

## **On the pathology and treatment of convulsive diseases / [by R. B. Todd].**

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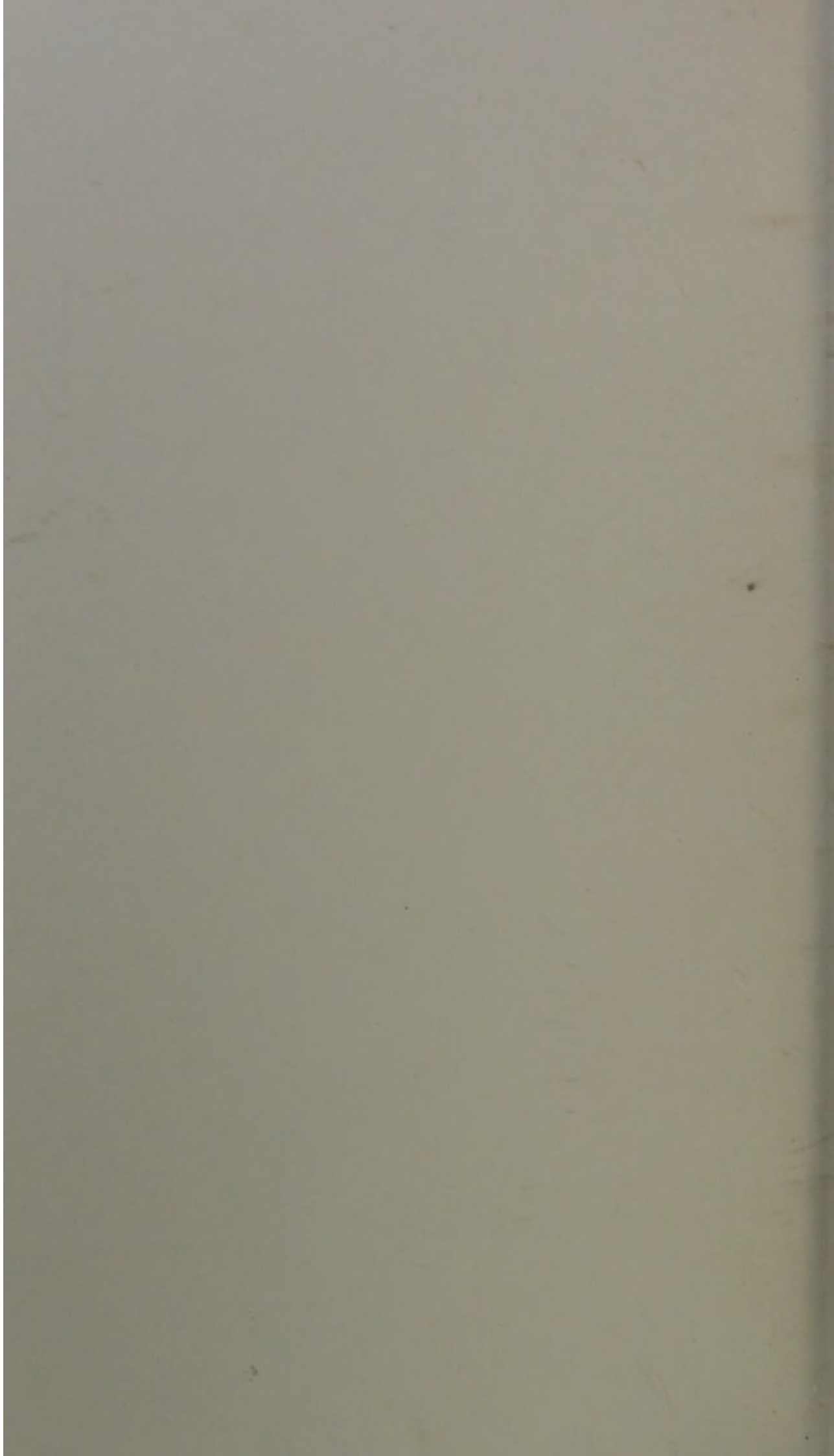
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THE

# LUMLEIAN LECTURES

FOR 1849.

*Delivered at the Royal College of Physicians, London,*

BY

R. B. TODD, M.D. F.R.S.

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ON THE

PATHOLOGY AND TREATMENT

OF

## CONVULSIVE DISEASES.

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*(From the London Medical Gazette.)*

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AT THE SIGN OF THE SHIELD, IN ST. PAUL'S CHURCH-YARD.

1813

## LECTURE I.

*Introductory remarks—Definition of Convulsion—Subdivision of convulsions—Choreic—Tonic or tetanic—Clonic or epileptiform—Chorea typical of the first class—Choreic convulsions in gouty and rheumatic states, in white softening of the brain—Tetanus typical of the second: to be classed with it, Trismus nascentium, and Laryngismus—Epilepsy typical of the third, including convulsions of infants, Puerperal convulsions, convulsions from renal or hepatic disease. Clinical history of chorea, two varieties, general and partial—Acute chorea—Non-symmetrical character of the choreic movements—Peculiar movement of the tongue—Paralysis following chorea, and preceding it—Disease of the heart accompanying chorea; this due to rheumatism—Choreic patients often of the rheumatic diathesis—Chorea not a fatal disease—Affections analogous to chorea—Peculiar affection of the hand—Fidgets of children—Points in the clinical history of Tetanus—Clinical history of Trismus nascentium—Of Laryngismus stridulus—Of Epilepsy, phenomena accompanying the fit—Paralysis after epilepsy—Acute Epilepsy—may occur without morbid lesion of the brain—Convulsions of infants—Toxic epilepsy—Puerperal convulsions.*

MR. PRESIDENT AND GENTLEMEN,—In every branch of science which depends upon, and, as it were, grows out of observation,—out of the observation not of to-day or yesterday, but of years and ages,—it is of vast importance that its cultivators should from time to time review the existing state of knowledge, examine well the bases on which it rests, and inquire whether the foundation be secure, and whether any change, either by addition or subtraction, should be made in the superstructure: as they to whom is confided the custody of some time-honoured edifice, planned, it may be, by the constructive genius of one man, but reared by the laborious industry of many, at various periods diligently inspect its foundations, search for such defects either in them or in the superstructure, as the less perfect architecture of a previous age may have suffered to exist, and supply these defects by alterations or additions suggested by the progress of knowledge, and the accumulation of experience.

Great advances have been made of late years in our knowledge of the physiology of the nervous system. We can now study the morbid phenomena of that system by the light which a more exact anatomy and physiology of it throws upon it, and likewise with the most important and valuable aid of larger views of pathology, and a more intimate knowledge of the various causes, whether external to, or inherent in, the body, which are capable of disturbing the normal action of the nerves or the nervous centres.

On this account I have chosen as the subject of these lectures a class of diseases, still obscure, but which the progress of general pathology seems likely to rescue from the darkness in which they have hitherto been shrouded. I propose to discuss the pathology of those diseases of which that kind of muscular action, to which the term *Convulsions* may be applied, is, if not a constant symptom, at least a very frequent one, and the prominent one when they are present.

It will be necessary for me to state in *limine*, that under the general term *Convulsion* I include all those irregular actions of sets of muscles which are wholly unrestrainable by the influence of the will, and which are excited and kept up by a physical irritant. Thus if the flexors of a limb are thrown into action of this sort, I would say they are convulsed, designating the peculiar mode of convulsion with which they are affected, and so also with larger sets of muscles, and the entire muscular system.

Under this definition of the word *convulsion* I would refer to three distinct kinds of convulsions, each of which denotes a different cause, and is produced by an affection of the nervous system distinct in kind as well as in position. These are, the jactitating, or chorea-like, or choreic; tonic, or tetanic; clonic, or epileptiform.

The first kind are peculiar jerking movements affecting the muscles of a limb, or of one side of the body and of the face: the movements are exceedingly irregular, wholly uncontrollable by any effort of the will, and only affected by it in this way, that an effort of the will directed upon the disordered muscles tends not to restrain, but greatly to augment the disturbance. There is in general no augmentation of power in the choreic convulsion; on the contrary, it is accompanied by a state of great debility and of imperfect muscular nutrition: sometimes it is preceded by a

paralytic state, and not unfrequently it is succeeded by it. If any attempt be made to restrain the convulsive movements, they immediately become increased in force and frequency, and there is danger that under a continuance of the restraint a partial or limited convulsion may be converted into a general one. Lastly. The choreic convulsions, however general and rapid, are controlled in the most perfect manner by sleep.

The second, or tonic or tetanic convulsion, is a state of greatly increased muscular tension, which never experiences any complete relaxation, but every now and then undergoes a sudden increase of tension. A muscle thus convulsed may be compared to the string of a violoncello, screwed up to concert pitch, and from time to time made more tense by a sudden turn of the regulating screw, which gradually allows it to return to its former tension. In these convulsions there is doubtless a preternatural development of the muscular force, to such an extent that some of the fibres become ruptured by their own force of contraction. Convulsions of this kind may be exactly imitated by the transmission of the galvanic current in rapid succession through the spinal cord, or by the continuous transmission of the *inverse* current for half an hour through a limb; when, upon the interruption of the current, the muscles of that limb suddenly assume the tetanoid state, which lasts for a few seconds,—or they may be developed by the poison of strychnia.

The third, or clonic or epileptic convulsion, consists of powerful active contractions of sets of muscles, resembling a succession of forcible voluntary contractions. Each act of contraction is rapidly succeeded by a state of relaxation, and again as rapidly followed by a fresh contraction, and so on until the muscular force is well nigh exhausted, or the exciting force is worn out. The power of these contractions is always great, sometimes extraordinary; so that the combined effort of several strong men is inadequate to hold the patient down: this kind of contraction is not favourable to the rupture of the muscles, but bones are sometimes broken in the epileptic convulsion. I have known even so strong a bone as the femur to have been broken by the muscular struggle.

Having thus defined the nature of the three forms which, I believe, include every variety of disturbed muscular action to which the term convulsion can be applied, I shall proceed to enumerate the various diseases which are characterised or accompanied by these unnatural manifestations of muscular force, and then take a rapid glance at the main features of their clinical history, which will form the premises whence we are to draw our conclusions regarding the pathology of these diseases.

The first class of convulsions is met with in the typical malady, chorea, and in states resembling chorea, although differing from it in many particulars.

The only other conditions under which such convulsions take place, so far as I am aware, are in particular rheumatic and gouty states, and in white softening of the brain, connected with, and probably arising from, a morbid state of arteries.

The type of the second class of convulsions is that formidable malady, Tetanus, the clinical history of which, as resulting generally from wounds, is now so well known.

Along with this I would class that remarkable affection which attacks infants, which has received the various designations of spasmodic croup, cerebral croup, *Laryngismus stridulus*.

The disease called *Trismus nascentium*, better known in the West Indies than in this country, is also very nearly allied to, if not identical with, tetanus.

I would name Epilepsy as typical of the third class of convulsions, and in the same category with it would come the common convulsive fits of children, the convulsions which affect puerperal women, &c.

My remarks on the clinical history of these various maladies will be made in the order in which those diseases have been referred to.

And first, of Chorea.—The disease is one of early life, very rarely occurring after puberty has been well established; and if it do occur later than the ordinary period of life, when the signs of puberty are developed, it is generally found that those signs have not as yet distinctly manifested themselves in the patient. The period to which chorea is most properly restricted is that between 6 and 15 years of age. Of 189 cases observed by Rufz in the children's hospital at Paris, 10 only occurred before 6 years; 179 between 6 and 15; and of the latter number, 118 occurred between 10 and 15.

It affects girls in much greater proportion than boys. Of the 189 cases collected by Rufz, 138 were in girls, 51 in boys.

The muscles affected in this disease are all those connected with the cerebro-spinal nervous system,—those of the head, face, trunk, and extremities. It is very doubtful that even the so-called organic muscles ever suffer; and if the heart is involved at all, its affection is probably of an entirely different kind, and takes place in a different manner from that of other muscles.

The examination of the phenomena of a great number of cases of chorea will show that the disease exhibits two varieties, namely, general chorea and partial chorea; and that the former may be either acute or chronic.

*General Chorea.*—In *general chorea* the

whole external muscular system is involved ; the patient is unable to stand or walk ; speech is either quite destroyed, or so inarticulate that it is impossible to understand what is said,—such is the disturbed state of the muscles that no voluntary action can be properly performed. The tongue is protruded with the same peculiar jerking effort which accompanies all the other muscular actions ; and one wonders that, as it emerges from the mouth, it escapes being injured by the teeth, which the irregular actions of the masticatory muscles press convulsively against it.

