, in a moribund state. It is a rule, indeed, for the fit to be followed by depression and increased pain in the head rather than by actual stupor.

4. Workers in lead, or those who have been drinking water that has been poisoned by leaden water-pipes or cisterns, and a few who have taken lead for medicinal purposes, are sooner or later affected very injuriously. A general state of cachæmia is induced, the muscles waste and lose their irritability, and at the same time they become subject to tremor, spasm, convulsion, and, last of all, to paralysis. In this change the extensors are affected more than the flexors. This state of bodily decay is attended by marked failure of the intellectual faculties,

and when the brain is examined after death it is found to be harder, darker in colour, drier, more bloodless, and containing an appreciable quantity of sulphate of lead. A similar state of *induration of the brain*, without the presence of the lead, has also been met with in cases of epilepsy where there was no reason to suspect the presence of lead-poisoning, and some authors have gone so far as to say that the brain is always more or less indurated in epilepsy.

The convulsion, which is a common occurrence in this state, has nothing to distinguish it from ordinary epilepsy, or if there be anything it is only in its greater violence.

— These cases of induration of the brain are often spoken of as cases of hypertrophy of the brain. According to this view it is supposed that the brain has become harder because it had no room to expand, and the flattened state of the convolutions, a not unfrequent appearance, is pointed to as an argument in favour of this view; but it is more easy to believe that the proper tissue, instead of being hypertrophied, is actually atrophied, and (as Dr. Bucknill has pointed out in certain cases of insanity¹) that the enlargement of the brain may be owing to the growth of structures of an inferior quality. In children, however, there are undoubted cases of true hypertrophy of brain, where the bones have yielded, and the head has acquired an appearance which is very similar to hydrocephalus; but such cases are rare, and the patients so suffering do not appear to have had any other inconvenience beyond the deformity. Two cases of the kind are quoted by Dr. Watson.

5. *Atrophy of the brain* may be a congenital condition,

¹ A paper on the "Pathology of Insanity" in 'British and Foreign Review,' January, 1855.

or it may be brought on by pressure, as in the case of chronic hydrocephalus or tumour, by the obliteration of arterial trunks, as in those cases of arterial plugging to which attention was first directed by Dr. Kirkes, or by other means of a more recondite character. Where it is congenital, the wasting, or rather the want of development, is usually more marked in one hemisphere than the other, and in this case the opposite side of the body is shrivelled and some of its muscles contracted. In this case, also, the patient is generally both idiotic and epileptic. Of the other cases of atrophy our knowledge is not sufficiently positive to enable us to speak with any degree of precision.

6. *Congestion of the brain* is an affection which belongs to declining years rather than to early life. A person so suffering is less "bright" than he was, confused instead of clear in his ideas, wanting in the power of attention, drowsy, and having these symptoms aggravated by stooping or lying down. He suffers, also, from vertigo, dulness of hearing, dimness of sight, weight and aching in the head, lassitude, and other symptoms of the same kind; but not from any kind of delirious disturbance. His head and face are injected and dusky, his features torpid, his jugulars full, his pulse and respiration slow and laboured, his hands and feet habitually colder than his head, his bowels in all probability constipated. These symptoms may fluctuate a good deal, and at times pass off altogether; but, sooner or later, severer symptoms are brought on by some unusual bodily effort, as in straining or stooping, or by some strong mental or emotional excitement. These severer symptoms are similar to those which belong to cerebral hæmorrhage; in other words, consciousness is suddenly lost, and the body falls paralysed and senseless. These symptoms, indeed, only differ from those of cerebral hæmorrhage in that the consciousness partially returns,

and the paralysis and numbness partially pass off after the lapse of a few minutes. It is the rule for the paralysis to be general—that is, not confined to one side, and for none of the muscles to be rigid. It is the rule, also, for these symptoms to pass off in a short time without leaving any of the limited and permanent paralysis which is consequent upon hæmorrhage or softening. In a short time, indeed, and after a little increased dulness, headache, and muscular feebleness, the patient returns to his former state. Such is the usual course of the severer symptoms belonging to congestion, but it is not the constant course. In some instances, indeed, these symptoms may have an epileptiform instead of an apoplectic character, and the convulsion may be very violent. According to M. Durand Fardel, this attack is not ushered in by a cry, but the cry is no distinctive feature of ordinary epilepsy, as he supposed, and a cry was certainly present in several instances of epileptiform convulsion arising from cerebral congestion in a patient (æ. 53, whose first attack occurred twelve months before) recently under my care. It is also questionable whether it is anything like a constant rule for the after-stupor to be less persistent and less prolonged than in simple epilepsy.

7. Many of the persons in whom *apoplexy* is a probable danger are of a sanguine and plethoric constitution, corpulent, with large heads, short necks, and full chests; but there are many who have none of these characteristics. Nearly all are considerably past the meridian of life. Nearly all present various evidences of an oppressed or debilitated brain—such as headache, weight in the head, want of memory, want of firmness, vertigo, ringings in the ears, specks before the eyes, apathy, or irritability of temper.

The different forms of apoplexy are arranged in three classes. In the first, the patient at once sinks into pro-

found and deep coma; in the second, he gradually passes into this state after an attack of acute pain in the head, with faintness and sickness; in the third, he suddenly becomes paralysed on one side of the body, without losing his consciousness.

In the first class of cases the patient at once sinks into a sleep, from which no shouting, or pinching, or shaking, will serve to wake him; and in this state he lies like a person dead-drunk, with his face flushed, his pulse slow, strong, and labouring, especially in the carotids, and his breathing deep, slow, and snoring. If a limb be pinched, it does not stir; if it be lifted up and let go, it falls like a log. The eye is absolutely indifferent to light, and the dilated pupil remains motionless under the full glare of the sun. In this state of deep sleep the patient lies for some time, and then he slowly wakes to find his judgment damaged, his fancy dulled, his feelings unmanageable, and half his body benumbed and motionless. Or this dangerous sleep may deepen into the sounder sleep of death, in which case the flush on the cheek gives way to a ghastly lividity, the pulse becomes weak, frequent, and irregular, the respiration shallower and interrupted with frequent sighs and pauses, the skin dusky and bathed in cold and clammy perspiration, and, before long, death is ushered in, either with or without convulsion. In a case like this convulsion is not a common occurrence, and when it happens it is, as we have said, in the period of collapse preceding death, and not during the period of vascular activity which marks the beginning of the fit.

Or a person may fall down suddenly in this death-like sleep without this flushing of the face, and the insensible and unconscious body may lie, cold, pale, comparatively pulseless, without any loud and snoring breathings, and—whatever the issue of the attack, whether fortunate or unfortunate—with scarcely a single sign of vascular re-

action from the beginning to the end. It is also another mark of distinction between this form of the attack and the one just described, that convulsion may mark the beginning of the fit or that certain muscles may be seized upon with spasm. In other respects it is the same, and if convulsion happen at another time it is shortly before death.

In the second class of cases, the fit begins with sudden and violent pain in the head, confusion of thought, faintness, perhaps sickness. Instead of being flushed and hot, the face is pale and ghastly, the pulse weak, frequent, and irregular, the breathing shallow and interrupted with frequent sighs and pauses. Instead of being perfectly relaxed, the limbs are agitated, rigid, or convulsed, in which latter case the mental faculties are suspended for the time. Then, after a short time, the pulse rallies, the heat returns, the mind begins to reassert its sway, and even the power of walking may be recovered; but the headache, instead of passing off, becomes more and more oppressive, until it is forgotten in the feelings of confusion, drowsiness, and coma, which succeed presently—the interval between the first attack of pain and the occurrence of coma varying from a few minutes to several hours. Then the face again becomes pale and ghastly, the pulse fails, the respiration resumes its interrupted and sighing character, a cold and clammy perspiration breaks out all over the body, and the patient dies. In some of these cases death is ushered in by convulsion.

In the third class of cases, one side of the body is suddenly palsied and the power of speech destroyed, but without any corresponding loss of consciousness. The patient sees and hears and feels; thoughts also and memories arise in his mind; but he is stunned and bewildered. He only half apprehends the nature of the evil which has befallen him. In some cases, this state of things may

continue for some time with little or no change, and then the patient may sink, gradually from exhaustion, or more speedily from coma. In other cases the mind may regain a great deal of what it had lost, and the palsy may gradually pass off—a change in which the leg is liberated before the arm, and the power of feeling before the power of motion. In other cases, again, life is prolonged for a considerable time, and the only change is a state of contraction in the palsied muscles, of which more will have to be said presently. In cases like these there may be violent agitation at the beginning, but true epileptiform convulsion is not met with, except occasionally in the state of coma or collapse preceding death.

Now, hæmorrhage may take place into the substance of the brain, into the ventricles, or into the cavity of the arachnoid surrounding the brain, and different symptoms mark these different lesions, though as yet the nature of the difference cannot be determined with sufficient exactness. Premonitory symptoms, such as pain in the head, dulness, drowsiness—evidences of congestion—are said to be less common before hæmorrhage into the substance of the brain than before the other two forms of hæmorrhage. It is said that convulsion and muscular rigidity are much less common in hæmorrhage into the substance of the brain than in the other two forms of the disease. It is said, also, that coma is less profound and more slowly developed, and that paralysis is less general and complete, in hæmorrhage into the cavity of the arachnoid external to the brain than in hæmorrhage into the ventricles. At any rate, there can be little doubt that ventricular apoplexy is the most formidable form of the malady. But there is some uncertainty in these rules, and a review of many cases would rather seem to lead to the conclusion that the manner of the attack, the depth of the coma, and the extent of the paralysis, are determined

by the quantity of blood effused and the suddenness of the effusion rather than by the site of the mischief—at least, by the site simply. And certainly it is more than difficult to connect particular symptoms with injury to particular parts of the brain. Andral, for example, has disproved the idea that paralysis of the arm is specially connected with hæmorrhage into or upon the *corpus striatum*, and that paralysis of the leg has any necessary connexion with hæmorrhage into or upon the *thalamus opticus*; and Cruvelhier has shown very distinctly that loss of speech does not necessarily imply hæmorrhage into the anterior lobes.

— Now, epileptiform convulsion cannot be said to be a frequent phenomenon in apoplexy; but when it does occur, it occurs at the times and under the circumstances which have been mentioned. The most frequent time for its occurrence is in the period shortly preceding death. With respect to the convulsion itself, there is nothing at all peculiar.

— Under the head of apoplexy, perhaps, it may be convenient to allude to those cases in which epileptiform convulsion is caused by the depression of a portion of skull upon the brain. In cases of this kind the patient is left in a comatose state, which may easily be mistaken for apoplexy by a person unacquainted with the history, or who does not examine the head carefully; and if convulsion happen shortly after the injury, the circumstances are, in every respect, similar to those which attend upon the convulsion which may happen in apoplexy. If convulsion happen at a later period after the injury, the patient may have recovered more or less completely from the state of coma and paralysis; and the probability is that some diseased state, inflammatory or non-inflammatory, acute or chronic, is set up in the brain, and that the convulsion is symptomatic of this state.

8. *Inflammation of the brain* may affect the membranes or substance of this organ. As commonly used, the term meningitis is restricted to inflammation of the pia mater. It is restricted, that is to say, to the inflammation which is far more common, and infinitely more important, than the inflammation of the two other membranes—the arachnoid and dura mater. As commonly used, the terms cerebritis or encephalitis, meningo-cerebritis, phrenitis, are equally restricted to inflammation of the substance of the brain. Almost invariably, however, the membranes, as well as the substance, are affected, and very often, if not generally, the membranes are affected primarily. Indeed, it is very questionable whether the membranes can be affected without implicating the subjacent portions of the brain; and “it is not less questionable whether,” (I quote from Abercrombie,) “our knowledge is sufficiently matured to enable us to say *with confidence* what symptoms indicate inflammation of the substance of the brain as distinguished from that of the membranes.”

(a) *Meningitis* may be subdivided into three forms—the simple, the tubercular, and the rheumatic; and it will perhaps elucidate the history of the convulsion arising in this affection to observe this threefold division.

Simple meningitis is the form which happens in healthy individuals. It begins with the precursory symptoms of fever—rigors, paleness of skin, cutis anserina, headache, depression, confusion, drowsiness, vomiting—often vomiting repeated many times. Or, as Dr. Copland first pointed out, and especially in children, the initial rigors may be exaggerated into epileptiform convulsion. Then follow rapidly the symptoms of high febrile reaction—the pulse becoming hard and frequent, the breathing irregular and oppressed, the skin hot, particularly over the head, the face flushed, the eye wild and ferrety. Along with these changes, the headache of the initiatory stage gives

place to acute stabbing pain, and under these stabs the patient at times will shriek and scream from sheer agony. In some cases, a wild delirium takes the place of this acute pain, though this is doubted by Dr. Watson. All this time vomiting continues to be a distressing symptom. In this stage, which is called the first stage, the irritability is extreme, the pupil is contracted to the size of a pin's head, and the ear and eye are altogether impatient to sound or light. There may be strabismus; but in no case is there any marked prostration of strength. This stage continues for two or three days, and then the delirium calms down into quiet muttering, the headache ceases to be tormenting, or ceases altogether, and the morbid sensitiveness passes off. As the fever dies out, the pulse becomes weak, small, irregular, intermittent, at one moment slow, at the next frequent; and coincidently with this change in the pulse the respiration becomes irregular and interrupted with frequent sighs and pauses. As the flush leaves the cheek, the eye and ear cease to be impatient to light and sound, the pupil passes out of the contracted state, oscillates, and then becomes dilated and immoveable, the countenance puts on a ghastly and cadaverous expression, the limbs give up their warmth, the body drips with cold and clammy perspiration, the restlessness of the first stage gives place to muscular tremblings or twitchings, or (especially in children) epileptiform convulsion makes its appearance. Itself ushered in by the symptoms which have been mentioned, the convulsion may usher in a state of fatal comatose prostration, in which, without cessation, convulsion may follow convulsion until the end. Or, without the convulsion, the muttering following the early delirium may quieten down into coma, and coma into death—an event which is occasionally hastened by the exhausting involuntary motions which have taken the place of the obstinate constipation of the early stage of the malady.

When the meningitis affects the base of the brain, there is, as a rule, less fever, less delirium, a greater disposition to convulsion, and a more speedy development of coma; but it is not possible to insist very positively upon the significance of these differences.

Tubercular meningitis, or that form which happens in scrofulous subjects, and generally in scrofulous children, is for the most part insidious in its course, and the symptoms are at times extremely vague. The acute stage of fever, with its wild delirium, distressing headache, and impatience to light or sound, are altogether wanting. There is enough pain to banish refreshing sleep, and now and then, particularly at night, there may be a pang so sharp as to provoke a scream. There may also be a little light-headedness in the evening or at night. The pulse is very variable, now quick, now slow, rising in frequency when the patient assumes a sitting or erect posture, and falling again when he lies down; and in the evening there may be some disposition to faint hectic excitement. The respiration is irregular, unequal, sighing. If the disorder be unchecked in its course, indeed, the patient rapidly and almost silently sinks into a state of collapse and coma, and not unfrequently he is seized with epileptiform convulsion before we can bring ourselves to think that a serious disease is in progress. In cases like these, where the symptoms set in insidiously, convulsion would seem to happen less frequently at the commencement than in the period of collapse preceding death—at the time, that is to say, when the pulse is altogether without power, and when all cerebral action is being rapidly extinguished in coma—but it may happen at an earlier period. In any case, however, the convulsion is not associated with anything like feverish activity of the circulation. Indeed, the disease may be said to be without fever from beginning to end.

Rheumatic meningitis arises occasionally in the course of acute articular rheumatism, and the sufferers from it are generally weak and exhausted. The acute pain in the head, which is one of the symptoms, appears to have been translated from the limbs, for these limbs whose least motion had been attended with great suffering, are now freely moved about in all directions. Or, furious delirium may take the place of the pain. Indeed, the principal symptoms are acute pain in the head or furious delirium, followed rapidly, in the majority of cases, by drowsiness, coma and convulsions, paralysis and death. There is little febrile excitement from the beginning, and what little there is has died out before the occurrence of the convulsion; and there is as little evidence of inflammatory action in the membranes after death—indeed, such evidence is frequently wanting altogether.

(b) *Cerebritis* is divided into two forms—general and partial.

General cerebritis (encephalitis, meningo-cerebritis, phrenitis) is commonly mixed up with a greater or less degree of meningitis, and any active delirium, or acute pain, or feverish excitement, may be owing to this complication. Dulness, rapidly passing through drowsiness into coma, would seem to be the most characteristic phenomenon in uncomplicated cerebritis. Instead of the agonising pain of simple meningitis, the headache is dull, oppressive, deep-seated; instead of a morbid impatience, even in darkness and silence, the eye and ear are almost indifferent to light and sound; vomiting is a frequent and distressing symptom; and obstinate constipation is always present. The pulse, at first, is slow, but presently it becomes variable, and readily influenced by changes of posture; the respiration, also, is full of breaks and sighs, or it very soon becomes so. Each hour, indeed, the pulse loses in power, more sighs and pauses are mixed up in the

respirations, and after the last semblance of excitement has passed off, and when the deepness of the drowsiness shows that coma is coming hand in hand with death, then, in all probability, the frame is shaken with one or two bouts of convulsion.

Partial cerebritis (red softening, acute ramollissement) has little or nothing to do with febrile excitement. It is marked by gradually increasing dulness and drowsiness, by various paralytic symptoms, by cramps or permanent rigidity in some of the muscles; and it may be marked by dull, fixed pain in the head, with achings in the limbs, particularly in those that are paralysed, and occasionally, perhaps, by increased heat of head, with constipation and vomiting. In the course of the disorder epileptiform convulsion may frequently make its appearance; and when it does, it is very often preceded by a state of deepened dulness and drowsiness—a state indicating a greater or less degree of cerebral congestion. And this would appear to be the rule. At any rate, there is no reason to suppose that the convulsion is ever connected with the rare moments in which there is a partial break in the habitual state of feverless apathy and palsied helplessness.

9. The convulsion connected with *fever* arises at two distinct periods—at the onset and at the end of the disorder. That which happens at the onset is a common event in the fevers of children, particularly in smallpox; and it is an occasional event in the fevers of adults. Its antecedents, in all probability, have been failure of appetite, unrefreshing sleep, feebleness, want of spirit. Its immediate accompaniments are a sense of great feebleness or oppression, sickness or vomiting, paleness of the face, coldness of the hands and feet, cutis anserina, shivering or shuddering, headache, pain and sense of coldness in the back or limbs, a feeble, soft, and fluctuating pulse, a respiration short, accelerated, and interrupted with frequent

sighs. These symptoms continue for a time varying from one to twelve hours, and then coldness gives place to unnatural heat, and the pulse becomes full and bounding, or wiry and incompressible,—in a word, that change takes place which is known as the establishment of fever. Under ordinary circumstances rigor or shuddering is the extent of the muscular disturbance attendant upon the onset of fever; but in some instances the rigor is, as it were, exaggerated into convulsion, in which case the depression and oppression of the primary period of collapse have been more marked, the depression under these circumstances amounting to prostration, and the oppression to stupor. In a word, the convulsion which is occasionally attendant upon the onset of fever is as distinctly related to the period of collapse preceding the outbreak of fever, and as distinctly separated from the period of febrile reaction, as is the rigor of which it is the representative.

And so also with the convulsion which may come on towards the close of fever. The true febrile reaction is over. The pulse is weak and thready, the hand frigid, the body is already clammy with the sweats of death, the face is putting on the ghastliness of the grave, the muttering remains of a previous delirium are upon the point of dying out, and then—when the hand of death is already upon the heart, and the fire of life is nearly gray—the twitchings of the muscles may be exaggerated into convulsion. These are the circumstances attending upon the convulsion which may happen towards the end of fever.

10. Epileptiform convulsion is often brought about by the retention of some of the constituents of urine in the blood, and occasionally by the retention of some of the elements of the biliary excretion, and the history of the convulsion arising under these circumstances is not at all obscure.

— The constituents of *urine may be retained* in the

blood in Bright's disease of the kidney, in cholera and typhoid fever, after scarlet fever, and occasionally during pregnancy, as when the emulgent veins are pressed upon by the growing uterus. The causes of retention may, indeed, vary considerably; but, however different the cause, the system is brought substantially into the same state before the convulsion happens.

In a confirmed case of Bright's disease, where the history of the convulsion is perhaps defined most clearly, there are unmistakeable evidences of debility and emaciation, the skin is pale and puffy, and in certain parts, as about the eyelids and ankles, it pits on pressure. Appetite is wanting, ammonia is present in the breath, as may readily be seen by breathing on a glass rod which has been dipped in hydrochloric acid, a pale yellow fluid containing ammonia is occasionally ejected from the stomach, diarrhoea is, it may be, an urgent symptom, and the urine is scanty, loaded with albumen or casts, and wanting in a proper quantity of urea. The patient, moreover, is drowsy, stupid, listless, despondent, seeing and hearing with some difficulty, drawling in his speech, tripping in his gait, perhaps palsied in one or other of his limbs to a greater or less extent, and frequently suffering from annoying cramps. Each day these symptoms are aggravated, the drowsiness becoming more oppressive, the bodily strength more exhausted; and when this aggravation has reached a certain point, convulsion and coma make their appearance. This state of coma is peculiar in not being complete. The patient breathes stertorously, and, if let alone, appears to be dead to all within and around him, but he starts at a loud noise, or withdraws his hands if it be pinched or pricked, and for a moment the stertor ceases. Occasionally, also, he may partially wake out of this comatose condition, and delirious mutterings may show that the brain has resumed some degree of action. The stertor, moreover, is peculiar

in being set at a higher pitch than that which is heard in ordinary apoplexy. The epileptiform convulsions occurring under these circumstances are, as a rule, not very severe, and they often alternate with rigidity of the limbs, which rigidity varies in amount and position, and is often increased by any attempt to move the parts. These convulsions may recur several times: they go hand in hand with the coma; and, like the coma, they are connected with a state which differs very little from collapse—a state of which a pale and cadaverous face and cold extremities are among the signs.

— Epileptiform convulsion is not a common consequence of *retention of bile*, and where it does occur it is in a comatose or semi-comatose state, which is very similar to that which is brought on by retention of urine. At an earlier stage of the malady there may have been high fever and active delirium, but all signs of true excitement have passed off, and the condition is closely akin to collapse and coma when the convulsion happens. In a case of this kind the jaundiced skin will serve to point out the seat of the mischief.

11. The children which have any difficulty in cutting their teeth are undoubtedly delicate rather than strong. Their gums are swollen, tender, and painful; they are fretful and disposed to cry; their sleep is short and disturbed with starts, and they suffer in all probability from vomiting and diarrhœa as well as from want of appetite. A little later, when they have become exhausted by pain and want of sleep, when they may be wasted by want of food and diarrhœa or vomiting, and when the pulse is comparatively powerless, convulsion may happen. In some cases, however, the convulsions of dentition may be connected with a state of active cerebral inflammation or injection, accompanied by high fever; and here it will be found that the convulsion (observing the law which it

observes in cerebral inflammation and fever) occurs either in the initial stage of rigor, or in the period of collapse and coma which comes on after the state of feverish or inflammatory excitement has altogether passed off.

Where convulsion is referred to "irritation" in the alimentary canal, the blame is often thrown upon worms, and worms are often present. But if worms are not present there are always some other evidences of constitutional debility, bodily or mental. Very often the appetite is wanting, the breath offensive, the pulse quick, the bowel confined and teased with aches and gripes; and these fits of febrile disturbance, brought on for the most part by the presence of undigested food, leave the patient jaded, irritable, perhaps drowsy, emaciated, and liable to convulsion.

Nor is there anything peculiar in the history of the convulsion which is not unfrequently connected with menstruation, or pregnancy, or parturition, and of which "irritation" in the uterus is often regarded as a cause.

In epileptiform convulsion connected with the menstrual periods it often happens that the fit occurs at the end of these periods, when mind and body are alike exhausted by pain or loss of blood. It often happens, also, that the persons who suffer in this manner possess all the mental and bodily characteristics which belong to epileptic or hysterical patients, and that the attacks themselves have nothing to distinguish them from epileptic or hysterical attacks.

A similar remark may also be made with respect to the convulsion which occasionally happens during the course of pregnancy. For this convulsion may be either epileptic or hysterical; or it may be brought about by pressure upon the emulgent veins, in which case the general condition is analogous to that which has been pointed out when speaking of the convulsion connected with urinæmia.

And so also with the convulsion which occasionally happens during labour. It may be an attack of ordinary epilepsy or hysteria, or it may be brought about by loss of blood or by sheer nervous exhaustion. In the case of flooding, the face and tongue are blanched, the hand is frigid, the body bathed in cold sweat, the pupil dilated, the sight swimming and dim, the ear stunned with ringing and booming noises, the head racked with throbbing pain, the limbs incessantly tossed about, the trunk agitated with frequent shudderings and shocks, the stomach straining with constant efforts at retching and vomiting, the thoughts indistinct and in a constant whirl, the pulse fluttering or imperceptible, the breathing a continued sigh or gasp. With such accompaniments and after the last trace of mental action has died out, convulsion happens. In the other case, where convulsion comes on in the course of labour without flooding, the patient has been exhausted by the pain and effort; straining continually, apoplexy may be the result of the impeded circulation in the brain; or pain, delirium, drowsiness, coma, may sufficiently show the kind of mischief that is going on within the skull. Now it is in this state of drowsiness or coma, when all cerebral action is annihilated or upon the verge of annihilation, and when the bodily powers are upon the point of yielding in the struggle, that the convulsion happens of which mention is now made. The convulsion itself is often as violent as the worst form of epilepsy, and the attendant suffocation is often very marked; and in this respect it differs from the convulsion of flooding, where the convulsion is most generally less marked and less prolonged.