The patient, unable to stand, is obliged to remain in bed ; and there tosses from one side to another, sometimes rolling incessantly to such an extent that the friction of the most prominent parts of the body against the bed causes stripping and ultimately troublesome sores. And when the agitation is violent, these patients literally shake themselves to death, which is brought on by the exhaustion induced by these unceasing movements.

When the convulsive movements are so violent and unceasing as I have now described them, the disease may be called *acute*, for it must destroy the patient, or cease, in a few days.

Happily, however, this form of the disease is rare, and general chorea occurs most frequently in a more chronic form ; the movements, however, being of the same character, less frequent and less active, and with longer intervals of sleep, which restores the patient and provides fresh strength for new agitation.

*Partial Chorea.*—The most common form of *chorea* is the *partial*. It is needless for me to enter upon any detailed description of the phenomena of a disease which must be so familiar to all my hearers. I will just point your attention to a few of its leading features.

1. The affection is not symmetrical, that is to say, one side of the body is more frequently affected than both ; or if both should be affected, one is always much more so than the other. In general, this difference of affection of one side remains with that side whichever it may be ; but in rare instances there is a disposition to shift, and for two or three days the right side will be chiefly affected, and then for a longer or shorter period the left side will exhibit the most active movements. This tendency to shift calls to mind a prominent feature of certain rheumatic and gouty affections.

2. Of all the movements which accompany chorea, that of the tongue is the most peculiar and characteristic ; indeed I would call it pathognomonic. The patient protrudes the tongue with a peculiar thrust to the fullest extent of which it will admit ; fre-

quently this is done by one effort, at other times it requires two or three attempts before it can be accomplished. And the subsequent retraction is also peculiar : the tongue is drawn back, supported and guided by the pressure of the teeth, and often very slowly and with great caution. Sometimes this peculiar mode of protrusion of the tongue is the precursor of the other choreic symptoms ; and if we had frequent opportunities of seeing the cases very early, I have no doubt that this symptom would be found more frequently the harbinger of the more extensive affection. I have, from this symptom only, been able to predict with accuracy that an attack of chorea was coming on.

3. The choreic convulsion is frequently succeeded by a paralytic state of the limbs previously convulsed ; the convulsive movements cease, and the limbs remain paralysed ; the paralysis is seldom complete, although considerable. When the choreic convulsion had affected one side, the paralysis will likewise affect the same side ; it will be *hemiplegic*, and will resemble very closely hemiplegia from diseased brain, for which it is very apt to be mistaken by those not aware of this fact. Not long ago a boy was brought to my house for me to advise whether the hemiplegic paralysis under which he suffered should be treated by antiphlogistic means, such as mercury, &c. I soon discovered that the case was one of choreic paralysis, and advised steel and shower baths, and exercise of the limbs ; under which he rapidly recovered.

In a very few instances paralysis *precedes* the choreic convulsion, but so far as my observation informs me, it is then accompanied by the peculiar thrust of the tongue ; by this sign I have been enabled to distinguish it.

That paralysis may occur in this way is evidently a fact of the highest practical interest, whilst it is not devoid of importance as bearing upon the pathology of the disease. Nevertheless I do not find it noticed in any of the descriptions of the disease in the practical works which I have consulted. The previous history of the case, the absence of any other symptom referable to the head, and the gradual mode of invasion of the paralysis, in addition to the peculiar mode of protrusion of the tongue, will always enable the careful practitioner to distinguish this paralysis from that caused by cerebral lesion.

4. The heart is very frequently morbidly affected in chorea ; and this morbid affection shows itself, not in any disturbance of the *rhythm* of the heart, which, so far as my observation extends, never is disturbed, but in a derangement of its sounds. A bellows sound is frequently present, and is either aortic systolic, when it is almost

always an accompaniment of the anæmic state of the patient, but *much more frequently* it is *mitral systolic* or *regurgitant*.

I am surprised that this feature of the clinical history of chorea has not attracted more general attention. It has been well known to me for many years, and I described it in the Croonian lectures on the pathology of rheumatism, which were given by me in this theatre six years ago. But to my friend Dr. Addison, of Guy's Hospital, is due, so far as I know, the merit of having first made public the frequent occurrence of this bellows sound in chorea.

What is the cause of this sound? is it due to any irregularity in the muscular action of the heart? a choreic state of that, as of the external muscles, whereby the action of the valves is disturbed? or is it caused by a morbid deposit on the mitral valves, which prevents their precise action; or can it, like the aortic sound, depend on the anæmic state of the patient?

I shall answer the last question first. The closure of the mitral valves is the result of the mechanical accumulation of fluid in the ventricle, which presses the valves towards the auricle, their tension being maintained by the chordæ tendinæ: this effect will be produced no matter what may be the nature of the compressing fluid; a mere colourless state of the blood cannot produce mitral regurgitant bellows sound; some imperfection, either of the valves themselves, or of their mode of apposition, must exist in order to produce such a phenomenon.

It is plain, then, that the bellows sound must be caused by some disturbance in the action of the mitral valves. What, then, produces the valvular derangement? Can it be any disturbance of the muscular action of the heart, any choreic state of the muscoli papillares? I apprehend not; for as the closure of the valves is not accomplished by the action of the muscles, it would not be affected by any such irregular muscular action. Moreover, no such choreic state of the heart's action could take place without its being indicated by a disturbance of the heart's rhythm, which, of course, would be very readily detected.

Hence, then, we cannot resist the conclusion, that the mitral bellows sound in these cases is due to such an organic lesion as prevents the complete closure of the mitral valves during the systolic effort of the heart.

But now we are met with another question not less important. What can have created this morbid state of the mitral valves? It is rare to meet any symptom of heart affection, either preceding or accompanying the chorea; and the valvular imperfection is often overlooked, and only discovered by careful auscultation of the heart's sounds.

The true answer to this question is, I

believe, to be found in the fact that many of the patients who suffer from chorea are of a rheumatic diathesis, and that in consequence of this rheumatic state they experience an insidious endocarditis which generally affects the mitral valves.

The proof that rheumatic diathesis and chorea are frequently met together, is derived from these facts,—that many of the sufferers from the latter malady, when carefully examined, are found to have previously suffered more or less from chronic articular pains, to have sprung from rheumatic or gouty parents, and to exhibit deranged secretions, such as occur in rheumatic states. I have seen many instances of choreic children passing urine of high specific gravity, loaded with lithates, and sometimes precipitating lithic acid in very notable quantity, and for a considerable time. Choreic patients often suffer from rheumatic fever; and, as the subjects of that fever are very liable to its recurrence, so a child who has once had chorea is apt to have it again and again.

The history of the following case will serve as an illustration of the kind of connection which sometimes exists between chorea and rheumatic fever.

Alfred Brewer, æt. 14, had good health until his eighth year, when he had an attack of rheumatic fever. Four months after this he became affected with general chorea. He was cured of this in seven weeks. A year afterwards he had a second attack of chorea, which affected the left side chiefly. From this time he continued well for four years, then he had another attack of rheumatic fever, which lasted three weeks, and six weeks after this he was seized with his third attack of chorea.

Chorea does not occur in children whose nutrition is in a healthy state, but in those whose digestive powers are feeble, or who are badly fed, either by too abundant and ill-adjusted diet, or by food defective both in quantity or quality. It is not unreasonable to expect to find many rheumatic subjects among such patients, especially among the children of the poor, who are exposed to the inclemencies of the weather.

Lastly, the history of chorea shows that its natural tendency is to recover, and that it very seldom proves fatal. Of the 14,725 deaths which occurred in London in the quarter ending at Christmas 1848, only one was from chorea; and, in the corresponding quarters of the seven previous years, the deaths from the disease did not exceed one, excepting in the year 1844, when there was the curious exception of six deaths from chorea registered; and in 1845, there was no death from that cause.

When we take into account the large number of children affected with this disease, as the records of every hospital and dispen-

sary will show ; and when we remember how many of these cases are neglected, left solely to nature, receiving neither care nor medical assistance,—we are fully justified in laying it down as a feature of the disease, that its natural tendency is to get well.

Whatever morbid disturbance may take place in any of the internal organs calculated to give rise to chorea, it may be presumed from the facts I have detailed that it is not such as does not admit of easy amendment ; and the few post-mortem examinations on record of patients dead of this disease, show that it leaves behind it no trace of any lesion in any organ of the body likely to produce it. In some of the cases no morbid lesion of any kind could be detected ; and it cannot for a moment be imagined that the slight opacities of the arachnoid, the effusion of liquid in the spinal canal, the marks of chronic ovarian disease, noticed in other cases, could at all have the relation of a cause to the phenomena of the disease. How many hundreds of spinal effusions, or opaque arachnoids, or irritated ovaries, have there been without the slightest indication of chorea !

So far for such points in the clinical history of chorea as may serve me as a basis for my argument respecting its pathology.

I have already stated that movements analogous to those of chorea may occur in rheumatic or gouty states. I have known such affections of the upper, and more rarely of the lower, extremity. The affection consists in a sort of fidgetty restlessness of the limb, and a difficulty, and sometimes an impossibility, of accomplishing certain actions with anything like accuracy or precision. In the cases which have come under my own observation there has been a considerable development of the gouty constitution.

There is a curious affection, of which I have met with two examples in my own practice, and of which five or six have been recorded by Albers of Bremen : the prominent feature of it is the inability to hold a pen and to write for any time. The patient begins to write—writes two or three words—when the hand forthwith is spasmodically extended, the fingers cease to be controllable, the pen falls away from them. If the patient attempt to control his pen hand with the other hand, or if any one else does it for him, the movements become worse, and he suffers excessive pain up the arm : yet all other actions are well done, and the power of the hand to grasp is good. One gentleman thus affected is a man of considerable mental endowments, and he has found it so impossible to overcome the irregular movements of his hand, that he has trained himself to write with his left hand, which he can now do with such

facility that he can carry on an extensive correspondence.

There can be no doubt that the impairment of speech, which is sometimes a forerunner of cerebral mischief, and which ought always to be regarded as a warning of such an invasion, is of this character. Emotional excitement will cause hesitation of speech in many healthy persons : many can do certain things very well if allowed to take their own time, but if hurried and flurried they are apt to bungle about them very seriously.

In one instance I found well-marked choreic convulsions of the right arm, with disturbed intellect, and gangrene of the toes, associated with extensive colourless softening of the left cerebral hemisphere, involving the optic thalamus.

Allied to the movements of chorea are the fidgets of children, and, perhaps, also those of grown persons. In some children these fidgetty movements are so excessive that the child becomes almost a nuisance in a room. All the muscles are affected ; the child incessantly makes grimaces of the most various kinds ; every minute he assumes a new attitude ; if any thing comes in his way he must handle or touch it : and these irregularities are always the greater when there is derangement of general health. Children thus affected might readily be thrown into the convulsions of chorea by a strong mental emotion.

My second class of convulsive affections is typified by the well-known malady, Tetanus.

The points to which I would beg your particular attention in the clinical history of this disease are these :—

1. The little variation of the symptoms in different cases of tetanus as compared with each other. If you see one well-developed case of tetanus, you have seen all ; the variations are only as to the degree of tetanic convulsion : in all you have the tetanic face, the rigid limbs, the locked jaw, the opisthotonos. In the acute form of the disease, indeed, the malady runs its course with much greater, sometimes with extraordinary, rapidity ; while in the more chronic form it lasts a fortnight or three weeks, and in a few a much longer period.

2. The fact, now abundantly proved, that these symptoms occur without any lesion of the nervous system appreciable by aided or unaided vision ; and what is equally remarkable, the artificial tetanus (if I may so speak) caused by the administration of strychnine in large doses, occurs equally without the slightest lesion of any part of the cerebro-spinal axis. I have examined, with the most scrupulous care, the spinal cords of rabbits which died in strong tetanic convulsions, kept up for some time after

large doses of strychnine, but I could not discover, even with the highest powers of the microscope, any indication of deranged structure.

3. The curious and interesting fact, that tetanus often appears endemically, and its development is favoured by some localities much more than by others. Thus it is well known that the disease, not only in its traumatic but also in its idiopathic form, is much more common in the West Indies than in more temperate climates: and it seems pretty well proved that under modern improvements in diet, ventilation, and cleanliness, as well as under a better mode of treatment of wounds, the disease has become much less common in that climate than formerly. Even in this country we meet with curious instances of several cases occurring in rapid succession in the same locality from exciting causes, which, although themselves frequent and common, have rarely before been followed by the same results.

4. The tendency in tetanus is to death by exhaustion and asphyxia. The frequent return of the attacks of spasm, affecting all the muscles of the trunk, have a most exhausting influence upon the patient; and as the respiratory muscles, and even the muscles of the larynx, are generally more or less involved, the respiratory function becomes seriously impeded. Hence, the most fatal sign is a rapid recurrence of the convulsive attacks, and the most auspicious sign is the prolongation of the intervals between them. Practically the disease should be regarded as one of extremely fatal tendency, and therefore requiring the diligent interference of art to counteract its debilitating influence.