Nor is there anything peculiar in the history of the convulsion which occasionally arises in the fortnight following delivery; for, if this convulsion be not simply epileptic or hysterical, it is connected with the last stage

of puerperal fever, and the attendant symptoms are the same as those which accompany the convulsion happening towards the close of any fever.

And in those cases of convulsion which are referred to "irritation" of a sexual character, the same history is sufficiently obvious—mental exhaustion, accompanied by more or less bodily exhaustion, of which so much has been said already, the fits happening in or near the times of greatest exhaustion.

12. Convulsion is a frequent symptom where the *moribund state* is brought about by the loss of blood, and in this case the collateral circumstances are those which have just been described as occurring in flooding. Convulsion may also be present when death is caused, not by loss of blood, but by loss of the power which carries on the circulation, as in cases of "shock," or in poisoning by hydrocyanic acid and certain other poisons; and in these cases, the surface is cold, the circulation and respiration are almost at a standstill, and all mental action is altogether annihilated when the convulsion happens.

Convulsion, also, is the natural accompaniment of death by suffocation, whether sudden or gradual. When the lungs are suddenly deprived of fresh air, the face and neck immediately become "black and full of blood," the veins stand out like thick cords, the eyes appear to be starting from their sockets. In the first few instants there is an agonizing struggle for breath; in the instants following the symptoms are delirium of a pleasant character (so it is said), vertigo, loss of consciousness, convulsion, relaxation, death. The pulse, at first feeble, becomes stronger and fuller as the turmoil proceeds; and, at the time when the suffocation is well nigh complete, the main arteries may throb with considerable force. After this it fails rapidly; but it may be felt at the wrist for a short time after the patient is apparently dead.

In cases, where the suffocation is brought about slowly, as in pulmonary congestion or inflammation and in effusion into the pleura, the distressing efforts to breathe, the dusky or livid countenance, the giddy feelings, the delirious thoughts, are some of the symptoms which show the terrible prostration of the vital powers, and, when convulsion comes, it is hand-in-hand with fatal prostration and coma. In cases where slow suffocation is the effect of the suspension of the action of the great nervous centres, as in apoplexy, the distressing efforts to breathe, the giddy feelings, and the delirious thoughts are not present, but the dusky and livid countenance, and the slow, irregular, and stertorous breathings, show very plainly that the blood is in that semi-venous state which involves deep prostration of all the vital powers.

2. *The pathology of the convulsive affections which are characterised by epileptiform convulsion.*

§ I.

In a case of general epileptiform convulsion, as in a case of ordinary epilepsy, the state of the circulation is that which belongs to absolute suffocation, or else it is closely akin to that which occurs in collapse and syncope. It is as far as possible removed from anything like excitement.

There are also many reasons to believe that the circulation is tending to suffocation, or collapse, or syncope, when epileptiform convulsion is being brought about, and that there is something altogether uncongenial between this kind of convulsion and an active state of the circulation.

1. In *chronic softening of the brain* the circulation exhibits the signs of innate weakness which belong to advancing or advanced age—coldness of the hands and feet, a weakened and perhaps degenerated heart, athero-

matous and calcareous deposits in the arteries, and so on.

2. In *chronic meningitis*, as might be expected from the evidences of a scrofulous habit and from the state of weakness and exhaustion which are so generally present, the pulse, for the most part, is quick, weak, and much affected by changes of posture. There may be some hectic excitement in the evening, the cheeks flushing, the eyes shining, and the aching head becoming a little hotter than it was before; but this faint excitement is not sufficient to raise the pulse to a normal pitch of activity. In no case, indeed, is this reaction of the circulation a marked and conspicuous phenomenon, and in the majority of instances it is scarcely sufficient to impart even a semblance of power to the weak and feverless pulse. And if there be little vascular excitement in chronic meningitis, there is, if possible, less in the form of disease which is known under the name of *chronic hydrocephalus*.

3. In *tumour of the brain* the pulse is quick, weak, irritable, fluctuating, or, if not, it will be so as soon as pain, want of sleep, and despondency—common symptoms of tumour—have had time to bear their natural fruit of weakness and exhaustion.

4. In *induration of the brain*, such as is met with in lead-poisoning, &c., the phenomena presented by the circulation differ very little, if at all, from those which occur in advanced stages of simple epilepsy, and any difference there may be is one which indicates a state still more fully removed from fever.

5. In *atrophy of the brain*, as in simple epilepsy, there is no evidence of anything like excitement in the circulation.

6. In *congestion of the brain* the head and face are congested and dusky, the lips purplish, the jugulars full, the pulse and respiration slow and laboured, the hands and

feet habitually colder than the head. There are, indeed, many evident signs which show that the circulation is not carried on with proper vigour, and which appear to point to imperfect arterialization of the blood as one cause of this defect.

7. In *apoplexy* the convulsion is most apt to happen at the end rather than at the beginning of the period of coma, when the purpled lips and the inadequate breathings show that the respiratory changes are rapidly failing. Or, if it happen at the beginning, it is in those forms in which the condition of the circulation at the time is more akin to collapse than anything else, and not in those forms in which there is an excited pulse and strong determination of blood to the head.

8. In *inflammation of the brain* the condition of the circulation may vary a good deal with respect to the inflammation, but little with respect to the convulsion.

(a.) *Simple meningitis* begins with paleness of the skin, a feeble depressed pulse, cutis anserina, vomiting, rigors, perhaps convulsion. Then follow rapidly the symptoms of high febrile reaction and cerebral inflammation—the pulse becoming hard and frequent, the breathings irregular and oppressed, the skin, particularly the skin of the head, hot and burning. After continuing for two or three days, these symptoms of high febrile reaction and inflammation give place to an opposite state of things, in which the pulse loses its force and becomes weak, small, irregular, and the breathings are interrupted with frequent sighs and pauses. Or if at this time the pulse retain any degree of resistance, it is evident, from the dusky colour of the skin and the sighing and labouring respiration, that the whole of this resistance is not due to the injection of arterial blood into the artery. Now it is in this stage of collapse or semi-suffocation which follows, or else in the cold stage which precedes, the febrile and inflammatory

excitement, and never during the actual period of excitement, that the convulsion happens. And this rule is constant. Indeed, the history of simple meningitis shows most conclusively that vascular excitement is as incompatible with convulsion as it is with rigor or subsultus.

In *tubercular meningitis* the pulse is weak and variable from the very first, now quick, now comparatively slow, rising in frequency when the head is raised from the pillow, and falling when it is laid down again; and from the very first the respiration is irregular, unequal, and interrupted. For some time there may be a little disturbance, of a hectic character, particularly in the evening, but this soon comes to an end, and the prostrate pulse forgets to put on even this faint semblance of fever. In some cases, there may indeed be a short stage of fever, and something like active cerebral inflammation, especially in young children; but as a rule, the symptoms are altogether of a passive, non-febrile character. In any case, however, the convulsion is connected with a depressed state of the circulation, and never with febrile and inflammatory excitement, if such state there be.

In *rheumatic meningitis*, also, there is little or no febrile excitement from the beginning, and the pulse has become both feverless and powerless before the convulsion happens.

(b.) In *general cerebritis* the pulse, at first slow, soon becomes variable and readily affected by changes of posture; the respiration, also, is full of breaks and sighs. From the first, indeed, there is scarcely any fever, and little heat of head, except the phenomena of cerebritis are mixed up with those of simple meningitis; but if such symptoms be present, they soon pass off, and give place to symptoms of slow sinking—a state in which hour by hour the breathing is more interrupted with sighs and pauses, and the pulse more powerless, unless it may have a fictitious power from the difficult circulation of imperfectly arterialized blood, in which case the dusky countenance

and the purple lips will show very clearly what is the true state of the case.

In *partial cerebritis* there is even less febrile disturbance than in general cerebritis, and at no stage of the malady is there anything like increased vascular action.

9. The immediate antecedents of the epileptiform convulsion, which may attend upon the onset of *fever*, are paleness of the face, coldness of the hands and feet, a feeble, soft, and fluctuating pulse, a respiration that is short, accelerated, and interrupted with frequent sighs. The immediate antecedents of the convulsion which may attend upon the end of fever, are a weak and thready pulse, and a frigid hand—a state from which the febrile reaction has long since departed, and the hand of death is already upon the heart. The convulsion, indeed, takes the place either of rigor or subsultus, and, like these forms of muscular disturbance, it is associated, not with the state of febrile excitement, but with the very opposite.

10. The convulsion which may follow *retention of urine* is preceded by cold hands and feet, by a retarded and sighing respiration, by a weak pulse, or if at all otherwise, the evidences of defective respiration are sufficient to show that the pulse derives a fictitious strength from the difficult circulation of imperfectly arterialized blood. And so, also, with the convulsion connected with the *retention of bile*, for before the convulsion makes its appearance all evidences of high fever and active determination of blood to the head have died out, and the symptoms of positive sinking have taken their place.

11. In the convulsion connected with *dentition*, there may have been little or no previous fever, and by quick degrees the pulse may have become excessively weak. Or there may have been symptoms of cerebral inflammation with high fever, and after them a state bordering very closely upon collapse. In any case, the immediate ante-

cedents of the fit are indicative of great vascular depression—great vascular depression brought on slowly without any very obvious fever or determination of blood to the head, or else occurring before or after active fever and determination. And so likewise with that convulsion which is connected with *worms*, or other sources of “irritation” in the alimentary canal; for if there have been any febrile disturbance, this has died out, and left the patient, not only feverless, but pale and chilly. Nor is it otherwise with those forms of convulsion which are referred to “irritation” in the *uterus*. In the convulsion connected with menstruation, the circulation is as it is in ordinary epilepsy or ordinary hysteria; and a similar remark applies to several of the convulsions which may happen in the course of pregnancy. In the convulsion of flooding, the face and even the tongue is blanched, the hand frigid, the body bathed in cold sweat, the pulse fluttering and wellnigh imperceptible, the breathing a continuous sigh or gasp. In the convulsion occurring in labour, in which there has been no flooding, the head is often greatly congested, and the aëration of the blood is seriously interfered with, partly in consequence of the way in which the lungs sympathise with the semi-comatose brain, and partly because the regular expansions of the chest are interfered with by the convulsion and the constant efforts at straining. In such a case the pulse may be full, but if so, the venous colour of the lips will show that this fulness is chiefly due to the circulation of black blood. In the convulsion which may happen during puerperal fever, the vascular antecedents are the same as those which may happen towards the end of any fever. And lastly, the condition of the circulation before the convulsion which is referred to “irritation” of a sexual character, if it differ at all from that which is met with in ordinary epilepsy, differs only in being one of still deeper depression.

12. Nor is there any trace of vascular excitement before the convulsion which may happen in the *moribund state*. In the convulsion attending death by hæmorrhage or asthenia, the blanched face and tongue, the frigid hand, the sighing or gasping respiration, the faltering pulse, are signs which require no comment; and in the convulsion attending death by speedy or gradual suffocation, the state of things is equally opposed to the idea of vascular excitement, for how—to ask this question once more—can vascular excitement and a state of suffocation be compatible conditions?

— In a word, there is no instance in which epileptiform convulsion can be supposed to have any connexion with an excited state of the vascular system, and there are many instances in which the circulation is, as far as possible, removed from such a state; and the only conclusion which can be drawn from these facts is one which harmonises with the physiological premises and with the previous conclusions respecting tremor and convulsion.

§ II.

In a case of general epileptiform convulsion the mental faculties are suspended as completely as they are in epilepsy. The dilated pupil remains immoveable under the brightest light, the ear is deaf to the loudest noise, and when the patient recovers, if he do recover, his memory is an absolute blank as to everything that happened during the fit. In partial epileptiform convulsion, such as occurs not unfrequently in chronic softening and tumour of the brain, as in a few instances of partial epileptic convulsion, the mental faculties may not be altogether suspended, and the memory is occasionally able to recall some of the circumstances attending the fit. In the case of general epileptiform convulsion, therefore, the condition of the cerebral hemispheres is evidently one

of *inaction*, and not of action. Of this there can be no doubt. Nor can there be any doubt that the state of these hemispheres is one of comparative inaction in partial epileptiform convulsion, for the utter bewilderment and the inability to collect and control the thoughts are signs that cannot be misinterpreted.

There is also good reason to believe that the occurrence of epileptiform convulsion is favoured by a state of functional inactivity in the cerebral hemispheres, and antagonised by the opposite state.

1. In *chronic softening of the brain* the fits are preceded by unquestionable and often very marked impairment of the mental faculties, and in some cases the mind may be a total wreck. Fire and energy are dying out, and dulness and drowsiness point to the coming coma, of which they are the forecast shadows. The brain, also, is blighted, not inflamed. It is pallid, whiter than it ought to be, deficient in red spots, in parts softer than natural, and on microscopic examination this softened substance is found to consist of broken-down brain-tissue, with a greater or less number of cells containing oil, and sometimes reddened with blood-corpuscles (for hæmorrhage is a common consequence of softening), but without any of the products of true inflammation—exudation and pus-corpuscles.

2. Impairment of the mental faculties, progressively increasing, is also a prominent symptom in *chronic meningitis*—impairment which would seem to be more marked by peevishness, impatience, fidgetiness, and a disposition to delirium, than by dulness. The mind wanders a little in the evening or at night, and not unfrequently this wandering may settle down into insanity. Or there may be no positive symptoms of any kind. After death the principal sign of disorder is effusion of serum beneath the arachnoid or into the ventricles, and this is often the only

sign. In some instances there may be congestion of the pia mater, or evidences of tubercular degeneration in this membrane and in the contiguous parts of the brain, but, as a rule, the appearances are altogether negative. Indeed, in some instances, where the quantity of effused fluid is large, as in chronic hydrocephalus, the brain has a blanched, bloodless appearance, and the effused fluid is much less rich in solid constituents than the serum of the blood—a fact which is somewhat calculated to show that inflammation has had no share in its production.

3. In the majority of cases of *tumour* the intelligence does not appear to suffer in any very marked manner, and when it is otherwise the difference is owing, in some degree at least, to the presence of chronic meningitis. The pain, however, the want of sleep, the depression of spirits, all combine to exhaust the brain, and this exhaustion is shown by vagueness in the ideas, by inability to fix the thoughts, or in various other ways. Nor is the pain which is usually so very distressing a symptom an objection to the idea that the brain is acting inefficiently in these cases. In many cases, indeed, pain in the head is a sign that the brain is insufficiently supplied with arterial blood, for it ceases and gives place to delirium when the arterial injection increases. Nay, there is reason to believe that the nervous energy is *lessened* during pain, for M. du Bois-Reymond has shown (p. 86) that there is a considerable fall in the *nerve-current* of the ischiatic nerve of a frog when the cutaneous ramifications of the nerve are subjected to a treatment which must give rise to great pain. But be this as it may in other instances, it must be difficult to regard the pain as a sign of over-action of the brain in tumour of the brain, for the companion symptoms during life, and the appearances after death, are alike opposed to such a conclusion.

4. In *induration of the brain*, such as results from lead

poisoning, there is as little evidence of any excitement in the mental faculties as in epilepsy, probably less; and the condition of the brain after death affords no countenance to the idea of inflammation, for the brain is harder, darker in colour, drier, more bloodless than it ought to be.

5. In cases of *atrophy of the brain*, where the condition is congenital, the probabilities are that the patient is idiotic as well as epileptic. And in cases of hypertrophy of the brain, which cases are occasionally met with in children, while the bones are sufficiently yielding to allow the necessary expansion of the organ, the patients have not had any other inconvenience beyond the deformity—a faint argument, possibly, that want of brain, and therefore want of cerebral action, had really to do with the convulsion which would seem to be a constant phenomenon in atrophy of the brain.

6. A person suffering from *congestion of the brain* is less "bright" than he was, his conceptions are wanting in clearness, he is deficient in the power of attention and application, his sight is dim, his hearing dull and perplexed with rumbling sounds, he is drowsy, and feelings of weight in the head and pain are familiar troubles. Everything, indeed, indicates an oppressed and inactive brain.

7. In *apoplexy* the mental antecedents are those of congestion or softening, and not of inflammation as such. There would indeed appear to be a strange absence of inflammatory tendency in the brain in apoplexy, and if there be any evidences of inflammatory action around the clot, it will generally be found that this action was anterior to the hæmorrhage in point of time; that, in fact, the blood has escaped in consequence of a previously softened state of brain. It is possible, also, that an argument in favour of a tendency directly opposed to the idea

of inflammation may be found in the fact, pointed out by MM. Andral and Gavarret, that the blood in apoplexy is deficient in fibrine, for if the effect of inflammation be to increase the amount of fibrine contained in the blood, it may be supposed that a deficiency of fibrine is indicative of a tendency which is the reverse of inflammatory.

8. Nor is there the least reason to believe that any overaction of the brain is directly concerned in bringing about the convulsion which is connected with *inflammation of the brain*.

(a.) In *simple meningitis*, convulsion may attend upon the very onset of the disorder. In this case, it coincides with the cold stage which ushers in the true inflammatory reaction—a stage of which the mental signs are depression, confusion, perhaps drowsiness. Or convulsion may attend upon the period of final prostration which follows the true inflammatory reaction—a period in which the mind is rapidly sinking towards a state of coma. Convulsion may occur at one or other of these times, but it never occurs in the true inflammatory stage. It never occurs, that is to say, when the pupil is contracted to the size of a pin's-head, when the impatience of the eye and ear is scarcely to be quietened by absolute darkness or silence, and while there is agonising pain in the head or fierce delirium.

In *tubercular meningitis* the acute pain, the wild delirium, the intolerance of light or sound, which mark the outburst of simple meningitis, are wanting, and the course of the disease is insidious. In ordinary cases, where the symptoms set in stealthily, the usual period for the convulsion is after the brain and the system generally have given many unequivocal signs of exhaustion. In other cases, where there may be marked febrile disturbance, the convulsion may happen in the initial cold stage, or after

the febrile symptoms have calmed down and left the system in a jaded and exhausted state. As a rule, however, the process of true inflammation has as little to do with this affection as with phthisis pulmonalis; for when the diseased products are put under the microscope, they are found to consist, not of products of inflammation, but of the well-known elements of ordinary tubercle.

In *rheumatic meningitis* the convulsion observes the same rule, occurring either in the initial period of rigor or collapse preceding the accession of the violent pain and delirium, or else being deferred until the excitement has died out and left the patient drowsy and semi-comatose.

(b.) In *general cerebritis* anything like wild delirium, or acute pain in the head, is absent, unless it is complicated with meningitis, and the characteristic state is dulness and drowsiness, rapidly passing into typhoid prostration. In *partial cerebritis* the course of the disease is less rapid, and the downward progress may be interrupted by moments of mental excitement or aberration; but it is no less certain that the epileptiform convulsions, which may happen now and then, are connected, not with these moments, but with moments in which the habitual dulness is duller than ever. And there is nothing (this will soon appear), in the fact of permanent rigidity of the muscles to show that the brain is in a state of over-activity.

9. In *fever* convulsion may precede the establishment of the febrile excitement, in which case the mental state is one of great depression, oppression, prostration, stupefaction. In other words, it may occur in the initial period of collapse or rigor—the cold stage. Or it may occur in the final period of prostration, when a few incoherent mutterings are the only traces of the previous delirium, and when the last traces of mental action are rapidly succumbing to the drowsiness of approaching death. It may occur

at one or other of these times, but not during the active period of the fever—the hot stage.

10. In epileptiform convulsion depending upon *retention of urine* the patient before the attack is drowsy, stupid, listless, despondent, his eyesight dim, his hearing dull, his speech drawling; and in convulsion depending upon *retention of bile* delirium is at an end, and the drowsiness is wellnigh comatose before the time for the attack has arrived.

11. In difficult *dentition* the brain is exhausted by pain and want of sleep, and drowsiness has taken the place of fretfulness and wakefulness before the time for the fit has arrived; or if there has been any cerebral inflammation the fit follows the rule which has been already laid down. In *worms*, and in other forms of “irritation” in the alimentary canal, the mind, no less than the body, is not braced up to the proper pitch of health, and the patient is jaded, irritable, or drowsy. In the convulsion arising from “uterine irritation,” the mental state is that which belongs to either epilepsy or hysteria. In the convulsion of flooding, the pupil is dilated, the thoughts are undefined and incoherent, and before the tossings change into convulsion, the last trace of mental action has died out. In convulsion occurring during a labour, in which there has been ~~no~~ flooding, the brain is exhausted by pain and straining, and upon the point of lapsing into a state of coma at the time the convulsion happens. In the convulsion of puerperal fever the mental history is the same as in the convulsion of ordinary fever. And, lastly, the epileptiform convulsion of “sexual irritation” has the same mental accompaniments as ordinary epileptic or hysteric convulsion, one or other.

12. Nor can there be any doubt as to the condition of mind preceding the convulsion of the *moribund state*. In death by hæmorrhage or asthenia, mental action fails

pari passu with the flowing of the blood out of the vessels, or its stagnation in them, and the sufferer has become altogether insensible to pain and trouble before he is convulsed. And when death is brought about by suffocation, whether slowly or rapidly, it is no less certain that mental action fails as the respiratory changes fail, and that no single trace remains when the time for the convulsion has arrived.

— Interpreted from a mental point of view, therefore, the condition of the cerebral hemispheres, in and before general and partial epileptiform convulsion, is that which would seem to be necessitated by that state of the circulation which has been pointed out in the previous section. Nor is it possible to suppose that the state of complete or partial inaction, which is necessitated by the state of the circulation, should be limited to the cerebral hemispheres; for if the functional activity of an organ is in direct relation to the vigorous circulation of arterial blood in that organ, it follows, as a matter of course, that the functional activity of every nervous centre in the body must be at or near zero during the convulsive seizure, and in a state inclining in this direction in the interparoxysmal periods.

— In a word, the pathology of epileptiform convulsion would seem to be in perfect accordance with the premises, pathological and physiological.

3. *The treatment of convulsive affections, which are characterised by epileptiform convulsion.*

In cases where epileptiform convulsion is symptomatic of cerebral disease of a *chronic character*—chronic softening, chronic meningitis, tumour, induration, atrophy—the treatment called for would seem to agree in all its

main features with that which is required in simple epilepsy.

In none of these cases does there appear to be any good reason why the patient should be put upon short commons. In chronic softening, indeed, it may be supposed that the disease, which is essentially atrophic, will gain ground if the diet be scanty and insufficient. It is, of course, necessary to avoid the risk of vascular fulness and possible hæmorrhage, by taking care that the diet does not err on the side of liberality ; but of the two errors it would really seem that the patient is damaged less by a diet which is too liberal than by a diet which is insufficient. In chronic meningitis, a little abstinence may now and then be necessary, in consequence of slight fits of feverishness ; but it must never be forgotten, that the part which inflammation has to play in this malady, is infinitely less important than that which is played by degeneration, and that an evident scrofulous taint is scarcely ever absent. It must never be forgotten, indeed, that the general history of the disorder is such as to demand a generous diet, if any inference is to be drawn from the history of other diseases in which the same taint is manifested. The same remarks are applicable, also, not only to tubercle of the brain, but also to cancer, for it is certain that both these maladies are likely to progress most rapidly where the system is low and ill-nourished. In both these cases of tumour, indeed, as well as in other cases of the kind, even in tumour of an aneurismal character, the exhaustion arising from pain and want of sleep and mental depression would seem to constitute a special call, not only for nourishment, but also for a fair allowance of stimulants. Nor is it more easy to suppose that induration of the brain or atrophy are reasons for enjoining a low diet.

In other respects, also, it would seem that the remarks respecting the arrangement of the diet and the adjustment

of the habits, which were made when speaking of simple epilepsy, are still applicable to those cases of epileptiform convulsion depending upon chronic disease of the brain.

Nor is there any sufficient reason for supposing that any essential change is required in the general plan of treatment by medicines. In chronic meningitis, or in tumour of the brain, cod-liver oil may be the most suitable tonic; in tumour, moreover, the pain and want of sleep and depression of spirits may require the addition of morphia or of some other remedy of the kind; in induration of the brain from lead poisoning it may be necessary to follow out the plan suggested by M. Melsens, and give iodide of potassium before anything else can be done; in every case, indeed, there will be some special point which requires to be attended to; but there is no reason for supposing that the essential line of treatment will be different from that which has been laid down when speaking of simple epilepsy. And this remark applies to the treatment of the fit as well as to the treatment of the intervals between the fits.