The disease called *Trismus nascentium* is essentially the same as acute tetanus affecting infants, and due to a peculiar state of system developed under deleterious local influences. Under modern improvements in diet, cleanliness, and ventilation, this disease is rapidly vanishing from this country, and has diminished considerably even in the West Indies. Dr. Clarke, by means of good ventilation, reduced the mortality from this disease in the Dublin Lying-in Hospital from seventeen per cent. to five or six: and in Dr. Collins' mastership of the same hospital, extending from 1826 to 1833, the mortality from this disease had fallen to a mere fraction per cent.;\* and Dr. Colles states that by adopting the plan of plunging the infants daily in cold water, and dressing the umbilicus with turpentine, the disease, on a particular estate in the West Indies, from having been exceedingly frequent became so rare as scarcely to be met with.

I have classed with the diseases accom-

panied by tetanic convulsions, that curious, and sometimes highly dangerous disease of infants, the laryngismus stridulus, as it is now generally named, because it has many points of resemblance to tetanus, and because I am of opinion that its pathology is essentially the same with that of that disease.

The features of this disease to which I beg your particular attention are these—

1. That the laryngeal spasms are truly tetanic, and occur in paroxysms: differing, however, from the ordinary tetanus in this, that a more complete relaxation of the muscles takes place in the intervals between the paroxysms, and that a considerable period often elapses before a recurrence of the paroxysm takes place.

2. As in tetanus the paroxysm is brought on by the sudden application of external stimuli, as by a sudden draught of cold air, a start, by sudden mental emotion, by the attempt to swallow, so the same causes tend to produce the paroxysm in the laryngismus.

3. Tetanus sometimes does not extend beyond the muscles of the jaw and throat: this is more frequently the case with laryngismus, but very frequently in the latter disease the tetanic convulsion extends to other muscles: the muscles of the extremities become engaged, and the so-called *carpopedal* spasm is induced; the limbs being stiffened, the wrists flexed, the thumbs turned in, and firmly bent into the palm of the hand, and at the same time a similar condition of the ankle and foot, and an inversion and flexion of the great toe. With these there is frequently well-marked opisthotonos, sometimes not extending beyond the cervical and dorsal regions, sometimes affecting the whole spine. During the paroxysm the child's consciousness is unaffected, and after its cessation the child returns to its previous state, appearing no otherwise changed than being more or less exhausted by the greater or less violence of the paroxysm.

4. Laryngismus does not manifest itself in robust and healthy children, but in children often weakly, but always in a state of depraved nutrition, from irregular and imperfect feeding, bad air, and frequently from the irritation of teething. A strong, healthy, well-fed child never exhibits laryngismus. Frequently the similarity of nervous constitution, as well as the mode of dieting of children in the same family, favours the development of the malady in successive brothers and sisters about the same period of infancy; and hence we often hear of instances of several members of the same family having been carried off in infancy by this malady.

5. There can be no doubt that under the continued operation of the exciting causes of the disease, laryngismus tends to a fatal

\* See Collins' Midwifery, p. 513.

result. It exhibits no spontaneous tendency to recover. Death takes place in some cases apparently from rapid asphyxia, much in the way it sometimes does in the cases of rapid development of œdema glottidis, or from the attempt to respire carbonic acid gas; generally the infant dies from exhaustion, brought on by the frequency of the paroxysms, and augmented by the injury to the respiratory function which each paroxysm causes. In such cases the mode of death is exactly as in tetanus. But in a third class the long continuance of the impaired nutrition generates a morbid state of brain, epileptiform convulsions are super-added to the tetanic convulsion, and dropsy of the ventricles takes place, favoured by the retardation to the venous circulation which these frequent attacks occasion, and also, sometimes, by the deposit of tubercular matter in the course of the *venæ magnæ Galeni*, which return the blood from the ventricles and the choroid plexus; and thus the patient dies with all the phenomena of hydrocephalus.

I come now to notice the clinical history of a class of convulsive diseases which is far more common and much more formidable in appearance than any of the others to which allusion has been made, but which is much less fatal than those of the tetanic kind.

And I shall take first the typical malady—*epilepsy*. This disease is characterised mainly by the occurrence at intervals sometimes remarkably uniform in duration, of attacks of loss of consciousness, frequently sudden, often preceded by some kind of warning. These attacks last for a longer or shorter interval, when the patient recovers, as if awaking from a sleep, but continues in a drowsy state for a variable term.

In by far the greatest number of instances these attacks are accompanied by a loss of *voluntary* and *locomotive* power, so that the patient, on the supervention of the attack, falls down, and is thrown into convulsions of the character which I have already described by the name of epileptiform convulsions. These continue for some time—they subside—the state of insensibility remains—and for a variable time the patient continues in a profound sleep, or in a state of heavy drowsiness.

The phenomena which precede the fit vary infinitely, not only in their nature, but in their intensity and in their duration. In by far the majority of cases the precursory symptoms of an attack consist in a disturbed state of sensation, or of intellectual action. Thus the patient has headache, or a sense of giddiness, or some altered state of vision, causing motes or flashes of light, or other more complicated ocular spectra. Sometimes hearing is affected: the patient com-

plains of tingling in the ears, and noises in the head. Or smell may be disturbed, as in the case mentioned by Heberden, where a smell of musk ushered in the attack.

The more complicated precursory phenomena also illustrate my position: thus with one of my patients the warning came on five minutes before the attack, and consisted in a feeling of terror, with violent tremors, and the fancy that he saw persons coming up to him and impeding his attempts to move on. Dr. Watson quotes from Dr. Gregory the curious instance that with a patient of his the warning consisted in the phantom of a little old woman in a red cloak coming sharply up to him and striking him on the head.

Not unfrequently before a fit the patient becomes delirious, sometimes raving mad; then comes the regular paroxysm, and when sensibility returns, the delirium again sets in, and remains for some time. The knowledge of this fact lately saved me from what might have proved a serious interruption and inconvenience. A gentleman, who had had one or two paroxysms of epilepsy, came to my house one morning to consult me: having talked quite sensibly for a short time, he suddenly seemed to lose himself, and then talked incoherently. This excited my alarm: I sent for a cab, and went home with him, and had just arrived at his door when he was seized with one of the most violent and one of the longest fits I ever saw.

The *aura epileptica* is a disturbance of sensation; but it is remarkable how extremely rarely this phenomenon, so often referred to by writers, is met with. It very seldom happens that we meet with an example of it presenting even a near approach to the description given of it by writer after writer.

The loud unearthly shriek which often ushers in the fit is probably excited by some sudden disturbance of sensation—some severe pain—which causes the patient to cry out, but which the sudden invasion of the epileptic state dissipates from his memory.

In most cases of epilepsy all the voluntary muscles are engaged, but in many instances those of one half the body are much more convulsed than those of the other, and in some the convulsions are altogether limited to one half of the body. I have frequently seen the most intense convulsion of the arm and leg, and side of the face on one side, while the corresponding parts on the other side were perfectly quiescent. The action of the heart is very much accelerated in the fit; the terror which its approach induces greatly excites this action, and this excited state continues for some time after the fit has subsided. A dilated condition of the pupil almost invariably accompanies the fit, however slight it may be.

Epilepsy is a disease of all periods of life: it seems not improbable that it may commence even *in utero*, being first developed by some powerful emotion excited in the mother, or due to some other disturbing influence upon the nervous system of the embryo.

Bouchet and Cazanvielh, from sixty-six observations, obtained the following results regarding the periods of life at which epilepsy is most apt to occur:—that from birth to 5 years there were 18 epileptics (9 being congenital); from 5 to 10 years, 11; to 15, 11; to 20, 10; to 25, 5; to 30, 4; to 35, 1; to 40, 2; to 45, 1; to 55, 0; to 60, 1.

A paralytic state remains sometimes after the epileptic convulsion. This is more particularly the case when the convulsion has affected only one side or one limb: that limb or limbs will remain paralytic for some hours, or even days, after the cessation of the paroxysm, but it will ultimately perfectly recover. This is a form of paralysis in a great degree analogous to that of chorea.

There is an acute form of epilepsy, as there is a chronic form also. The acute form is distinguished by the number and violence of the fits, and the rapidity with which they succeed each other: so violent are the fits, that they exhaust, and, if long continued, kill the patient. I have seen patients exhibit a rapid succession of fits for a whole day, with intervals of not more than five or ten minutes, and the exhaustion induced by them has been so great that I have been forced to give stimulants in large quantities to keep the patient alive.

In the chronic form the disease is remarkable for the kind of periodicity which regulates the development of the paroxysms: thus the fit will occur once in 24 hours, or once in 48 hours, or once a week, or once a month, but not with unvarying exactness as to the interval, nor without many disturbances to the ordinary rate of its occurrence. Although this periodicity exhibits some analogy to that of ague, it wants greatly its precision.

Of the tendency to the propagation of epilepsy by hereditary descent there can be no doubt. Cazanvielh and Bouchet make this remarkable statement: 14 epileptic mothers had 58 children: of this number 37 died, the eldest at 14, the rest nearly all of convulsions; 21 were living, of whom 14 were healthy, still young, and 7 already epileptic.

Epilepsy seldom kills: in the record of deaths we meet comparatively few deaths from that cause: epileptics die nominally of other diseases: they are liable to the vicissitudes of other men—accidents, and consequent injuries, not unfrequent (and to these

their epileptic condition peculiarly exposes them), fevers, diarrhoea, cholera, &c. These dispose of a large number of them; but a considerable proportion gradually fall into dementia or other forms of insanity, and then they become classed with the insane.

The tendency of epilepsy is to the destruction of the nervous force, in the brain especially, but ultimately throughout the whole cerebro-spinal centres. The earliest indication of the failure of the nervous power is in the weakening of the perceptive powers and of the memory. No doubt the power of attention suffers primarily, and then the retentive faculty becomes damaged. In this way only can we explain the fact that the power of memory for events of *recent* occurrence suffers first, the patient recollecting old events, or facts stored in the mind at a remote period, as well as ever. Ultimately, however, the memory of events of all kinds and at all periods is lost, and the patient becomes fatuous. This failure of the intellectual powers takes place the more rapidly as the paroxysms are more frequent and more severe: it will occur more surely where the fits have been accompanied with much mental disturbance or stupor, than where the convulsive character predominates. Frequent attacks of the epileptic vertigo of Esquirol—the *petit mal*—are in general pretty certain precursors of a state of dementia or fatuity.

It is an important feature in the clinical history of epilepsy that the fits sometimes cease spontaneously, or without any cause which can be recognised. Many a remedy for epilepsy has acquired the credit of curing the disease, when it has happened to be exhibited in a case in which this spontaneous tendency to recovery has manifested itself.

Epilepsy and insanity frequently go together: in some cases the epilepsy precedes, and, as it were, induces the insanity; in others the insanity has the precedence; and in a third class the patients are insane only for a short time before, and for a short time after, the epileptic paroxysm, and then resume a tolerably healthy state of mind until the access of the next paroxysm.

A remarkable and most interesting point in the clinical history of epilepsy is that it may go on for a considerable time, even in its most acute form, without the existence of any morbid change in the brain to be recognised by our means of examination.

Nothing is more certain than that not only one, but several paroxysms of epilepsy may be induced in a person whose brain and spinal cord present all the ordinary appearances of health.

The congestion which is apparent in the brains of epileptics, but which is seldom present excepting in those who have died in the fit, or very shortly after it, is attributable

entirely to the impediment to respiration, and consequently to the venous circulation, caused by the epileptic struggle. When death has been not immediately consequent upon a fit, the appearances indicate rather that the brain has not been supplied with its usual amount of blood, and that whatever congestion may exist is found in the veins. Those bloody points on the surface of the centrum ovale which are so often referred to by authors as indicative of a state of congestion are mostly the mouths of veins divided in slicing the hemispheres.

The opaque arachnoids, the enlarged Pachionian bodies, the adherent dura mater sometimes partially ossified, are often found where there has been no epilepsy, and are rather the signs of the altered nutrition brought on by any cause which creates frequent disturbance of the actions of the brain, than the causes of that disturbance. These morbid appearances are constant, but the diseased actions are periodical: it is therefore highly improbable that the lesions above described would develop the phenomena of the epileptic fit.

The brain itself also suffers in its nutrition; it diminishes in size, the convolutions become small, the sulci between them large: the subarachnoid fluid, which always, in point of quantity, bears an inverse proportion to the bulk of the brain, is increased in amount to a greater or less degree.