— In cases where the epileptiform convulsion is symptomatic of disorder of an *acute character*, the practical question which must be decided before any other is whether the fit occurs at the beginning or at the close of the disorder. If at the beginning—as at the onset of fever or of certain forms of inflammation of the brain—little, in all probability, will have to be done, for the speedy establishment of the fever or inflammation will put an end to the convulsion, and prevent its recurrence for a time. If the fit occur at the close of the disorder, the treatment of the convulsion resolves itself into the treatment of the disorder. Now, according to the premises, convulsion in itself is not a reason for the adoption of antiphlogistic measures. On the contrary, it is a phenomenon which would be likely to be brought about by anything which would exhaust the brain and nervous system; and there-

fore a question arises, whether the occurrence of convulsion at the end of certain disorders might not be prevented in some degree, by more carefully husbanding the strength of the patient during the active period of the disorder.

— In the routine treatment of *congestion of the brain and apoplexy*, bloodletting, purgatives, cold to the head, mercury, blisters, figure conspicuously, and the cases are supposed to be quite exceptional in which such measures are not required, but it is difficult to assent to all that is taught and practised on this subject.

There are, no doubt, cases of congestion of the brain and apoplexy, in which the state of coma is unusually prolonged, in which the occurrence of epileptiform convulsion has left the veins of the head and neck in a state of great engorgement, and where it would seem to be the rational course to open a vein and let blood. The danger of apoplexy, or of renewed hæmorrhage, or the engorged state of the lungs, may indeed seem to necessitate such a course. At the same time, much may be done by the application of cold to the head, and by other measures, which will be considered presently; and it certainly may be a question whether bloodletting has any peculiar advantages over those measures. Nay, it may even be a question, whether bloodletting has any advantages at all. No doubt, there is enough of authority in favour of the lancet, but is there enough of reason? Is the theory sound? Is the practice sufficiently encouraging? These are questions which will be answered differently by different persons, and while many will answer unhesitatingly in the affirmative, others will have doubts, which will be expressed in acts, if not in words. If asked, indeed, they may perhaps deny the existence of their doubts, or speak as if they had none; but, in actual practice, the lancet will scarcely ever be out of its case. A great change, indeed, has already taken place, and what the end will be it is difficult to say. In

the mean time it would seem to be better to err on the side of doing too little, than on that of doing too much; and on this account, for my own part, I have always dispensed with the lancet, or any mode of bloodletting in cases of congestion of the brain or apoplexy. I have done this without what may seem to be good reason; indeed, I should find it difficult to cite any reason, unless such may be found in the change which has come over the habits of society and the doctrines of the schools. The habits of society are far more temperate than they were formerly, and the people, in consequence, would seem to have become less plethoric and less tolerant of bloodletting. At any rate, plethora is not a common characteristic of patients now-a-days. The doctrines of the schools are also changed or changing in one most important particular. Formerly, every disease was referred to inflammation, and the pathologist was unhappy if he did not discover the traces of this lesion after death; now, many diseases are referred to a process which is the very reverse of inflammation—degeneration, and, instead of bleeding, it has been found to be desirable to enrich the blood and promote nutrition. Nay, the idea of inflammation itself would seem to be undergoing a change, by which it is becoming less fiery or inflammatory, and more akin to the process which has just been named. At any rate, I have not been able to bring my mind to order bleeding in any of these cases; and so far as I am able to form an opinion upon the practice which has fallen to my share up to this time, I have never had occasion to suppose that a better result would have been brought about by a different line of practice.

There are times in every case where congestion of the brain or apoplexy is a danger to be apprehended, and these times will occur not unfrequently in those cases where the venous system is overloaded and the constitution thoroughly

debilitated, where nothing would seem to afford such immediate relief as mercury, and where the *modus operandi* of the medicine, in part at least, would seem to be that of a purgative. In order to produce this relief, however, it is by no means necessary to give the mercury in a quantity sufficient to cause it to act powerfully upon the bowels, or to follow it up by a black draught or other purge, and therefore the good results of this practice cannot be cited as an argument in favour of purging. On the contrary, the mercurial would often seem to do most good when it provokes the *kidney* to a natural degree of action. Indeed, I have seen several cases in which the increased action of the kidneys would seem to have had a much greater share in relieving a congested state of the venous system than any increased action of the bowels. In cases where the evil has gone further, and the patient has actually been struck down by coma, it may be necessary not only to resort to purgative enemas, but to purge by putting croton oil upon the tongue, or in other ways; but even here it can scarcely be said that violent purging is called for.

It is somewhat surprising that *diuretics* have not had a more extensive trial in cases of threatened coma from congestion of the brain, for it might be supposed that remedies of this class would have the power of reducing the quantity of the blood with little or no inconvenience. Indeed, it is only possible to account for this apparent omission by the fact that diuretics are less certain in their action than purgatives. It is not mere theory, however, which would point to diuretics under these circumstances. On the contrary, it has long been the practice of my friend and late colleague, Dr. Hamilton Roe, to give tincture of cantharides in cases of threatened congestion of the brain, with a view to lessening the amount of circulating fluid and relieving the congested veins, by rousing the torpid kidney to a freer action; and in the cases in which I have

had an opportunity of trying this plan of treatment upon my own responsibility, I am able to corroborate the favorable opinion entertained by Dr. Roe. The plan is to give fifteen minims of the tincture in a little mucilage every hour for three or four doses, and the usual result is free secretion of urine and unmistakable relief to the state of venous congestion, without any of the feelings of depression which so commonly arise from the free action of purgatives. It is not improbable, also, that the quality of the blood may be changed for the better by this freer action of the kidney, for it may be supposed that the increased secretion of urine involves a freer elimination of matters which cause great depression and prostration when retained in the blood—urea and products allied to urea. Where the symptoms of threatened coma are less urgent, colchicum may, perhaps, prove to be a more suitable remedy than cantharides. At any rate, I have often seen the kidney excited to freer action, with unmistakable relief to the state of venous engorgement, by giving for two or three days a few doses of this tincture, generally in association with sweet spirits of nitre, or by giving a pill containing two grains of the extract of colchicum and one grain of blue pill for two or three nights successively.

Of the importance of applying *cold to the head* there can be no doubt; indeed, this measure may be said to be the natural mode of affording relief in cases of congestion of the brain. If the head be raised, and a stream of cold water poured upon it, particularly if warmth be applied to the feet at the same time, it is indeed difficult to imagine a state of coma which will not yield; and if cold be applied judiciously afterwards, it is as difficult to believe that the coma will not continue in abeyance. At any rate, a state of coma which is manageable in any way may be managed in this way. It would seem, also, that the

great difficulty which has always prevented the full realization in practice of the theoretical advantages of cold to the head—the difficulty of applying it for a sufficient length of time, with steadiness, without wetting the patient, and without requiring the whole attention of the nurse—has been overcome by a plan recently proposed by Dr. James Arnott—a plan which for the first time promises to make cold a manageable and available agent in the treatment of disease. “A current of water of the appropriate temperature is made to flow through a thin waterproof cushion in close contact with the body. The water runs into the cushion from a fountain reservoir raised above it, through a long flexible tube; and again, escaping from the cushion, it passes through another tube into the waste vessel. The cushion is of a size and form adapted to the part of the body in which the water is to act; and, by a particular contrivance, any pressure from its weight may be prevented. The part in contact with the cushion is kept moist, either by previously wetting the cushion or by interposing a piece of wet lint, flannel, or other bibulous substance.” Speaking of this apparatus, Dr. Watson says that it promises to be an essential auxiliary to the lancet and cupping; but this, I take it, is not all that may be said. I take it, indeed, that this apparatus will be an essential agent in the treatment of all affections of the head where active inflammation has to be subdued, or where congested veins have to be unloaded, and that it will be a source of great comfort where the sole object is to dispel a feeling of weight or discomfort in the head. I can, indeed, imagine a time when a modification of this apparatus, within the reach of all, may enable any aching and wearied head to enjoy the luxury of a douche or wet towel without the inconvenience of wetting the hair or deranging the dress.

It is not possible that the body or mind can be exerted

beyond moderate bounds, or the appetites indulged with impunity, where there is a predisposition to congestion of the brain. At the same time it is very possible to err on the side of abstinence, and the necessity for putting the patient on very short commons may well be called in question. It is very desirable to keep down the quantity of the blood, and in order to this it will be of prime importance (seeing that there is often an inactive condition of the kidney) to stint the quantity of sloppy drinks; but it can scarcely be desirable to run the risk of impoverishing the blood, if, as the researches of MM. Andral and Gavarret would seem to show, there is actually less fibrin in the blood of apoplectic patients than there ought to be. In a word, the history of congestion of the brain would not seem to furnish a sound warrant for carrying out what is usually known as the antiphlogistic plan of treatment.

— It would seem, also, that a somewhat similar line of remark is applicable to the treatment of *inflammation of the brain*.

There are cases of inflammation of the brain in which bloodletting may seem to be called for by the intensity of the febrile and inflammatory symptoms; but even in these cases it is difficult to carry out this plan of treatment without some misgiving. For if, as Professor Bennett maintains,* the inflammation in which bloodletting was thought to be most indispensable—inflammation of the lungs, may be treated satisfactorily without bloodletting, may it not be so also with inflammation of the brain? Now it appears, from the investigations of Dr. Bennett, that the result of the rigorous antiphlogistic treatment of pneumonia, as practised formerly in the Edinburgh Infirmary, the Charité Hospital at Paris, and in several other hospitals, is a mortality of 1 in 3; that the result of the

* 'Edinb. Medical Journal,' March, May, June, 1857.

treatment by tartar emetic in large doses, as practised by Rasori, and more recently by Dietl, is a mortality of 1 in 5, or, according to Laennec, of 1 in 10; that the result of moderate bleedings, as in the practice of Grisolle, is a mortality of 1 in 6.50; that the result of a dietetic treatment, with occasional bleedings and emetics in severe cases, as carried out by Skoda, is a mortality of 1 in 7, or, if purely dietetic, as under Dietl, of 1 in 13. It appears, also, that the mortality from pneumonia in the British army, where the malady for the most part has arisen in healthy able-bodied men, is also 1 in 13. And, lastly, it appears that the mortality has been reduced to 1 in 21—to one seventh, that is to say, of the mortality of twenty years ago—under the treatment pursued by Professor Bennett, during the previous eight years, in the Royal Infirmary of Edinburgh. In this practice, no attempt is made to cut the disease short or to weaken the pulse and vital powers, and the sole aim is to facilitate the necessary changes which must take place in the inflammatory exudation before it can be excreted from the system. To this end salines are given in small doses during the period of febrile excitement, and, as soon as the pulse becomes soft, these are changed for good beef-tea and other nutriment. When there is evident weakness, from four to six ounces of wine are allowed daily; and as soon as the period of crisis approaches, the excretion of urates is favoured by giving, three or four times a day, a diuretic, consisting generally of half a drachm of nitric ether, with a few minims of colchicum wine. Or if the crisis occur by sweating or purging, care is taken not to check it in any way. The question, no doubt, is one of considerable difficulty, and much remains to be proved before an unchallenged answer can be hoped for; but this much is plain, that bleeding and other severe antiphlogistic measures have not been shown to be less necessary now than formerly because

inflammation has assumed a more asthenic character, and that authority, however high, must be disallowed, if in such a matter it does not rest upon something more stable than mere precedent. And, if the necessity for bleeding may be called in question in inflammation of the lungs, where the diminution in the respiratory capacity of the lungs would seem to demand a corresponding diminution in the amount of the circulating fluid, it may well be doubted whether bleeding can be regarded as an essential measure in inflammation of the brain. In an organ so delicate as the brain, it is, no doubt, of the highest moment to check inflammatory action as soon as possible; but it is no less important to preserve those reparative powers by which the mischief is to be repaired subsequently, and this the more, seeing that a scrofulous taint may often be detected in inflammation of the brain, and that this inflammation under any circumstances is more akin to degeneration than inflammation elsewhere.

Nor is it easy to assent to the routine practice respecting purgatives. In many cases of inflammation of the brain, the best results will follow the use of purgative enemata at the commencement of the affection, and it will be well to persevere in their use until all effete matters are brought away; but the beneficial results of swallowing purgatives from the beginning to the end are not quite so certain. At any rate, an experience extending over several cases has served to show that there is less sickness where the purgatives are withheld, except in the form of occasional enemata, and that, to say the least, the progress of the case is not less satisfactory.