Whether tumors, the remains of old apopleptic cysts, spicula of bone growing from the interior of the cranium, indurations of portions of the brain, or the so-called hypertrophy of the brain, can give rise to that which has been called *centric epilepsy*, I must reserve for further discussion when I come to inquire into the pathology of this disease.

There are conditions remarkably resembling epilepsy, and not to be distinguished from it as regards the actual phenomena of the fit, but which occur under such constant circumstances that the paroxysm evidently is related to them as effect to cause. I allude to the convulsions of children—the convulsions induced by the introduction of some poison into the system, or the retention of some material in it which ought to be excreted as soon as formed as urea (the *epilepsia* or *eclampsia toxica* of some authors), the convulsions of puerperal women (*eclampsia parturientium*).

The ordinary convulsions of children resemble closely the epileptic paroxysm: in many instances, however, there is a great deal of tetanic convulsion combined with the epileptic convulsion. The unconsciousness of epilepsy is always present in the convulsions of children, and the torpor before and after the fit is rarely absent. Signs of the

coming struggle are afforded in restlessness, peevishness, diminished intelligence, a heavy and torpid, but not a sleeping state.

Convulsions do not, in general, attack robust and healthy children, but those who are weakly and ill-nourished; not those who are furnished with a good supply of healthy blood, but those whose blood is poor in quality, and often deficient in quantity; children that are improperly or insufficiently fed; or in whom various resources of irritation exist—such as dentition, irregular secretions, &c. And it may be remarked that these sources of irritation favour the development of convulsions more by the disturbance which they create in general nutrition, in the state of the blood, and in the nutrition of the brain, than by any direct influence upon that organ through the nerves.

Of 28,378 deaths of children from infancy to 15 from all causes in London and its environs, in 1848, those from convulsions numbered 2047, or nearly nine per cent. The children in general die from exhaustion, worn out by the repetition of the fits; and, in the vast proportion of cases, anatomy discloses nothing to explain why convulsions should have existed before death more than other morbid phenomena. With proper care and attention to the child, convulsions have not necessarily a fatal tendency—no more than epilepsy; for, judging from the immense number of children who are attacked, the proportion of deaths to recoveries must be small; but on this point we have no accurate statistics.

Let me remark here, that such morbid appearances as are met with most commonly in the heads of children dead of convulsions, are attributable to the state of general disturbed nutrition of the nervous centres, as in epilepsy, and cannot be regarded as the *cause* of the convulsive paroxysm. The appearances to which I allude are increased sub-arachnoid fluid, or intra-ventricular fluid: they never exist together, as the one may be regarded as antagonistic of the other:—for the most part a pale state of brain, especially of the vesicular matter, with numerous bloody points formed by the orifices of veins cut in slicing the brain. Now these are appearances constantly met with in the children who have died of other maladies, but in whom no convulsions had been present. Indeed, I think that the position will not be gainsaid, that the pathological state which favours the development of convulsions does not, any more than that of epilepsy, leave any signs which even the most experienced anatomist would recognise.

There can be no doubt that a large number of the cases of convulsions in children from infancy to fifteen years of age, are referable to the introduction into the blood, or the retention in it, of some new material, and

therefore may be classed with the convulsions to the clinical history of which I am now about to refer.

So excitable is the nervous system of children, that the development of a fever, especially if caused by the introduction of a poison, is very apt to be ushered in by convulsions, which sometimes prove fatal, and certainly will be more likely to prove fatal if the treatment be conducted with the idea of suppressing the present evil, neglecting to look to what the child has to undergo after the convulsions have ceased. All practical men are familiar with convulsions in connexion with the early development of small-pox, measles, and especially of scarlet fever.

An artificial epilepsy may be produced by the administration of certain poisons; as the *œnanthe crocata*, conium, and prussic acid. In poisoning by this latter substance, the epileptic convulsions are frequently complicated to a very great extent with those of the tetanic kind.

These facts prepare us for the doctrine that the retention of certain important excretions in the blood, which their proper organs are incapable of eliminating, favours the development of a state exactly resembling epilepsy in the phenomena of the convulsive fits. When the liver or kidney are in such a state that the elements of the bile, or those of the urine, are not separated in their normal quantity, the patient is very apt to have several convulsive fits, which often terminate his existence. Renal disease is a much more fruitful source of convulsions than hepatic, and the form of disease in which they are most apt to occur is in that small contracted kidney which by some is regarded as a stage of Bright's disease, but which, in reality, is a special morbid state of kidney, a chronic nephritis, frequently produced by gout, and therefore forming the finale of many a gouty patient's career, but often resulting from other causes. In this state of kidney, the gland has shrunk chiefly at the expense of the cortical substance; the growth of epithelium (the immediate agent in the separation of organic products) is greatly impaired, many of the renal tubes are stripped of it, and no development of epithelium at all takes place in them. Hence, while in many of these cases water is freely eliminated, and even a diuresis takes place of an urine pale, and of low specific gravity, the organic products, urea and uric acid, accumulate in the blood, and act, especially the former, as a poison to the nervous system. Any state of kidney, however, if it be unfavourable to due elimination, may give rise to these symptoms; and accordingly I have seen them in that acute affection of the kidneys which is associated with inflammatory dropsy, and with dropsy after scarlet fever, to which my

friend Dr. Geo. Johnson, has given the appropriate name, *Desquamative nephritis*; and also in fatty disease of the kidney, where the accumulation of fat has taken place to such a degree as greatly to congest the Malpighian bodies, and to interfere seriously with elimination.

Among the first, if not the first, to announce the important fact, that imperfect action of the kidneys may induce convulsions with fatal tendency, is Dr. Wilson, of St. George's Hospital, who communicated his views in a valuable paper read to this College in 1833. Dr. Bright and Dr. Addison have also contributed largely to our knowledge of this important subject. The latter distinguished physician has published a valuable practical paper on the affections of the brain which are associated with renal disease in an early volume of Guy's Hospital Reports.

I shall conclude by referring to another, and a very formidable form of convulsions, which, as occurring at a period when the patient stands much in need of mental composure, as well as of physical strength, often occasion serious consequences to her, and great embarrassment to the practitioner,—I allude to the form of convulsions commonly called puerperal.

These convulsions are truly epileptic: the paroxysm, excepting as regards the concomitant circumstances, is not to be distinguished from that of epilepsy. There are the insensibility, the foaming at the mouth, often the bitten tongue, the convulsive actions, and the torpor after the subsidence of the fit. These convulsions are sometimes accompanied by a maniacal state, either before or after the fit, as in ordinary epilepsy.

As far as I can collect from the records of the experience of others (for on this subject I must confess that I have no experience myself), there seem to be three classes of cases in which convulsions form prominent symptoms.

The first class consists of women pregnant for the first time, previously in good health, or, at least, with whom no sign of deranged health has been detected; or, in the words of a high obstetrical authority, Dr. Collins, "strong plethoric young women, with their first children, more especially in such as are of a coarse make, with short thick necks."

The second class consists of women, of greatly impaired health, who may have suffered recently from hæmorrhage.

And in the third class we may place women who suffer from disease of the kidney as manifested by an albuminous state of the urine.

A few cases likewise seem to result from the sudden development of apoplexy during parturition; one or more vessels give

way, and the convulsive paroxysm is induced by the laceration of the brain's substance, caused by the rush of blood into it.

And it must be borne in mind, that some of the patients may have been previously epileptic, and the occurrence of a fit near to, or during the period of parturition, may be merely a coincidence.

Such cases as these, therefore, and those of the apoplectic kind, ought not to be placed in the same category with *puerperal* convulsions.

My friend Dr. Murphy, Professor of Midwifery in University College, in some very valuable lectures lately published by him in the *MEDICAL GAZETTE*, makes a distinction between *sthenic* and *asthenic* convulsions, which is obviously most important in a practical point of view.

Puerperal convulsions occur before, during, or subsequent to parturition; sometimes they will begin before labour commences, come again during labour, and finally recur after parturition. Of 200 cases collected by Dr. Murphy from various sources, forty-six occurred before labour, twenty-five after

labour, and ninety during labour; respecting the rest no precise information could be obtained. Enough, however, is known to indicate that the change in the system preparatory to and during labour has most to do with the convulsive paroxysm.

The statistics of these convulsions show that they must be considered, on the whole, as of a fatal tendency, for of 192 cases 48 died; but it does not seem probable that in these cases the patient died solely from the convulsions. The results denote that the mode of delivery had something at least to do with the fatal issue of the case, as the deaths when turning took place were one in two; whilst those when the labour was natural, were one in four and a half.

Of the post-mortem appearances nothing more is to be said than what I have already stated respecting the other convulsive diseases—namely, that the brain presents an essentially normal appearance. If the patient die in the struggle, the brain is much congested,—otherwise the amount of congestion is not excessive; and, in some of the cases, the brain is in an anæmic condition.

## LECTURE II.

*Are the muscles or the nerves primarily affected in convulsive diseases? Certain postulates respecting the Nervous system. Pathology of Chorea—proofs that the nervous system is primarily affected in it—the brain the seat of morbid action—influence of sleep—nature of the morbid process—a state of enfeebled and depraved nutrition. Chorea a humoral disease. Conclusions respecting its pathology. Treatment of Chorea. Pathology of Tetanic convulsive diseases—particular states of the blood favour the development of Tetanus. Pathology of Trismus nascentium and of Laryngismus. Dr. M. Hall's views. Treatment of Tetanus—use of cold, and of chloroform. Treatment of Trismus, and of Laryngismus.*

MR. PRESIDENT AND GENTLEMEN, — Having in my first lecture briefly laid before you the prominent facts of the clinical history of the principal convulsive diseases with which we are familiar in this country, I shall now proceed to inquire whether, by the aid of physiology in conjunction with our knowledge of the clinical phenomena, we can form any reasonable view of the intimate nature of the pathology of these diseases.

It must be borne in mind that a disturbed state of the muscular system, although a prominent symptom of these maladies, and most commonly *the* prominent symptom, is nevertheless in many of them accompanied with other signs which cannot be referred to the muscular system,—such as disturbed sensations, weakness of mind, delirium or mania, loss of consciousness, coma; but that in others, such as the tetanic convulsions, while the obvious phenomena are confined to the muscular system alone, the convulsions occur with a harmony and symmetry which denote, at least, that the common bond of union of the muscular system, that upon which depends the association of the movements of its various portions—namely, the nervous system—must be in some way or other involved in the disease. Why in Tetanus are the muscles of both sides of the body equally affected? Why in Laryngismus is the muscular convulsion limited in many instances to the laryngeal muscles, extending in some to those of the neck and back? Why in epilepsy and chorea have you an affection not only limited to certain muscles, but exhibiting, as regards the cha-

racter of the convulsion, essentially different features from each other, as well as from those of Tetanus?

It thus becomes a preliminary point in our investigation to determine whether in these various classes of disease the primary and essential seat of morbid action is in the muscular or in the nervous systems,—whether the morbid phenomena of the muscular system be dependent on certain disturbances of the nervous system,—or whether the muscular convulsions are primary, and the disturbed state of the nervous system consecutive and dependent on them,—or whether, in fine, the two systems are affected by one common cause, creating simultaneously disturbances in each, which predominate sometimes in one, sometimes in the other.

And let me here remark, that although we are much more accustomed to see the muscular system affected by changes originating in the nervous system, it is by no means unreasonable, nor inconsistent with fact, to affirm that changes originating in the muscular system may affect the nervous system.

Muscle and nerve generate each its proper force; and experiment teaches us that, while the nervous force ordinarily is the excitant of the muscular force, the latter may excite the former in a similar way. This important point in physiology was first demonstrated by my distinguished friend Matteucci, of Pisa, whose researches on electro-physiology have placed him in the highest rank of natural philosophers.

Matteucci's experiments show that a muscle during its contraction, evolves a force which acts upon a nerve brought into contact with it just as a galvanic current would do, causing the muscles which that nerve supplies to contract instantly. Now, if the muscles of a limb be thrown from some cause into powerful contraction, it is not unreasonable to suppose that the muscular force thus evolved may excite a corresponding evolution of nervous force, which in its turn may create a fresh evolution of muscular force, and thus by successive reactions, great disturbance of both the muscular and the nervous system may be kept up until both forces shall become weakened or exhausted.