There are, no doubt, many difficult points to be attended to in the treatment of inflammation of the brain, but much may be done with cold to the head, with a little clear ice, with mercury, with colchicum, with chloroform, if care be taken to keep the sick-room dark and quiet, and the

patient himself in a semi-erect posture. Cold to the head is an all important measure, and if much could be done by means of wet rags and bladders containing ice, we may fairly expect that more will be done by means of the apparatus of Dr. James Arnott, already referred to. Applied in this manner, indeed, we may even expect to use cold so as to vanquish any amount of inflammatory action. A little clear ice is also of great service to allay the sickness and quench the thirst. Indeed, a morsel of ice, sucked now and then, will answer these indications better than any kind of effervescing draughts. While the symptoms are at all active, mercury, in all probability, is indispensable, and the most convenient form of giving it will be that of a pill. Two or three grains of calomel in a little conserve, by themselves, or associated with a small quantity of extract of colchicum, and repeated every six hours, may indeed be all the medicine wanted. It is useless, perhaps hurtful, to want to go too fast, and more than enough has been given if the mercury salivate the patient or act violently upon the bowels. By these means, if care be taken to keep the head high, and the sick-room dark and quiet, it is not likely that the inflammation can hold its ground long. Nor is it likely that the pain or delirium should refuse to yield, but if not, a few whiffs of chloroform will generally be very serviceable.

The difficulty, however, is not in mastering the inflammation; it is in knowing when to have recourse to restoratives. The question is—are these measures only to be tried, and then very cautiously, as is recommended on high authority—“when an extreme degree of collapse occurs?” Surely not. Surely no theoretical considerations respecting inflammation can justify a plan of treatment which will allow the very faintest degree of sinking to show itself, without at once taking steps to supply food and other restoratives? Surely it cannot be in accordance

with any sound rules of physiology or pathology to persevere with antiphlogistic measures until it is dangerous to go on with them any longer, and then at once and suddenly to have recourse to ammonia, Hoffmann's anodyne, beef-tea, wine, and so on?

— If a rigid antiphlogistic plan of treatment be not necessary in inflammation of the brain, it is not likely to be necessary in fever. But, be the treatment of the earlier stage of fever what it may, there can be no doubt that an energetic restorative and stimulant plan of treatment has become necessary when the occurrence of subsultus shows that convulsion is not an unlikely danger.

— Where convulsion has resulted from a suppressed state of the renal and biliary secretions, the patient, in all probability, is sunk in a state of collapse from which there is scarcely any possibility of rousing him, and the local disease, of which the suppression is a sign, is altogether beyond the reach of art; and, therefore, it is of little moment what is done.

— In convulsion depending upon "irritation" in the gums or alimentary canal, the cue as to treatment will have to be taken from the degree of fever and the condition of the circulation in the brain. If there be anything like inflammation in the brain, the remarks which have been already made on this subject are applicable; and the existence of any special cause of "irritation," in pointing out an additional cause of exhaustion, would only offer an additional objection to bleeding and purging. Cold to the head, repeated warm-baths, a dose or two of gray powder, lancing the gums or removing any carious teeth, if this be necessary, giving turpentine enemata if worms or effete matters have to be brought away, with beef-tea from the beginning, and wine and other restoratives very soon after the beginning—such are the measures which would seem to be necessary. Where convulsion is connected with

“irritation” in the uterus, or in the sexual apparatus, some special local measures may be required; but there is no reason for supposing that the general treatment will be different from that which has been pointed out when speaking of epileptic and hysteric convulsion, except in this—that the very presence of the “irritation” would seem to be an additional reason for distrusting any measures of a lowering character. Where convulsion arises in connexion with puerperal fever, the circumstances are for the most part analogous to those belonging to the convulsion connected with ordinary fever, and the treatment must also be analogous. With respect to the convulsion of flooding, only one course can be proper, and that is to keep the patient alive by wine, transfusion of blood, and so on. Indeed, there is only one case under the present head in which the history of the convulsion would seem to demand a different treatment to that which has appeared to be necessary up to this point, and this is the convulsion occurring in labour without flooding. Now, in this case, the head is often greatly congested, and the aëration of the blood is seriously interfered with, partly as a consequence of the way in which the lungs sympathise with the suffering brain, and partly because the regular movements of the chest are greatly interfered with by the convulsion and the constant straining. Now, if bleeding can be necessary in any case, it must be in this, for these constant strainings are continually putting the patient in danger of fresh convulsion and apoplexy, by the way in which they interfere with the respiratory movements and prevent the free return of blood from the brain. At the same time, it may be well even here to procure a state of artificial rest by means of chloroform vapour, and to take some steps for relieving the uterus of its burden, before having recourse to this measure.

— And, lastly, it is scarcely to be supposed that there is anything in the history of the epileptiform convulsion connected with the moribund state which can invalidate the conclusion which arises uniformly out of all the previous considerations.

CHAPTER V.

THE CONVULSIVE DISEASES WHICH ARE CHARACTERISED BY SPASM.

THE third category of convulsive diseases is characterised by prolonged muscular contraction or spasm. It includes catalepsy, tetanus, cholera, hydrophobia, ergotism, the rigidity of cerebral paralysis, the spasm connected with certain diseases of the spinal cord, and certain minor spasmodic disorders.

1. *The history of the convulsive affections which are characterised by spasm.*

1. In *catalepsy* the muscles are rigid and slightly contracted, and the patient retains the expression of countenance and the posture which he had before the seizure. The muscles also are pliable, and the limbs remain in any position in which they may be placed. The appearance during the attack is that of a corpse, and the condition is only one short degree removed from that of a corpse. In some instances, however, the muscular rigidity is less marked, the corpse-like sleep less profound, the pulse less imperceptible, the face and head less pale and cool than the rest of the body; and, in other instances, there may be less activity in the respiration than in the circulation, and, as a consequence of this, the veins of the head and neck may stand out somewhat more distinctly; but usually the state is one which may easily be mistaken for that of a corpse.

2. The spasms of *tetanus* begin in the muscles of the

face, and give to the features a drawn and aged expression; and then, extending to a wider range of muscles, they lay hold, first, upon the neck, jaws, and throat, and afterwards upon the limbs, the trunk, and perhaps the diaphragm. In the height of the disorder the eyeballs may be firmly fixed, and the tongue stiff and immoveable. Sometimes all muscles are affected equally; sometimes certain groups are affected more than others, in which case the jaws may be locked and the rest of the body free, or the body may be bent backwards, forwards, or sideways, as the case may be. The spasms occur in paroxysms, without any perfect remissions, except during sleep. At first the surface of the body is of the natural heat and colour, but as the malady progresses the temperature falls and the skin becomes drenched in perspiration. The respiration, never free, becomes more and more laboured as the spasm gripes with firmer hold upon the respiratory muscles, and towards the end there are moments in which the struggle for breath is agonizing. The pulse, never excited by the least semblance of fever, soon becomes feeble and frequent, except during the moments of unusual difficulty of breathing, and then the colour of the skin shows very clearly that any increase of fulness is not altogether due to the injection of red blood into the arteries. The reflex incitability of the system is greatly increased, and ordinary impressions on the senses are sufficient to bring on a paroxysm. As to the rest, there is, with very few exceptions, excruciating pain in the cramped parts, at the pit of the stomach, and in the wound or cicatrix, if such there be; and, lastly, there is no stupor, except towards the end, when the action of the brain has begun to suffer from the circulation of imperfectly aërated blood. The causes of this sad malady are not at all obvious. Wounds, no doubt, are a most important cause, and in some instances these would seem to have acted by depriving the patient

of blood, by the shock to the system, or by the natural depression resulting from the thoughts of danger, or of a maimed and helpless future; but in other instances the history of the case presents nothing so obvious, and it is necessary to be content with the supposition that the wound has set up a state of inflammation or morbid incitability in a nerve, and that the propagation of this state to the incito-motory nervous centres is the cause of all the evil. Wounds, however, are not necessary to the production of tetanus, and wounds, in all probability, are never of themselves sufficient. At any rate, it is the opinion of army surgeons that tetanus is most apt to occur when soldiers are dispirited, exhausted, ill fed, and exposed to cold.

With respect to the appearances after death, some very valuable information is presented in a recent report, by Mr. Poland, of 72 cases of tetanus which occurred in Guy's Hospital between 1825 and 1857.* Thus, of 20 cases in which the *brain* was examined, this organ was found to be healthy in 11, congested in 4, darker than natural in 1, dark and flabby in 1, pinkish in 1, ulcerated on the under surface of the anterior lobes in 1 (the result of injury to the head co-existing with the tetanus), decomposed in 1 (an acute case, fatal in four days, the examination being made forty-one hours after death, and the muscles still remaining rigid). Of 19 cases in which the spinal cord was examined, this organ was healthy in 8, firm, rigid, and of pinkish hue in 1, of natural firmness but injected in 1, congested in 2, darker than natural in 1, pinkish in 2, of a "higher tint" in 1, softened in 1, decomposed in 1 (the case in which the brain was decomposed). Of 14 cases in which the nerve at the seat of the wound was examined, the nerve was healthy in 3, inflamed in 5, united after division in 2, the end bulbous in 1, the

* 'Guy's Hospital Reports,' 3d series, vol. iii, 1857.

nervous twigs entering the cicatrix pinkish in 1; and, of the remaining 2, the filaments of the facial nerve were spread over the ulcer, but their condition not stated, in 1, and in the other, in which tetanus followed amputation through the thigh, the divided sciatic nerve lay exposed to the extent of two inches, with granulations around and over it, but the nerve itself apparently healthy. Of the appearances in other organs nothing need be said.

Under the present head, moreover, it may be well to notice certain anomalous affections which are not remotely akin to tetanus, which are too variable in their characters to admit of being comprehended in a general description, and which are well illustrated, both as to characters and causes, by a case which is quoted by M. Romberg from a paper by Mr. Mitchell in the 4th volume of the 'Medico-Chirurgical Transactions:'

CASE.—"A female, *æt.* 50, was suddenly attacked with spasms of the facial muscles and tongue, which, after the lapse of a fortnight, extended to the neck. The paroxysm commenced with a sense of weakness and oppression at the *præcordia*, and a violent shooting pain passing from the sternum to the spine, rising upwards to the tongue, which then became as stiff as a piece of wood, bending the point upwards to the left side of the arch of the palate. A sense of numbness attacked the left side of the nose and the chin. The left angle of the mouth was opened and distorted, the teeth were closely compressed, all the muscles of the face became rigidly contracted, the nose was drawn over to the left side, and the forehead and eyebrows were corrugated by the spasm of the occipito-frontal and corrugator supercillii muscles. The muscles of the neck rotated the head to the left shoulder, the left arm became extended, and a sense of numbness ran down in a straight line from the neck to the thumb and forefinger. Consciousness and the action of heart and lungs continued unaffected. After three minutes there was a remission, commencing with a tremor of all the affected muscles. These paroxysms returned day and night at intervals of ten minutes. As the treatment pursued produced no effect, another physician was consulted, who had seen a similar case of facial and lingual spasm cured by the extraction of a carious tooth; on examining the teeth of this patient, though she did not complain of toothache, one tooth was found in the upper left row to be in a morbid condition and sensitive to the touch. The gum was inflamed, and a fetid matter discharged. After the first molar was extracted,

and the gums had been scarified, the paroxysms diminished in intensity and frequency, and they entirely ceased after the extraction of all the carious teeth."

3. The cramps of *cholera* begin in the alimentary canal, and extend successively to the abdomen, thighs, legs, chest, arms, and hands, and, once established, they continue, with few intermissions, until death. The surface of the body is cold, clammy, and blue. The pulse rapidly becomes imperceptible. The respiration is laboured and panting, and the breath cold. The sense of pain and suffering is greatly blunted, for the mental energies have succumbed to the blow which has prostrated the bodily powers.

4. The spasms of *hydrophobia* occur in paroxysms, which increase in violence and frequency as the malady progresses. They begin in suffocative and strangulatory contractions of the muscles concerned in respiration and deglutition; then they extend to the limbs and trunk; and, eventually, they may pass inwards and seize upon the bladder and intestines. In some instances, the disorder has been mistaken for tetanus. In the intervals there is the greatest inquietude and restlessness, and every voluntary movement is hurried, impulsive, almost convulsive. Occasionally, there is unceasing tremor and tremulous agitation. The hands and feet are cool and cold, and so is the surface generally, though in a less marked degree. The pulse is quick and feeble, and the respiration quickened and often interrupted by sobs and sighs. The mental state is one of fear or even despair, with occasional outbursts of delirious violence, in which there may be a tendency to bite others. There is more or less pain in the wound or cicatrix, and—what is far more distressing than any mere pain—there is an abiding sense of suffocation as from some impediment in the throat. The most marked and distressing symptom, however, is the excessive irritability of the whole system, and the extreme facility

with which a paroxysm may be brought on by the most insignificant cause. A gust of air, a beam of light, a sudden noise, a single touch, may be sufficient for the purpose. The gullet is not less sensitive; and because drinking, or any attempt to drink, provokes the paroxysm, the patient dreads to drink, though he does not fear the water, as the name of the disease would imply. All the secretions appear natural, except the saliva, which is viscid and abundant. This secretion is a source of great distress to the patient, for it cannot be swallowed or expectorated without great difficulty, nor can it be rinsed away, for the contact of water with the throat brings on the paroxysm.