In the present state of science we cannot fix on any cause not resident in the nervous system, which would excite extensively active movements of the muscular system *before* the nervous system,—such, for instance, as

would occur in an epileptic convulsion. The only way in which such excitation of the muscular system could be produced, would be through the blood. Now a stimulus operating through the blood directly upon the muscles would be very unlikely to produce active contractions alternating with relaxations, such as occur in epileptic convulsions; on the contrary, it would give rise to permanent or tetanic contractions. The cramps with which the muscles of a limb are affected just prior to gout fixing itself in the joints, might be taken as an example of such a contraction induced in muscle by a morbid excitant conveyed to it by the blood; but even these, although often severe and extremely painful, are very partial. Yet I confess that this explanation of a phenomenon so simple as that of cramp is rather far-fetched; and this becomes at once apparent, when we reflect that a stimulus operating upon a nerve is a much more effective excitant than one operating directly upon a muscle. Take, for example, the best stimulus of muscular contraction, the galvanic force,—the contractions excited by passing a galvanic current through the twentieth part of an inch of the main nerve of a limb are much more powerful than those developed by the direct application of the galvanic power to the muscles themselves.

The argument, then, may be thus briefly stated:—We know by experiment that a cause operating upon a very small portion of the nervous system, may produce considerable muscular disturbance, not of one, but of several muscles; but that the same cause, acting on the muscular system directly, is much less effective, nor does such a stimulus excite *several* muscles by acting only upon one.

Is it not, then, going too far for the explanation of such a phenomenon as *muscular cramp*, to pass by the nerve, and to limit the influence of the cause to the muscle itself?

Is not this argument alone sufficient to direct us to search first in the nervous system for the immediate exciting cause of the muscular convulsions? Nor is there any reason why both systems should not be influenced by the same excitant, which, even though it were more especially accumulated upon the muscular system, would, in all probability, act primarily and most effectively on the nerves, and *through them* on the muscles, which, from their already excited state, would respond the more readily to this stimulus.

This is probably the case in poisoning by strychnine, one of those energetic substances which, if introduced into the blood in only a very minute quantity, seems to multiply

itself\*: thus to become diffused with extraordinary rapidity through the blood, affecting the two great tissues which are mainly concerned in the manifestation of the vital forces,—muscle and nerve,—but evincing a greater affinity for the latter than the former, and therefore acting with greatest energy upon it.

I have felt it necessary to deal with this point thus early in our discussion of the pathology of convulsive diseases, in order that we might, by the elimination of a question, which more or less encumbered the problem proposed, attend exclusively to inquiring into the nature and situation of the changes in the nervous system which give rise to the clinical phenomena of the convulsive diseases already referred to, of which the convulsed state of the muscles is only one.

I do not pretend to have exhausted this question, nor to have answered all the arguments stated with great perspicuity and ability by a distinguished Fellow of this College, Dr. J. A. Wilson, of St. George's Hospital. All that I have said goes only to show that the advocates of the muscular doctrine are so far unphilosophical in their mode of procedure, as they neglect the more simple and obvious explanation of the phenomena, and adopt a more complicated and obscure method.

In considering the share which the nervous system takes in the development of the clinical phenomena of convulsive affections, I must ask your assent to certain propositions respecting the mode of action of the nervous system in health, which the present state of knowledge warrants us in assuming to be proved.

1. That the nervous system, accumulated in masses, and spreading by its nerves throughout the body, mingling with the proximate elements of the various tissues, consists chiefly of a proximate element, of complex composition—namely, albumen, in combination with phosphorus and certain fatty substances, all well suited to the maintenance of constant and lively chemical action.

2. That this material is developed in two forms,—the vesicular or grey nervous matter, the fibrous or white,—and that the combination of these two different kinds of nervous matter, differing probably both in chemical composition, and in the form of their elementary parts, but more strikingly

\* Or it is not unlikely that strychnine, prussic acid, and certain other substances remarkable for the *rapidity* as well as the energy of their action, may immediately on their introduction into the blood assume the gaseous form which is highly favourable to rapid diffusion.

different in the one than in the other, takes place in the masses called nervous centres.

3. That at the points of junction or of intermingling of these two forms of nervous matter, blood is very freely supplied—very much more freely than where the fibrous matter exists alone. At these points there is generated constantly and unceasingly a force, which in its nature resembles very closely the galvanic force, or current electricity, as developed in the galvanic battery, or the magneto-electric machine. Adopting the language of the illustrious Faraday, which expresses with clearness and precision the fundamental phenomena of the electric force, we may call the nervous power a polar force, generated in the centres, and propagated by the rapid polarisation of the neighbouring particles in various directions. The mode of generation of this force, the velocity with which it is transmitted to distant parts of the body, the beautiful provisions adopted for its insulation, have long attracted the notice of physiologists, and have led many to adopt the notion of its identity with electricity. But there are ample grounds for the opinion, that, although nearly allied to electricity, it differs from, and is probably a higher relation of force than that power.

4. The provision made for the generation of this force has an interesting relation to the conditions whereby the galvanic current is generated in the battery. Two dissimilar forms of nervous matter, both bedewed by the same blood, represent the zinc and copper, with the intervening electrolyte of the battery; and as the activity of the battery, and the energy of the galvanic force, depend on the amount of chemical action between the fluid, which is interposed between the metals and one of them, so the activity of the nutrient or chemical changes in the nervous centres determines the degree of development and vigour of the nervous force. And as in the galvanic battery we look to the nature of the metals as well as to that of the interposed fluid, to enable us to judge of the probable quantity and quality of the force generated; so, in the nervous battery, if I may so speak, we must look to the nature of the nervous matter of both kinds, and to the quantity and quality of the blood, to determine the amount and energy of the nervous force developed. Hence we may fairly assume that while the development of the nervous force is much influenced primarily by the nature of the nervous matter, it likewise is greatly dependent on the quantity, but more especially on the *quality* of the blood.

5. That the brain and spinal cord (and I beg to state that I use these terms to signify respectively the intra-cranial and intra-

spinal nervous mass), as the great centres of the nervous system—the great nervous battery of the body—show distinctly a division into two portions: one in which nerves are implanted; the other which has no immediate connection with nerves, and communicates with them only through the medium of the former part. Thus, in the brain, the hemispheres, the corpora striata, the optic thalamus, the tubercula quadrigemina, and the large mass of vesicular matter connected with them and the cerebellum, have no immediate connection with nerves—no nerve is implanted in any one of these centres. The functions of these centres are all of a mental nature,—either the development of intellectual change, or the exercise of volition, or perception, or emotion, or the balancing and co-ordinating of movements,—and they exercise a control over the various segments of that portion of the cerebro-spinal centre in which nerves are implanted through commissural fibres which pass from them to the various segments of that centre. That part of the cerebro-spinal axis in which nerves are implanted,—which was fully recognised by the distinguished Prochaska, and by him designated the *sensorium commune*—this part is in itself incapable of originating any nervous action, except in virtue of some physical change in it: it cannot develop any mental action except in obedience to a stimulus from some of those centres already mentioned as belonging to the encephalon. This part extends from the upper part of the crura cerebri to the extremity of the spinal cord, and all its actions are physical, unless when excited and guided by any of the encephalic centres. It is the centre of all those reflex actions which take place through the spinal or encephalic nerves. This part corresponds to the true spinal cord of Dr. Marshall Hall, who, however, recognises in it two sets of fibres—one which does not pass beyond it, and another which extends from the various segments up into the brain, and which, according to this author, would establish a direct communication between the brain and the nerves. To this latter doctrine, however, there are many and fatal objections.

Having stated these postulates, I shall now proceed to inquire how the phenomena of several forms of convulsive diseases may be explained.

In the choreic affections it may well be inquired, first, whether the parts affected primarily are the nervous or the muscular system.

To this I answer at once, undoubtedly the nervous system; but I hope to show that in all these cases the muscular system

participates in a depraved nutrition, which renders it more prone to sympathise with the irregular motions of the nervous system.

That the nervous system is primarily affected in chorea, appears to me clearly established, 1st, by the fact that the most common exciting cause of chorea is *fright*, or strong mental emotion. Thus in one of my cases, the patient, a girl of 19 years of age, was rudely accosted and laid hold of in the street by a person under the garb of a gentleman, and she became greatly alarmed, and escaped to the house of a relation. Next morning the symptoms of chorea showed themselves. Fright would act upon the nerves,—not upon the muscles. Grief also will induce chorea. A little girl in King's College Hospital, a veritable "Tilly Slowboy," experienced great grief at the loss of a child to whose care she was devoted. A few days afterwards chorea showed itself.

These mental causes do not act primarily upon the muscles, but upon the nervous centres and the nerves; and so frequently do mental causes give rise to chorea, that Ruzs, in investigating 18 cases, traced the origin of 11 of them to fright.

2. In chorea, the nervous system is obviously peculiarly excitable. Choreic patients are much more agitated in the presence of others, than if left to themselves. Of this we have abundant exemplification in hospital practice. When the physician approaches the bed of a patient with chorea, accompanied by a crowd of students, the movements become greatly augmented, and continue so until the cause of excitement has been removed.

This view obtains further confirmation from the fact that we can explain the various phenomena of chorea much more distinctly by referring them to a primary morbid disturbance of the nervous system.

Admitting, then, that there is good ground for the belief that the nervous system is primarily affected in chorea, what portion of that system can we assign as the more special seat of the disturbed action? Is it in the spinal cord, or in the brain?—in that part in which the nerves are implanted, or in that which does not receive nerves?

All evidence tends to show that the part of the nervous system more especially affected in chorea is the *brain*. The hemiplegic character of partial chorea denotes that the disease is of cerebral origin; that the morbid process is most active somewhere above the decussation of the anterior pyramids. Were the spinal cord the seat of disease, you would find commonly that the disorder of movement affected both sides of the body.

Again, its frequent origin from a mental cause points to the brain as its seat. It seems in the highest degree probable that emotions of the mind affect that part of the brain which is most nearly connected with the points of implantation of the auditory and optic nerves—the channels through which emotional excitement is so frequently produced. This part was long ago pointed out by our celebrated Willis as being the corpora quadrigemina, and the large mass of nervous matter which constitutes the greatest part of the mesocephale, a centre which forms the common bond of union between the hemispheres of the brain above, the medulla oblongata below, and the cerebellum behind.

An affection of this part of the brain may, from its extensive connections, produce all the phenomena of chorea, either partial or general: partial, if the morbid action be on one side, as is so often the case in cerebral affections, when the muscular disturbance will be on the opposite side; general, when the affection is on both sides; and in the cases of extensive and acute chorea, the parts involved would not be limited to the centre of emotion, but would extend to the cerebellum, giving rise to the disturbance of the locomotive and co-ordinating powers; and, on the other hand, it would involve the corpora striata and optic thalami, and through them the hemispheres impairing voluntary power and sensibility, and contributing to that imbecility which frequently accompanies chorea of long duration.

The readiness with which sleep controls the choreic convulsions may be adduced as a further indication that this disease has its seat in the brain: sound sleep is undoubtedly a peculiar affection of the brain,—a state in which the particles of that organ seem to assume a condition of quiescence, which contrasts remarkably with that unceasing, ever active change, of which it, more than any other part of the body, is the seat. This state, whatever it be, is incompatible with that which gives rise to the choreic convulsions; and hence the moment that sleep steals over the patient, and overwhelms his senses, the movements of the limbs, however active, cease, but return immediately the state of sleep has withdrawn.

I would remark here that this fact of the influence of sleep in controlling the movements of chorea, tells strongly not only against the view which localizes the morbid process of chorea in the spinal cord, but also against the doctrine which affirms the existence of a certain antagonism between the spinal cord and the brain. Were the spinal cord the seat of chorea, and did such an antagonism as I have referred to exist,

the state of sleep would be the signal for the immediate development of increased and more active movements, and waking would rather tend to control these movements by the renewal of the activity of the brain.

And now we may inquire, what is the nature of the morbid process upon which the development of chorea depends?

With reference to this inquiry, we have the negative evidence of morbid anatomy that the morbid process in the brain is not such as to produce lesions recognisable by our means of observation; and we have the positive evidence of clinical observation that the disturbance which it produces easily admits of repair by natural means, or may be completely controlled by sleep.

If, with these facts, we consider the nature of the movements themselves, which are precisely those of an asthenic state, combined perhaps with some irritation, we get some further insight into the choreic morbid process. The movements are not unlike those which we observe under other circumstances of great debility, and when general nutrition is impaired,—the subsultus of typhus—the paralytic shaking of old age—the tremblings of the habitual drunkard. In all these cases the development of the nervous force is materially impaired—the material of the nervous battery is nearly exhausted—the exciting fluid is weakened; and hence you have a feeble nervous force irregularly developed.

And the general history of the patients who suffer from chorea abundantly confirms the view that the nutrition of the brain, and, indeed, of the whole nervous system, must be impaired. They are ill-nourished, weakly persons, often badly clad, with a poor and pale blood, more or less infected with the matter of scrofula, or of rheumatism, or perhaps with some morbid matter peculiar to chorea itself, or generated in the system by a depraved primary assimilation.