The appearances after death in 46 cases, the histories of which were carefully analysed by my brother,* teach very plainly that "the disease may be fully developed, run its course, and terminate fatally, without leaving any appreciable lesion of structure, that the lesions which are observed in certain cases are not constant," "that no satisfactory link of connexion can be found in many, if in any, instances between the appearances observed after death and the symptoms noted during life." "In the majority of cases, however, there were indications, more or less marked, of morbid action in various organs. Thus, morbid appearances were found in the dura mater in 8 cases, in the arachnoid 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 medulla oblongata and pons Varolii in 4, in the cerebro-spinal and dsympathetic nerves 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 œsophagus 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

* J. N. Radcliffe, 'Lancet,' September, 1856.

lesions consisted of every grade of injection of the blood-vessels, from the slightest blush to the most vivid, or dark, black congestion; of alterations in the consistency of the tissues, principally softening; of effusion of blood, and certain products of perverted nutrition and secretion. 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 the lesions were of that character which is found in structural changes occurring in asthenic conditions of the system."

5. The cramps of *ergotism*, according to M. Romberg, who borrows his description from the accounts supplied by MM. Wichmann, Taube, and Wagner, occur in the following manner:—"The feet and hands are attacked with cramp in the flexor muscles. The fingers of both hands are bent like hooks, the thumb being pushed under the fore and middle fingers in an oblique direction, the wrist is strongly curved inwards, so that the hand assumes the shape of an eagle's beak. The toes are also doubled under the foot. The spasm extends over the fore and upper arm, which are bent one upon another at an acute angle; it also extends over the thigh and legs, and over the back of the neck and jaws." These symptoms end either in tetanus or epileptiform convulsion. The skin is dull and dry, except during the paroxysm, when it is perspiring. The pulse and respiration are affected in the same way as in tetanus. All the senses are considerably dulled, and that of feeling is well-nigh annihilated. The cramps are accompanied with pain, which is generally relieved by extending the cramped parts. The intelligence, not sensibly affected at first, fails as the disease proceeds, and before death the state may be almost one of fatuity. As the disease proceeds, also, paralysis may supplant the cramps. If life be prolonged sufficiently, a state of slow

mortification is set up in the extremities, and the fingers and toes, or even the hands and feet, may have disappeared before death. The main cause of this malady is the habitual use of grain affected with the ergot disease, and a residence in the low, malarious, damp districts in which this disease takes its origin.

6. The *rigidity of cerebral paralysis* may come on at the time of the cerebral attack or shortly afterwards, or it may be deferred for a while; and this difference in the time of occurrence, as Dr. Todd has very well pointed out, is a very important difference.

(a.) In the "early rigidity," or that which comes on at the moment of the attack or shortly afterwards, Dr. Todd indicates two varieties—one in which the rigidity is slight and confined to one or two muscles, the other in which the state is co-extensive with the paralysis. Where the rigidity is slight and partial, it may only appear when the muscles are put on the stretch. Upon moving the arm, for example, the biceps may become stiff and rigid, and prevent perfect extension of the limb, or the triceps may contract in the same manner and prevent perfect flexion. This rigidity is more commonly manifested in the flexor muscles than in the extensors, and most of all in the flexors of the arms and hands: it is rarely met with in the muscles of the face. In its most marked degree it is firm and constant. In these cases, the nutrition of the muscles is not materially damaged at first, and there is little or no wasting; but after a while, if the palsy continue, the muscles waste away, though never so rapidly as in those cases of paralysis in which the muscles are loose and flabby from the beginning. At first, also, the circulation in the part is vigorous, the heat is maintained at the proper standard, and the muscles are as sensitive to the galvanic current as they were before the paralysis, or even more sensitive.

(b.) "Late rigidity," or that form which does not occur for some time after the paralysis, may seize upon those muscles which were left by the paralysis in a lax and flabby state, or it may supervene, with or without any interval, upon "early rigidity." It never happens until the paralysed muscles have wasted considerably; it is established by slow degrees; and where it is perfect, the wasted muscles are stretched like tense cords between their points of attachment. It agrees with "early rigidity" in its preference for the flexor muscles, particularly for the flexors of the upper extremity; and when it is most marked in this latter part, the fore-arm may be tightly flexed upon the upper arm, and the fingers as tightly bent into the palm. In this form of rigidity the muscles are always wasted, and they may be reduced to mere membranous shreds. And, as might be expected from this state of wasting, the circulation in the paralysed parts is very feeble, and the heat very imperfectly maintained. It would seem, also, that the muscles have ceased to respond to the influence of galvanism before they pass into this state of rigidity. At any rate they do not respond to this influence after they have passed into this state.

7. The spinal cord is subject to all the diseases which may affect the brain, and spasm may be a symptom in almost any case.

The symptoms of *acute spinal meningitis* are pain in the neighbourhood of the affected part, violent from the beginning and rapidly becoming almost intolerable, increased by motion, and by the application of a hot sponge, but not by pressure—pains and feelings of pricking or numbness in the course of the nerves proceeding from the affected part, and a cord-like sense of constriction around the body upon a level with this part—spasms in the muscles of the back and neck, increased by motion and varying in severity from mere stiffness to complete opistho-

tonos—often morbid incitability in the sense of touch—spasmodic breathing—obstinate constipation—retention of urine—and if the inflamed part be higher than the lumbar region of the cord, priapism. “The cramp,” says M. Romberg, “is rarely persistent, being generally remittent, and recurring spontaneously after a pause, or as soon as the patient is required to make a movement.” The lower limbs, and the upper limbs too if the mischief be sufficiently high, are feeble, but not paralysed. At first, the mind is little affected; but as the disease progresses, a state of coma, sometimes preceded by wild delirium, may be developed. At first, there may be symptoms of active fever, but if so, these very rapidly lapse into those belonging to the typhoid condition. Indeed, in all cases the powers of the system must soon succumb to want of air, for the respiration is difficult and laboured from the beginning. Acute spinal meningitis is not unfrequently associated with the corresponding disease of the brain, and among the assigned causes, local violence, exposure to cold, and great fatigue, take the highest rank. *Spinal meningitis in a chronic form* is rarely met with, except in connexion with caries of the vertebræ, and its symptoms are for the most part sufficiently marked—paroxysms of pain in the neighbourhood of the part affected and along the nerves connected with this part, paralysis of motion extending centripetally, stiffness and spasm in the muscles of the neck and back, hectic, emaciation, œdema of the legs, a peculiarly dry and scurfy condition of the skin, and before death mischief in the brain of one kind or another.

Myelitis, however rapid in its course, has few of the characteristics of an acute disease. Its symptoms are feelings of numbness and tingling or pain beginning in the fingers and toes and creeping upwards, a sensation as of a cord tied around the body upon the level of the affected part, and paralysis of the regions below this part. Pain

in the back is not a prominent symptom, and what little there is, though increased by heat, is not materially aggravated by motion—a point in which this pain differs essentially from that of acute spinal meningitis; but pains in the parts to which the nerves connected with the diseased spine are distributed, often of considerable severity, are not at all uncommon. The characteristic symptom, indeed, is paralysis, not spasm or pain, and in some instances there may be scarcely any pain or spasm from the beginning to the end of the malady. If the site of the disease be low down, the paralysis may be confined to the legs, and life may be prolonged for a considerable time; if the site be sufficiently high, all parts of the body may be paralysed except the head, and death may be speedily brought about by paralysis of the muscles connected with the processes of respiration and deglutition. In the beginning the mind is clear, but this clearness is of short duration, and eventually the state is one of coma. The circulation never exhibits the slightest tendency to excitement. On the contrary, there is almost from the beginning a marked disposition to mortification in all parts which have to bear anything like pressure—a disposition which is utterly inconsistent with anything like true activity in the circulation. The symptoms of chronic myelitis, which most frequently make their appearance in connexion with caries of the vertebræ, would seem to differ from those which have just been mentioned in nothing beyond the comparative slowness of their development. They are always accompanied by hectic disturbance and extreme emaciation.

The immediate symptoms of *spinal apoplexy* are pain and marked paralysis of motion. If the site of the hæmorrhage be sufficiently high, there will be great dyspnœa and convulsive agitation and spasm in the parts below the injury. If the site be still higher, death may

happen at once. If life be prolonged, there is rapid wasting, and a great tendency to slough away in all parts below the injured portion of the cord.

The history of spasm in connexion with *other diseased conditions of the spinal cord* is not marked in very clear characters; but this much may, I think, be said, that the cases of paraplegia in which there is the greatest disposition to spasm in the paralysed limbs are the chronic cases in which there is no tenderness in any part of the spinal column, and not the more acute cases in which there is an opposite state of things. I have, within a short time, seen three cases of partial paraplegia of motion, with little or no impairment of sensation, in which strong and prolonged spasms were often produced in the paralysed limbs by attempting to move them, or by voiding the contents of the bladder or rectum, and in which the most careful examination failed to detect the slightest tenderness in any part of the spinal column, or any point anywhere which might be the source of reflex disturbance. This state of things had come on gradually in each of these cases; it had lasted for a period varying from two to four years; and it had never been attended by pain or tenderness in the back, or by the least sign of febrile disturbance.

8. There are many varieties of what may be called *minor spasmodic disorders*. There are, for example, cramp in the muscles of the calf of the leg, the laryngeal spasm of laryngismus stridulus and whooping-cough, carpopedal contractions, and writer's cramp.

Cramp in the calf of the leg is a very common form of minor spasm. It occurs more frequently in women than in men, and most frequently in the more irritable and weakly of women. It is the close companion of tremulousness; it increases in frequency as age advances. It is very prone to happen during sleep, and the liability to it

is infinitely increased during a state of fatuity. In this form of spasm the circulation in the limb is always very inactive, and not unfrequently the system is depressed at the time by some bowel complaint—diarrhœa or dysentery, or by pain, particularly by sciatica.

The spasm of laryngismus stridulus occurs suddenly and without any very obvious premonition. It may be a solitary phenomenon; it may be associated with cramps in the hands and feet, or with general convulsions; or it may alternate with these cramps and convulsions. So long as it lasts, the spasm causes an agony of suffocation. When it is over, the air finds admission to the lungs with a crowing sound, and the patient is relieved. There is no pain, no alteration of voice, no fever, and in these negative features the affection differs mainly from croup.

Nor is the history of the analogous spasm of whooping-cough less marked. The disease in which this spasm arises has two stages—the catarrhal and the convulsive. The first of these is attended by all the symptoms of coryza or catarrh, the cough being more sonorous and violent than usual, but *without any whoop*. The second stage is marked by the subsidence of all febrile disturbance, and by the supervention of the whoop. The whoop, moreover, disappears if pneumonia or bronchitis be developed after its establishment, and remains in abeyance so long as the inflammation continues. In the paroxysm itself the general condition is that of suffocation.

The spasms called “carpedal contractions” may occur periodically, and remain for days and weeks at a time. They flex the thumb across the palm, and bend the fingers over it; sometimes they bend the entire hand upon the wrist; they double the toes inwardly, and extend the foot. These spasms are unattended with pain, but any attempt at extension makes the patient cry out. They are confined to the first three years of life; they are often associated

with some other spasmodic affection, as laryngismus stridulus; and, so far as is known, they do not depend upon any special cause.

Writer's cramp is a spasmodic affection of the muscles of the hand and arm. Every attempt to write produces spasm in the muscles of the thumb and the two adjoining fingers, and sometimes in the muscles of the fore- and upper-arm; and what is not a little curious, these very muscles are capable of performing every other movement, except those which are involved in the act of writing. The case is singular, but not unique. Thus, M. Romberg speaks of a blacksmith whose arm became rigid and painful whenever he took hold of a hammer and attempted to strike, but who had no difficulty in using his arm for all other purposes besides hammering. In both these cases, local exhaustion from inordinate exertion of the affected muscles would seem to be the main cause of the trouble.

Many other varieties of minor spasmodic disorder might be cited, but none which would throw any further light upon the history of these disorders; and, therefore, I pass on without delay to speak of—

2. The pathology of convulsive disorders which are characterised by spasm.

§ I.

In all the varieties of spasm which have been noticed, the state of the circulation is one which is not to be mistaken.

During the attack of *cataplexy*, the appearance is that of a corpse, and it may be necessary to apply the ear to the chest, to know of a certainty that the heart continues to beat.

In *tetanus* there is no fever. All observers are agreed upon this point. It is found, also, that the spasms are

apt to become more general and violent as the pulse becomes feebler and the animal heat departs. The bouts of spasm, moreover, are distinctly coincident with paroxysms of difficulty of breathing, and in this way the spasm would seem to be connected, not with increased arterial injection, but with a state in which the aëration of the blood is considerably interfered with. And in the tetanus arising from strychnia, there is no reason whatever for a different conclusion; for the experiments of Dr. Harley, which have been referred to on more than one occasion already, show very plainly that the effect of the poison is to interfere with the proper arterialization of the blood, to destroy the incitability of the muscle and nerve, and to hasten the state of rigor mortis.

In the spasms of *cholera* the skin is frigid and clammy and blue, the breath cold, the pulse well-nigh imperceptible, and that the coincidence of this state of collapse is more than a mere accident would seem to be evident in the fact that the spasms relax *pari passu* with the reaction of recovery.

In *hydrophobia* the state of the circulation is the very opposite of fever, as is proved by the cold hands and feet, the perspiring skin, the quick and feeble pulse, the sobbing and sighing respiration, as well as by the fact that the agitation and spasm and convulsion increase in violence as the circulation fails. It would seem, too, that this agitation and spasm and convulsion must have a direct connexion with this depressed state of the circulation; for, on looking over the histories of a large number of cases, I find that there was less agitation, less convulsion, less spasm, where the circulation was less depressed than it is in the ordinary run of cases.

In *spasmodic ergotism*, so far as we know, the pulse presents no sign of excitement throughout the whole course of the malady.

In the "*early rigidity*" of cerebral paralysis there is at first no very evident alteration in the circulation, and the heat does not fall below the normal standard, but before long both pulse and heat fail in the paralysed parts. In "*late rigidity*" the local circulation is always feeble, and the heat in the part is kept up with great difficulty.

In *acute spinal meningitis* there may be symptoms of active fever at the onset, but, if so, they very shortly lapse into those belonging to a typhoid condition. Usually, however, the symptoms have a typhoid aspect from the beginning, and the respiration is too laboured and imperfect to allow of a different state of things. In *acute myelitis* the circulation is utterly without power, and as an additional evidence of this, there is a marked disposition to slough in all parts subjected to pressure. In *chronic spinal meningitis* and in *chronic myelitis*, the state is one of hectic exhaustion.

In the different forms of *minor spasmodic disorder* there is, for the most part, no evidence of over-action in the circulation. Nor is it otherwise where the phenomena of fever would seem to be mixed up with the spasm, as in whooping-cough. For what is the fact? The fact is that the whoop, which is the audible sign of the spasm, does not make its appearance until the febrile or catarrhal stage has passed off; that it disappears if pneumonia, bronchitis, or any other inflammation be developed in the course of the malady; and that it returns again when the inflammation has departed. In this case also, as in laryngismus stridulus, the way in which the spasm is mixed up with the phenomena of partial suffocation is an argument that the spasm is favoured by imperfectly arterialized blood.

§ II.

In the more severe forms of the disorders which are characterised by spasm, the mental state is indicative of

exhaustion, prostration, or inaction. In *cataplexy* the mind is in a deep sleep, or else lost in some dreamy vision. In *tetanus* the patient is alarmed, absorbed in his sufferings, agitated. The cramps of *cholera* are attended by indifference to the future and hopelessness, than which there are no surer signs of utter mental prostration. In *hydrophobia* everything denotes the want of mental energy, for the state is an exaggeration of delirium tremens. In *spasmodic ergotism* the mental state borders closely upon fatuity. In both forms of *the rigidity of cerebral paralysis*, early as well as late, the brain has been seriously damaged by white-softening, by apoplectic effusion, by red-softening, or in some other way, and the mental power has suffered accordingly. Nor is the case different in other forms of spasm. There may not be such obvious want of mental power, but if a careful search be made, there is always certain evidence of some want.

The functional condition of the cerebral hemispheres, indeed, is what might be expected from the depressed state of the circulation, and the depressed state of the circulation (to use once more the argument already used on so many previous occasions) is one which necessitates, as it would seem, a corresponding state of functional inactivity, not only in the cerebral hemispheres, but in the medulla oblongata, the spinal cord, and all other parts of the nervous system. Nor is this conclusion contradicted by the facts which remain to be noticed.

The fact that in the majority of cases of tetanus and hydrophobia there are no traces of inflammation of the brain, or spinal cord, or nerves, is in itself a plain proof that the inflammation which left these traces was not essential to the production of spasm in these diseases. The fact, also, that there may be violent and general tetanic symptoms where the spinal cord is untouched by inflammation, and that these symptoms are comparatively slight

and confined to the back of the neck, where the cord and its membranes are actually and unmistakeably inflamed, are arguments to the same effect—arguments which seem to show that the inflammation of the cord or its membranes may even have had the effect, not of causing, but of antagonizing and mitigating the spasm.

In hydrophobia, moreover, it must be remembered that the traces of inflammation are met with, not simply in the nervous system, but anywhere and everywhere. In this disease, indeed, there is a vagueness in the seat of these traces, which would seem to show that the inflammation which left these traces may have been a depurative process, by which the system strove to rid itself of the virus, and that these traces would be met with in all cases if death did not usually happen before there was time for their development. It would seem, indeed, as if the steps of the history of hydrophobia were in some degree parallel to the history of smallpox by inoculation. In the latter case, a virus is introduced into the system by a wound. Then, after an interval in which the morbid workings within the system are scarcely perceptible, rigors and other symptoms of collapse appear suddenly, and the system is at once laid prostrate. After this comes a period of reaction, in which inflammation and suppuration are set up in the skin and elsewhere. In hydrophobia, also, a virus is introduced into the system by a wound. Then, after an interval of delusive quiet, the baneful workings of the virus make themselves felt, and the system is suddenly overwhelmed in the fearful collapse of the disease. It would seem as if no other hypothesis but that of a poison working in this way upon the blood would account for the suddenness of the outburst, and for the rapidity with which the fatal issue is brought about. And if so, then hydrophobia is at once removed from the category of idiopathic inflammations to that of fevers, and

the inference is that the inflammation which now and then leaves its traces in this disease is a secondary and not a primary phenomenon, a depurative, perhaps, and therefore curative process, and that the rapidity with which the symptoms hurry on to death is the reason why these traces are not met with in all cases. In this point of view, indeed, it would seem as if a case of hydrophobia were analogous to a case of smallpox in which death happened in the cold stage which precedes the development of the characteristic pustules.

It is not improbable, also, that the workings of a virus, generated in the wound, may have somewhat to do with the production of traumatic tetanus: but in this case the morbid process at the wound would seem to be one which tells upon the nervous system, rather than upon the blood. How this morbid process tells upon this system is a problem of no ordinary difficulty, but there is no necessity to suppose that it tells by producing a more active development of nervous influence. On the contrary, the explanation of reflex contraction advanced in former pages may apply here. It may be supposed, for example, that pain was produced by the diseased tooth in the case of local spasm which was cited when speaking of the history of tetanus (p. 293),—that this pain implies an equivalent expenditure of nerve-current in the nervous arcs connecting the diseased tooth with the contracted muscles,—that this expenditure disturbs the equilibrium and produces movement in the nerve-current in these nervous arcs,—that this movement will be of greater duration when the chief source of the nerve-current (the nervous centres) of these arcs is so far exhausted as to prolong the time which is usually occupied in restoring the current to its proper level,—and that this movement in the nerve-current of the efferent nerves belonging to these arcs will bring about contraction by giving rise

to the development of momentary induced currents within the muscles to which these efferent nerves are distributed. In the case in question, the morbid process which springs from the diseased tooth is one which is exhaustive in its character, rather than stimulative. And there is no reason to suppose that the *modus operandi* is essentially different in any other case in which a state of spasm is brought about by a morbid change in a nerve.

And a contradictory conclusion is in no sense warranted by the presence of spasm in any case, or by the presence of morbid reflex incitability in some cases.

When speaking about the continued muscular contraction which may exist in certain anomalous forms of chorea (pp. 213—224), it was shown that this phenomenon is one which points to a disturbance in a particular part of the nervous centres—the incito-motory tract, and that the idea of inflammation or any other kind of stimulation had nothing to do with this disturbance. It was shown, in fact, that the phenomenon was in accordance with the premises, physiological and pathological, rather than with the current notion which looks upon spasm as the consequence of the excessive supply of nervous influence to the seat of spasm. And if this be true with reference to the spasms of tetanus, catalepsy, and all the different forms of “early rigidity,” it must be still more true with reference to “late rigidity,” for when this form of continued muscular contraction occurs, the contracted muscles have lost their incitability, and become wasted. It would seem, in fact, as if “late rigidity” were nothing more than an anticipation of rigor mortis, for it is preceded by the extinction of the muscular and nerve currents in the part affected.

And so also with morbid reflex incitability. For, after what has been said in the chapter which discusses the operation of nervous influence in muscular motion, it is

impossible to look upon this phenomenon as other than a sign of defective innervation.

— Looking back, then, at the facts which have been passed under review, it would seem that the occurrence of spasm is favoured, not by fever and inflammation of the nervous centres, but by wanting vigour in the circulation, and defective innervation. As in the different varieties of tremor and convulsion, so in the different varieties of spasm, the facts would seem to be altogether at variance with the theory which supposes that the muscles are provoked to contraction by excessive stimulation of one kind or other, and altogether in harmony with the theory which is propounded in this volume. It would seem, in short, that the key to the pathology is supplied by the physiology, and that the physiology is confirmed and established by the pathology. It is the same story throughout.

3. *The treatment of the convulsive disorders which are characterised by spasm.*

If the previous conclusion respecting the pathology of spasmodic disorders be correct, it is evident that anti-phlogistic measures will hold no very prominent place in a sound plan of treatment. It is even probable that such measures will be as little wanted as they were in the treatment of convulsion or tremor. And certainly there is no evidence to the contrary in the results of past experience.

In catalepsy, a hot bath, enemata of hot wine and water, ether, and other remedies which will rouse the system, with tonics and restoratives in the intervals, will be the measures in which most persons would put their trust. Of this there can be little doubt.

“In all cases,” says Dr. Watson, in his remarks upon the treatment of tetanus, “I should be more inclined to administer wine in large doses, and nutriment, than any particular drug,” and this, I take it, is also the opinion of

not a few of the soundest practitioners of this country. Many cases also are now on record in which the beneficial results of chloroform inhalations have been rendered evident, in relieving the pains as well as the spasms, and if care be taken to pour in wine and nourishment at the same time, there would seem to be no better plan. Another remedy, which will in all probability be found to be of great use, perhaps indispensable, is quinine. In some cases, it may be well to divide the nerve proceeding from the wound; but this measure is not likely to be required very often, particularly if the wine and food and quinine have been given vigorously, and the chloroform used judiciously, from the very beginning.

A similar line of treatment would also seem to be required in cholera, though here it is not likely that much good will be done by it unless the disease is taken in time. It is necessary, unhappily, not to be too sanguine as to the powers of medicine in the fully formed collapse; but there is no reason to doubt as to the practicability of preventing the disease by timely measures, even where the locality is highly unfavorable. At any rate, it is possible to prevent a disease which is in many respects analogous to cholera, and not less deadly—the remittent fever of the West Coast of Africa—by giving, night and morning, a good dose of quinine in wine. With quinine and wine, indeed, it is possible to live for weeks and months, where without the quinine and wine a residence of a few hours might lead to a fatal result.

Nor is there any reason to suppose that a different line of treatment is required for the relief of the symptoms of ergotism.

The rigidity of cerebral paralysis, in all probability, is an irremediable evil. If the cerebral mischief be irreparable, and the power of using the muscles voluntarily be lost irrecoverably, the paralysed muscles, sooner or later,

will pass into the state of "late rigidity;" and all that may be done is to defer the advent of this state by the proper use of electricity. Or if there be anything unusually inconvenient in the manner of the contraction, it may be necessary to consider the question of an operation by the subcutaneous method. Where the case is one of "early rigidity," the best treatment is perhaps that which does as little as possible. At any rate, after what has been said already, it can scarcely be necessary to have recourse to any kind of depletion.

In inflammation of the spinal cord or its membranes, chronic or acute, or in spinal apoplexy, the treatment must be guided by the same principles as those which were laid down when speaking of the corresponding affections of the brain.

In the treatment of the minor forms of spasmodic disorder, a main point will be to remove any local cause of reflex disorder, such as carious teeth, worms, and so on. In other cases, of a more chronic character, the aid of the surgeon may not be unfrequently wanted in order to divide contracted muscles. And if any general treatment be required, it may be supposed that it will be successful in proportion as it departs from anything of an antiphlogistic character. I know, for example, of four cases where attacks of laryngismus stridulus, which had frequently recurred under a treatment in which it had been thought essential to "regulate the secretions" by gray powder and so on, were put a stop to once and for all by a tonic and restorative plan of treatment—wine, beef-tea, steel, chloric ether; and I can speak with confidence of the beneficial results of a similar plan of treatment in a considerable number of cases of whooping-cough.