And, looking to the state of the muscles themselves, we cannot pronounce them to be in a healthy state. In choreic patients the whole muscular system is in a more or less soft and flabby condition; and, if you compress the affected muscles, you do not find the firm resistance of a muscle contracting with its normal vigour, but rather that which indicates that the muscle acts with feebleness, and is incapable of perfectly developing the muscular force.

But some one will ask, if the phenomena of chorea are in any way dependent on a depraved and poisoned state of blood, how comes it that they are so frequently localized? This localization of chorea to one side of the body, or even to one limb, or to the muscles of the tongue, is by no means inconsistent with what we know of the laws

of action of morbid poisons. Witness the fixation of the syphilitic poison to the skin and periosteum,—of the scarlet fever poison to the tonsils and lymphatic glands,—of measles to the lungs,—of cholera to the gastro-intestinal mucous membranes,—and look at the morbid localization of the matter of gout, as well as of that of rheumatism, even in cases where the blood is largely charged with it. It in no degree militates against the humoral view of the pathology of gout, that the morbid matter selects the great toe as its favourite locality; nor against a similar view of the pathology of rheumatism, that its morbid poison is prone to attack the heart.

I have now, I hope, said enough to justify me in drawing the following conclusions respecting the pathology of chorea:—

1. That chorea is a disease occurring at a time when the nutrition of the brain is passing as it were through a state of transition, from that of infancy or very early childhood, to that of the adult period, when that organ is peculiarly prone to suffer from mental shock, or other causes of disturbance of the system, and more especially when the blood is in an unhealthy state, deficient in some of its staminal principles, or containing some morbid element.

2. That the part of the brain mainly affected is the centre of emotion, but that the extent to which it suffers is sometimes limited to one side of that centre, sometimes to both, again extending to the cerebellum or to the corpora striata, or optic thalami.

3. That the nature of the cerebral affection is one of weakened nutrition, with some degree of irritation, as poor blood, rendered perhaps impure by the presence of the matter of scrofula, or of rheumatism, or by some morbid matter peculiar to chorea, excites the nervous battery, and causes it to generate its force feebly and irregularly. The centre of emotion thus feebly excited and irritated by the presence of an abnormal ingredient in the blood, extends its feeble and irregular polarity to that portion of the centre of implantation of the nerves, which, as the polar state of the conductors of the battery is regulated by that of the battery itself, exhibit the same enfeebled polarity as the centre in which they are implanted.

Lastly. The disease is essentially one of depraved general nutrition, which must be set right before the symptoms which arise out of the local disturbances can be removed; and this is the point of practical interest which must regulate our treatment of the disease.

These conclusions respecting the pathology of chorea receive confirmation from the facts to which I have already alluded—namely, that choreic movements occur in certain

gouty states, and also in white softening of the brain, arising from diseased arteries. Under both these conditions the nutrition of the brain, as well as that of the nerves and muscles, must be depraved and weakened; depraved, especially in the former instance, by the matter or poison of gout; weakened in both.

And the treatment which is now by general concurrence, and with an unanimity which scarcely exists as regards the treatment of any other disease, pursued in cases of chorea, goes further to confirm these views. No one now-a-days thinks of adopting for this malady any other treatment but one eliminatory and corrective of the various secretions, by the use of purgatives, and at the same time tonic and bracing to the nervous system, by the free application of cold water to the surface, and also tending to improve the quality of the blood, by a nourishing diet, by chalybeates, quinine, and various metallic tonics. And if, notwithstanding the use of these strengthening remedies, the nerves of the limbs still exhibit debility or paralysis, the application of the stimulus of galvanism rarely fails to excite and promote their nutrient activity.

This plan of the treatment of chorea has not been adopted with such general consent without the previous trial of other plans. Sydenham, as is well known, treated chorea by bleeding; but he appears to have had very little experience of the disease. The authority of Sydenham appears to have led his successors to continue this mode of treatment; and even Cullen had not wholly abandoned it,—so hard is it to give up a practice sanctioned by high names. Heberden, indeed, had arrived at a different conclusion. This eminently practical physician says—“Bleeding and purging, and violent medicines, can hardly be judged proper for a distemper attended with no inflammation nor heat, and particularly incident to a very tender age, and to the weaker sex; and which, if left entirely to itself, would, I believe, generally cease spontaneously, and leave the constitution unhurt. This reasoning, he adds, appears to me to have been justified by fact and experience. When they have been used, I never saw any good effects from them, and rather suspect that they have done mischief.”

In the acute form of general chorea, it is very important that the practitioner should not suffer himself to be led by the urgency of the symptoms, and the violence of the movements, into the adoption of any depleting or depressing treatment under the notion of stopping the movements by reducing the powers of the patient. The more you weaken the patient under these circumstances, the more incessant and urgent will the move-

ments become. Great diligence, however, is needed in administering support and stimulants with the utmost freedom, and in throwing in large quantities of quinine and iron, or other tonics, care having been previously taken to unload the bowels by clyster or by purgatives. Great benefit also arises from the free and frequent use of the cold bath; from what I have myself seen, I would say that the greatest immediate benefit arising from the use of the cold bath is from its exciting a disposition to sleep much more decidedly than opiates, which do not generally act favourably in any of the forms of chorea.

The next subject to which I shall beg your attention, is the Pathology of those convulsive affections, of which Tetanus is the type.

We have very satisfactory means of demonstrating that in tetanus the phenomena are dependent on a peculiar state of the nervous system, as it is so easy to produce all the phenomena by the application of stimulants to the spinal cord.

Thus by successive shocks from the magneto-electric machine, transmitted through the spinal cord, the state of tetanus may be produced, and it will remain for some time after the current has ceased to be transmitted. If similar shocks are passed through the chief nerve of a limb, a similar tetanic condition of the muscles immediately follows, but it ceases *instantly* the transmission of the current is discontinued.

So, also, the administration of a few grains of strychnia will produce tetanus; and in cold-blooded animals, as in the frog, opium will also develop tetanus, but in a less degree than strychnine.

The phenomena developed by the transmission of a rapid series of galvanic shocks through the spinal cord, are not to be produced by stimulating any other nervous centre in the same way. You may apply the galvanic power to any part of the brain, only keeping clear of the centre of implantation of the nerves, and you will fail to produce any tetanic phenomena whatever.

The most striking proofs may be obtained to show that the spinal cord is the seat of excitation in poisoning by strychnine. If you give an animal strychnine so as to produce tetanus, you may pith him, and yet the tetanic state will continue. Or in a cold-blooded animal (in which the nutrient actions, although more feeble, are more enduring), if you pith the animal first, and then administer strychnine, the tetanic state will nevertheless come on, and last for a considerable time; but the moment you destroy or remove the spinal cord, that instant all rigidity of muscles disappears, and the stiffened limbs become immediately relaxed. And it is very remarkable that while, by

the introduction of a minute quantity of a particular alkaloid, or, in cold-blooded animals, of opium into the blood, you can produce the state of tetanus with great facility,—it is very difficult, if not impossible, to obtain tetanic symptoms by wounds inflicted for that purpose, even in the most highly sensitive parts; a fact of much interest in reference to the frequent endemic occurrence of Tetanus, and of other convulsive affections of a tetanic kind, and tending to show that a particular state of the blood is favourable to the development of Tetanus.

The symptoms of Tetanus as it affects man and some of the lower animals, the horse for example, so precisely resemble those of the disease as it is artificially produced, that it is impossible to resist the conclusion, that not only the part of the nervous system affected, but also the nature of the affection, must be the same in both (if I may use the distinction), the artificial and the natural disease.

The part of the nervous system affected is clearly the spinal cord, and at least the greater part of its intracranial continuation, the medulla oblongata; and it seems highly probable that the anterior horns of the gray matter of the spinal cord and the centres of implantation of the encephalic nerves, are more particularly affected.

And the nature of the morbid process is not one of inflammation, nor does it arise out of any alteration in the *quantity* of blood flowing through the cord; it is not due to any congestive state, nor to any anæmic state: it consists of an exaltation in the special property of the nervous matter; an exaltation in the generating power of that portion of the great nervous battery in which the chief cerebro-spinal nerves are implanted: in short, it is a state of exalted polarity of that centre, produced either by the extension of a similar polar state, developed at the distal extremity of some nerve, or by some modification of the nutrition of the cord caused by the development in the blood of a material having similar properties to that of the alkaloid, strychnia.

Or, in the language of physics, the exalted polar state of the battery is due either to an altered condition of its generating and conducting plates, or to an increased activity of the fluid by which they are excited—perhaps, indeed, to both.

Such, then, is the pathology of Tetanus. A man receives a wound—this wound occasions an irritated, a polar state of certain nerves,—and this polar condition is communicated by a process of induction to the whole spinal cord, which, by some modification of the nutrition of the cord due to some altered state of blood, assumes that state the more readily, and retains it the longer.

Unless we admit that the blood has some

share in the production of the phenomena, we shall fail in explaining the occurrence of Tetanus without injury, or the tetanus of infants, but more especially we shall fail in explaining its greater proneness to be developed in certain localities, or in certain states of atmosphere and climate.

And surely the fact to which I have already adverted, of the much greater facility of inducing this polar state of the spinal cord by the introduction of certain substances into the blood even in almost infinitesimal quantity, than by mere mechanical irritation of the nerves, strongly points to and favours the conclusion, that a change in the natural condition of the blood may greatly promote, if not wholly cause, the development of the tetanic state; and this change in the blood may be caused either by the introduction into it of some new material from without, or by the generation within it of some new matter possessing highly poisonous qualities.

All that I have said respecting the clinical history of the Trismus nascentium, and of Laryngismus stridulus, shows that these diseases must be precisely similar to tetanus in the essential points of their pathology.

The Trismus, indeed, is only a very acute form of Tetanus, and its endemic character, and its proneness to occur under circumstances unfavourable to free ventilation and good general nutrition, are highly confirmatory of the view I have ventured to advocate of the probable influence of a morbid state of blood in promoting the polar state of the spinal cord and medulla oblongata.

Laryngismus differs from Tetanus in the fact that the polar state of the nervous centre is less permanent, and that the medulla oblongata is more affected than the spinal cord. In Tetanus, the spinal cord is more affected than the medulla oblongata. The proximity of the medulla oblongata to the centre of emotions explains the extreme excitability of children suffering from Laryngismus by mental emotions.

In Laryngismus, there is nothing whatever of an inflammatory nature, nor is there anything of a congestive kind: it is most important that practitioners should be well impressed with this fact.

Whatever congestion may arise in the nervous centres is venous, and is caused by the convulsive paroxysms, which impede respiration, and consequently delay the return of the venous blood. It is, then, an effect and not a cause of the convulsive state.

We have in laryngismus only an exalted polarity of the nervous centres, caused either by peripheral irritation, as that of dentition, or by a morbid blood produced by imperfect feeding, artificial nursing, bad air, neglect of cleanliness, or associated with a highly scrofulous diathesis.

These views of the peculiar condition of the spinal cord in Tetanus and the allied affections were, so far as I know, first put forward by Dr. Marshall Hall, to whom science is under great obligation for the introduction of sounder views on many points in the application of physiology to pathology. That distinguished physiologist, with whom I regret to find myself at variance on many points, in an interesting and valuable essay on convulsive affections, makes these remarks:—"Tetanus and Hydrophobia consist in an induced spinal erethismus, the paroxysms consisting of augmented or excited spasm, from external excitants. The infant, under the influence of what I will designate the convulsive tendency, susceptibility, or erethismus, may therefore be compared to the patient attacked by tetanus. The causes are similar—irritated nerve, inducing spinal erethismus, and great susceptibility to excited reflex actions; the effects are analogous—tetanoid affections."

Hitherto the various plans of treatment pursued in Tetanus have received but a small amount of success. I am inclined to attribute their failure partly to the fact that practitioners have not sufficiently kept in view the important point, that death takes place, not by any disorganization of important vital organs, nor by the destruction of processes essential to life, but by the exhaustion consequent on the frequent renewal of the paroxysms of tetanic convulsion,—and partly, also, to this fact, that our treatment has been hitherto so empirical, that no definite plan seems ever carried out with sufficient fixedness of purpose in a sufficiently large number of cases.

The pathology and clinical history of Tetanus point out three objects which the practitioner ought constantly to keep in view in the treatment of it.

1. To support the strength of the patient as much as possible, so as to oppose the exhausting effect of the convulsive paroxysms.

2. To remove all possible sources of irritation or of depravation of the blood, in vitiated secretions, bad diet, impure air.

3. To diminish and reduce the exalted polarity of the nervous centres to their normal condition, and if possible, to effect this by means which will not exhaust or reduce the powers of the patient.

Now, the first indication is of primary importance: if the patient can be duly supported, we gain time, and we have abundant proof that the natural powers of the system are often capable of doing the rest without any assistance from art.

To accomplish this object, the practitioner should set himself diligently to devise means of giving nutriment and stimulants

in the manner in which they may be most readily assimilated. As a general rule, this may be done best by giving them frequently, and in small quantities; and it is wonderful how much may be taken in this way if they are only administered with care and judgment.

Any process of depletion,—by loss of blood, or by evacuants of any kind, carried to a great extent, would be highly objectionable, as being calculated to oppose the object in view.

In addition, then, to the various articles of food best adapted to the assimilating powers of the patient, and also to wine, or other alcoholic stimulants, the practitioner ought not to withhold the liberal use of quinine, ammonia, iron, or other drugs, which experience tells us exercise a tonic power over the system in general,—and he may give them in large doses.

The second point may be best attained by means calculated to promote the various natural secretions, especially those of the bowels, skin, and kidneys. But in administering remedies for these objects, care should be taken not to use those of a violent and irritating nature: drastic purgatives should be avoided; too profuse sweating or diuresis should be guarded against; and the practitioner should bear in mind that the object is to alter and improve, not to pull down.

To obtain the third object—namely, to reduce the polarity of the spinal cord, is at once the most important and most difficult. The various sedative remedies—opium, hydrocyanic acid, belladonna, conium, tobacco—have been fairly tried and failed. Opium is not a sedative to the spinal cord; its use in tetanus may be laid aside excepting in small doses as a sudorific. In cold-blooded animals it exalts the power of the spinal cord, and it is not impossible that in warm-blooded animals it may have a similar tendency: it is, therefore, a remedy of little value in tetanus, save as a sudorific, and in large doses it may be of an injurious tendency.

Neither is hydrocyanic acid a sedative to the spinal cord; on the contrary, it tends to produce epilepsy, and to excite the polar state of the cord by induction from the brain.

Belladonna has a decidedly sedative influence, but it disturbs the action of the brain so much, that it is not a safe or manageable remedy. The same may be said of conium. Tobacco undoubtedly reduces the polar state of the cord, but it produces at the same time a state of fearful depression. It is likewise an unsafe and not a manageable remedy. I have seen more than one patient die, *cured* of Tetanus, under this remedy.

There are two agents which certainly

exert a considerable power over the polarity of the spinal cord, which have not yet been tried sufficiently fairly, and which I think fully deserve to be put extensively to the test of experience. These are—cold and chloroform.

Of cold I can speak favourably from my own experience; I have tried it by the application of ice in ox gullets to the spine, taking care to renew them frequently. I have adopted this practice in Tetanus, and in convulsions in which the spinal cord was much involved, and in other more partial states of exalted polarity of the spinal cord, and with a sufficient amount of success to justify me in forming a very favourable opinion of the powers of cold as a depolarizing agent.

In order to give the remedy a fair trial, great vigilance is required on the part of the attendants of the patient to renew the bladders very frequently, otherwise they become elevated to the temperature of the body. This should never be permitted, as it is only by the long-continued application of cold that the spinal cord can be reached by it.

Another point which the practitioner who uses cold in this way must keep in view is, that when the cold fairly reaches the cord, its influence is shown by a marked depression of the action of the heart, which leads to general depression and debility. During the application of the bladders in this way, increased vigilance will be necessary in the administration frequently, and at short intervals, of nutritious and stimulant substances.

Sometimes the depression of the heart's action reaches a point below which it would be unsafe to suffer it to go. Under these circumstances the ice must be removed, and kept off until the heart in some degree recovers itself, when it may be resumed. Thus by removing and reapplying the bladders from time to time, the influence of the cold may be kept up sufficiently long to produce a favourable effect upon the cord without producing such an amount of depression as may operate to the disadvantage of the patient. But to carry out this plan, great vigilance, and care, and judgment, will be needed on the part of the practitioner, in order that all precautions may be duly observed, and that the patient may be encouraged to persevere in the use of a remedy which involves some degree of personal discomfort.

Of chloroform, in tetanus or tetanoid affections, I have no experience. Two years ago, however, I performed some experiments, by which I satisfied myself that the analogous agent, ether, when introduced into the system by inhalation, exercised a most powerful controlling influence over the

polar state of the cord, as induced by strychnine.

In these experiments I found that previous etherization retarded the polarising influence of the strychnine; and that, after poisoning by strychnine, life might be prolonged for a considerable time by the administration of ether.

A guinea-pig was thrown into tetanus by a quarter of a grain of strychnine: immediately the tetanic symptoms showed themselves, he was etherized; the state of tetanus immediately passed off; in a short time, however, it recurred, and was again controlled by ether: and by thus repeatedly etherizing the animal when the tetanic symptoms reappeared, life was prolonged for many hours, whereas another animal of the same size, and poisoned at the same time by the same dose, but not etherized, died rapidly.

These experiments illustrated in a very striking manner the antagonistic power which ether (and chloroform, as possessing identical virtues) can exert over the polarity of the cord. The great point which ought to engage the attention of practitioners in the application of these agents to the treatment of such cases as Tetanus, is to know how to avail themselves of its depolarizing power, and at the same time to guard against the injurious effects of its depressing influence. Chloroform weakens the force of the heart,—sometimes diminishing, at other times increasing the frequency of its action. Hence, while administering it in tetanus, we shall find it necessary to use with increased diligence the means calculated to uphold our patient's strength; and we must take care that the sleep of chloroform does not prove to him the sleep of death, by depriving him too long of those supplies which alone can counteract the destructive asthenia.

In the absence of any experience of the use of chloroform in tetanus, I should advise that a patient be not kept under its influence for any length of time, and that if, after the administration of it, and after its more profound effects have passed away, he should continue to sleep, the sleep should be interrupted at intervals of an hour or of half an hour, according to his strength, in order to administer support. If the patient does not sleep after the administration of the chloroform, and if the effects pass off, then it should be administered a second time, and a third time; after which several hours should elapse before it is again administered, and then it should be given in a similar manner, taking care that it be given not more frequently than at three, or, at most, four different periods in twenty-four hours.

These suggestions, however, are prior to experience: they want the support or the modification which observation only can give, and I should not venture to offer them

were it not that it seems to me of great importance that we should not discard an agent so powerful as chloroform, against certain morbid states, without giving it fair play, sifting its merits and demerits, and employing it in various ways, so as, if possible, to secure the benefit of the former, and to guard against the disadvantages of the latter. In some of the cases of tetanus in which I have heard of chloroform being employed, it has seemed to me that the patient was kept too long under it at once,—that its renewal was at too short intervals,—and that sufficient precautions were not taken to counteract its depressing influence.

We have yet much to learn respecting the application of so powerful an agent as chloroform to the relief of nervous affections. But I dare say the time is not far distant when we shall be able to form a more exact judgment of its powers and applicability to these diseases. To those who are interested in this subject, I recommend the perusal of some interesting essays now in course of publication in the *MEDICAL GAZETTE*, by Dr. Snow.

The rapid course which trismus nascentium generally runs, and the feebleness of the little subjects of it, do not permit us to expect that we shall ever be more successful in the treatment of that fearfully acute tetanus than our predecessors. What we may really hope for is, that as careful attention to cleanliness and ventilation, and the early good management of the infant, has greatly diminished the number of cases of the disease,—so improvements in these respects may yet blot it out entirely from the list of ills to which the new-born infant is exposed.

The views of the pathology of the disease which I have endeavoured to advocate, suggest that the most promising mode of treatment is that by very frequent copious affusion of cold water, especially over the back and along the spine; such precautions being taken as may ensure the speedy restoration of the animal heat, and prevent its undue depression: such as good friction of the skin, quickly enveloping the infant in warm blankets after each affusion, maintaining a uniform temperature of the apartment, which, however, should at the same time be most carefully ventilated. To these should be added good feeding, and such attention to the state of the bowels as will secure the discharge of their contents without any active purging, which might prove an additional cause of exhaustion. Change of air would be most desirable, when it would be possible. But these cases run a course so rapid that it rarely happens that there is time for the adoption of such means.

Judging from the reports of cases as they

are from time to time published in the medical journals, I would say that the views of practitioners are becoming pretty well agreed as to the most effective treatment for laryngismus stridulus. The points which should especially engage our attention, are—first, as to the best means of removing all sources of irritation; secondly, as to the most appropriate mode of promoting a healthy and vigorous nutrition; thirdly, the proper measures to adopt for diminishing that extreme excitability of the nervous system which keeps up the frequent repetition of the convulsive paroxysm.

It is very important that all sources of irritation should be removed as early as possible. What we most frequently find to excite irritation, are—accumulations or morbid secretions in the bowels, and the inflamed gums in dentition. The first may be easily removed by the action of purgatives, so regulated as not to impair the strength of the patient, nor themselves to act as irritants. Inflamed gums are likewise easily relieved by free incision with the gum lancet, which it may be necessary to repeat once or twice. But I must remark that the gum lancet must not be used indiscriminately; to many children the mere attempt to open their mouths occasions great disturbance,—nay, a greater disturbance than the inflamed gums which it is sought to relieve,—and it may excite a paroxysm which may prove fatal. Hence I hold that this operation ought not to be performed unless it be rendered absolutely necessary by *a really inflamed and swollen state of gums*. Under such circumstances, the relief to be obtained by the free division of the inflamed mucous membrane is frequently so immediate and so great, that we may legitimately encounter the risk which the operation occasions, in order to obtain so great a benefit. But where there is no inflammation of the gums, where it is plain that the teeth are not impeded in their advance by inflamed mucous membrane, no good can be obtained by the incisions.

Still more objectionable must be the practice of frequent or daily division of the gums: by such a procedure you expose the child to a daily repetition of a scene of great excitement, without any corresponding benefit. For what can be gained by such a practice? The teeth are not delayed in piercing the gums in consequence of any mechanical obstacle; their tardy progress is the result of a slow and inactive nutrition: in short, they grow slowly, and therefore are slow in cutting their way through the gum. Is it to be expected that the frequent repetition of the operation of cutting through the gums will hasten the growth of the young teeth? I think not: on the contrary, there appear to me to be some strong grounds for the apprehension that such operations may

retard the growth, and injure the nutrition of the young teeth.

There are many cases, however, in which dental irritation does not exist; that is, in which there are no teeth exciting painful inflammation of the gums. In such cases there will not be any need for surgical interference with the mouth. But in these and in all cases the practitioner will have to apply himself diligently to devise the best mode of promoting a healthy and vigorous nutrition. And for this purpose he must attend to the quality of the food, its quantity, the manner in which it is given, the periods of feeding: on one or all of these points he will certainly find something to alter and amend. Either he will find that an infant is being brought up by hand when it ought to be nursed, or it is nursed too often, or with milk of bad quality, or it is fed in some imperfect manner.

Time would fail me, were I to enter upon the large and important subject of the best method of feeding infants. I must content myself with briefly referring to certain precautions which ought always to be taken, in order to insure to the child the full benefit of its food. First, it should be fed *slowly*: this rule, so important in the adult, is not less so in the infant; and the question, in cases where the infant is not nursed, between feeding with the bottle, by suction, or with the spoon, entirely turns upon this—whether, by the one or the other method, it is fed sufficiently slowly. The former mode is the preferable, as coming nearest to the mode of prehension most natural to the infant—that, namely, by suction; it is *objectionable*, as affording to careless nurses too easy opportunity of rapidly feeding the child.

A second point of importance is, that the child should not fast too long, and that too much food should not be given at each meal. Frequent meals carefully adjusted in quantity are undoubtedly best suited to the infantile economy, which is the seat of rapid growth, and therefore needs frequent supplies. Attention to the *rate* at which the food is administered will help in adjusting the quantity: a child fed slowly will take less food than one hastily and rapidly fed.

Lastly, the quality of the food should be determined mainly by the period of infancy, but also by the power of digestion evinced by each individual. Speaking generally, in the earliest periods the food should be simple and uniform: if the infant cannot be nursed by its mother or by a healthy wet-nurse, its food should be as nearly as possible assimilated to human milk. As the infant advances in life, farinaceous matters obtained from the flour of wheat, as containing gluten, should be gradually and cautiously added; and after the fourth or fifth month,

gelatinous and albuminous matters derived from animal food may be given in small quantities.

But whilst the practitioner is carrying out these reforms in diet, he must not forget that he is urgently called on to diminish the frequency of the paroxysms. For this purpose he must cut off as much as possible all external causes or means of excitement, and he must adopt measures to diminish the excitability of the nervous system.

Let me here remark, that there are few occasions in which the practitioner stands more in need of that support which is so fully derived from confidence in himself and in his principles, than in the treatment of such a malady as laryngismus. The anxiety of parents, always great when a child is in danger, is wound up to its highest pitch when it is afflicted with a malady which threatens every moment to deprive him of life. They therefore naturally look most eagerly for the speedy adoption of the means which will mitigate the severity of the paroxysms, and diminish their frequency. Under these circumstances the practitioner must let it be seen that, while he is fully alive to the imminent danger in which his little patient is placed, he has a sufficient command of resources, and he is guided by certain principles of action, which he will carry out with decision and vigilance.

The plan of dashing cold water in the face, to avert an impending paroxysm, or to shorten an existing one, is generally attended with success, as regards the paroxysm. It is also, I think, useful in shortening the intervals between them, by diminishing the excitability of the cutaneous nerves. This dashing with cold water should be administered liberally and rapidly, notwithstanding the concomitant inconvenience of wetting the child's clothes. In some instances the operation of drying the child and putting on fresh clothes is itself useful. On similar principles, I find it very useful to have the child well sluiced with cold water night and morning, and sometimes even a third time in the day. A large quantity of cold water should be dashed *rapidly* upon the back and shoulders, and along the spine,—a moderate pitcher of water being *at once* emptied upon them. The value of the remedy is greatly enhanced by the suddenness of its application. In this suddenness consists the skill of its administration; and unless this be well done, it had better not be used. For this reason it is expedient that the practitioner should at first superintend its administration himself, and teach the nurse how to apply it.

The cold affusion not only diminishes the excitability of the cutaneous nerves; it likewise improves general vigour, stimulates the circulation of the skin, and consequently

increases the action of that great emunctory, and contributes to promote a more healthy state of general nutrition.

If drugs are used, the most valuable (in addition to such purgatives as may be necessary to keep up a moderate action of the bowels) will be found to be those of the tonic and antispasmodic class. I have myself seen most advantage from the use of iron in combination with alkalies, potash, or ammonia. *Assafoetida* is extolled by some. Sir H. Marsh commends the old *Tinctura fuliginis*. The infusion of bark, the liquor cinchonæ of Battley, the tincture of bark, quinine itself (the chief objection to which is its intense bitterness of taste), will all prove more or less useful.

Depletion by bloodletting, whether general or local, may, I think, be completely discarded from the list of appliances at the command of the practitioner in the treatment of this malady. In most instances it may do great harm by impoverishing the blood, and increasing the excitability of the nerves, and weakening the patient. I have not met any cases in which it has appeared to be advisable, unless as a means of satisfying the prejudices of parents or others. The disease is clearly not inflammatory; all who have seen it on a large scale agree in this view: and whatever congestion may arise in the nervous centres is the result of the paroxysms, and not their cause. Some may think it advisable to use depletion for the relief of this congestion. Upon this point it is obviously impossible to lay down any general rule. In my own experience I have not met with any instance of congestion of this kind which has not been sufficiently easily relieved by purgatives and attention to diet.

Of all the remedies which are adapted to increase the tone and vigour of the nervous system, none is so potent as the exposure of the child to fresh air, especially to that of a locality different from that in which it had been accustomed to live. There are few who cannot bear testimony to the efficacy of this practice. In principle it should be carried out throughout the whole treatment of the case. The child should be kept if possible in an airy, well-ventilated apartment, of moderate temperature. Every thing like what is commonly called "coddling" should be avoided. The child should be lightly *but sufficiently* clad, and be kept free from all such articles of clothing as might in any way impede the superficial venous circulation, or interfere with the free action of the respiratory muscles. If complete change of

air cannot be obtained, it is a good plan to take the child out daily in a carriage into the country, to some distance from its usual residence, where, if the weather be not too cold, it may be carried about in the open air.

Many practitioners have a fear of adopting the change of air system, lest the patient should incur the risk of bronchitis. This fear is, within certain limits, well founded. But I think it right to mention here, what I have not seen referred to elsewhere, namely, that one of these patients may have the physical signs of bronchitis without the disease. A spasmodic state of the bronchial muscles frequently coexists with the spasm of the glottis, often precedes it, and remains for a considerable time after it. This is accompanied by a generally diffused *ronchus*, such as would occur in bronchitis. It subsides immediately the general state of spasm subsides. Of these points I have had frequent opportunity of satisfying myself.

Children often fall into what I would call a *laryngismoid* state, *i. e.* a condition in which the tendency to laryngismus is manifest, with great nervous excitability. If the child cough, inspiration is attended with a croupy noise. Crying, or any forced respiratory act, exhibits similar phenomena. But no regular convulsive paroxysm occurs. In these cases great benefit is derived from daily exposure to the air, and from a treatment conducted upon the same plan as for the fully developed malady.

Such, then, are the principles and the plan of treatment of laryngismus. It is but just that I should mention that these views of the pathology and of the treatment of this disease have long been *practically* recognised by men of experience. The late Dr. Cheyne has left a brief but clear description of the disease, published thirty years ago; and I know no more accurate and clear account of the disease than that given by my friend Sir Henry Marsh, in the Dublin Hospital Reports, in 1830, where principles of treatment similar to those I have endeavoured to lay down, are inculcated. Dr. Marshall Hall has also ably illustrated the pathology of the disease, and given some judicious instructions for treating it. Some valuable practical remarks on the treatment of the disease were published by Dr. Robertson, of Manchester. And, lastly, a very able essay upon it has lately been published by Dr. James Reid, of this city, which is interspersed with excellent observations, both pathological and practical.

In my next lecture I shall discuss the pathology and treatment of epilepsy.

## LECTURE III.

**THEORY OF EPILEPSY.**—*Phenomena essential to epilepsy—Epileptiform convulsions dependent on superficial disease of the brain—Case—Points to be explained by an adequate theory of epilepsy—Epilepsy not due to primary irritation of the spinal cord—What part or parts of the brain affected in epilepsy. Not the Medulla oblongata, nor the Corpora Striata and Optic thalami—Affection of the hemispheric lobes explains the loss of consciousness—Can superficial irritation of these parts of the brain cause convulsions? Periodical action of the brain—Sleep—Dr. M. Hull's theory of sleep—Objections to it—The mesocephale affected in epilepsy—Experiments by the magneto-electric machine on different parts of the cerebro-spinal centre—Clonic convulsions caused by irritation of the mesocephale—Conclusion as to the parts of the brain concerned in the production of epilepsy—Inquiry into the nature of the cerebral disturbance in epilepsy—Is it inflammation? Congestion? Anæmia? Theory of the disease—Humoral view—Treatment by drugs—by discipline and regimen—Use of chloroform in epilepsy.*

MR. PRESIDENT AND GENTLEMEN, — To frame an adequate theory of the pathology of epilepsy, is, I feel, a task of no ordinary difficulty. Although much has been done of late years towards forming exact views of the functions of the several parts of the great cerebro-spinal axis, we are still very uncertain as to the precise influence which each part has on the others,—as well above as below it,—and how far a disturbance of any one may involve the rest in a like disturbance. While such obscurity exists respecting the office of important parts of the nervous system, it is impossible not to approach the subject of the pathology of epilepsy without a feeling of hesitation and mistrust.

The leading and pathognomonic features of true epilepsy are these:—loss of consciousness and sensibility, most frequently accompanied by convulsions of the kind which I have described as epileptic, *i. e.* of movements which consist of alternate relaxations and contractions of muscles rapidly succeeding each other: these paroxysms occurring at intervals of variable length, with something of periodicity, the patient during the interval recovering himself completely. These phenomena are most frequently

ushered in without any warning whatever, so that the patient is suddenly, and on the instant, seized, rendered senseless, more or less violently convulsed, and then awakes more or less suddenly. In many instances all this is done in scarcely as much time as it takes to describe the phenomena; at other times the fit is of much longer duration.

Now it must be carefully kept in view that the complete epileptic paroxysm is compounded of many phenomena, of which the primary and constant ones are the loss of consciousness, and the disturbances of sensibility which usher in the fit, or remain after it. We never meet with instances of epileptic convulsions affecting the whole system, and recurring periodically, which are not ushered in with loss of consciousness, or accompanied by it. We do, however, frequently meet with instances of the sudden loss of consciousness, lasting for a longer or shorter time, without any muscular disturbance; and this loss of consciousness is accompanied by a peculiar vacant look,—an aspect entirely devoid of all indication of intelligence,—expressive, indeed, only of the temporary abeyance of the intellectual power, and attended with a dilated condition of the pupils;—a feature of the epileptic state quite as constant, perhaps, as the loss of sensibility and consciousness, and which is very apt to remain, although in a less degree, after the fit, and throughout the entire period intervening between the fits.

There are, however, certain epileptiform disturbances of motion not accompanied by loss of sensibility, which it may be as well that I should notice here, partly to draw the line of distinction between them and the true epileptic convulsions, and partly because a right appreciation of the nature and character of these pseudo-epileptic movements will assist me in the course of this discussion. These movements occur in paroxysms: they consist of alternate rapid contractions and relaxations, with, however, a great tendency to muscular cramp or rigidity. If, for instance, the arm is affected, the movements will consist of rapid flexions, alternating with extensions, but in the intervals the muscles will feel firm, and more or less rigid. Sometimes, however, the reverse takes place, and a state of paralysis ensues upon a violent convulsive paroxysm. In such cases the convulsion is never general,—always partial, and confined to one side, one arm or one leg, or both, with the side of the face. In many instances

the movements are no more than twitchings, and in all, the experienced eye would readily detect the difference between their half tonic, half clonic nature, and the forcible plunging truly clonic character of the proper epileptic convulsion.

Such movements as these accompany superficial irritation of the hemispheres of the brain or of the cerebellum. I have seen them most frequently in children, associated with the so-called tubercular meningitis; or, more properly, meningitis and encephalitis, excited by the presence of tubercles in the pia mater.

A case of this kind came under my observation some years ago, which afforded a striking instance of these pseudo-epileptic movements, as I would call them. As the case has been made public by Mr. Dunn, the surgeon under whose care the child was, and who kindly afforded me the opportunity of seeing him, I shall refer to his clear and able account of it published in the *Medico-Chirurgical Transactions* for details, and content myself with mentioning those points which are most applicable to my present purpose.

The boy was two years old, and of strumous habit. He had for some time been failing in health, and shown excessive perverseness of temper. On the 7th of October he awoke, as he was accustomed, between six and seven o'clock in the morning, and while amusing himself with his sister in bed, and attempting to turn over, his left hand suddenly began to take on a jerking or convulsive twitch, which did not extend beyond the wrist. Mr. Dunn saw the patient immediately, and found that these convulsive jerkings formed the only symptom exhibited by the child. He was laughing and talking, quite sensible, and in every other respect seemed perfectly well. These jerkings continued for about twenty minutes, when they subsided, and the child was free from them the whole day. The next morning, the 8th, at nine o'clock, the convulsive movements returned, and continued for half an hour. The day after this the jerkings returned again at seven o'clock in the morning, but were much slighter than before. On the 11th, the convulsive movements returned again, and were not confined to the upper extremity, but involved the leg as well, the face also, and the angle of the mouth. On the 12th and 13th, the child had two fits in the day, affecting the whole of the left side: he cried and screamed towards the termination of the fits, but he was sensible throughout them. Each fit was followed by profound sleep.

Early in the morning, the 14th, Mr. Dunn was called up in consequence of a very severe paroxysm, which lasted three hours: the convulsive movements, affecting the left side

from head to foot, were very severe: at times he screamed, at others he was quiet, and *throughout knew all about him.*

Under the increased frequency and severity of these attacks, and an extension of them, though in a slight degree, to the right side, with other symptoms of irritation of the brain, the patient died, and, on examination, it was ascertained that there existed an extensive deposit of tubercular matter on the surface of the right hemisphere of the brain, the opposite to the side on which the convulsions took place, and that a slight and recent deposit had taken place on the surface of the left hemisphere, and that the cerebral substance around the tubercles was in a state of red softening, interspersed with inflammatory products, in considerable quantity. The tubercular deposits exciting inflammation of that portion of the brain which was in their immediate vicinity, no doubt gave rise to the convulsive movements on the left side, as well as to those which had begun to show themselves on the right side of the body.

I would lay it down, then, that epileptiform convulsions without loss of consciousness are essentially distinct from true epilepsy, and may be developed by even a slight morbid irritation of the brain.

You will see that what we have got to explain by any theory of the pathology of epilepsy, is—

1st. The sudden occurrence of complete loss of consciousness.

2nd. The sudden privation of voluntary and locomotive power, with more or less violent epileptiform convulsions.

3rd. And all this occurring *at certain periods*, with more or less uniform and regular intervals, during which the patient may, and frequently does, return to a condition perfectly normal.

Lastly, our theory must explain why it is that these fits are so often accompanied with a maniacal state, and how it happens that their frequent repetition tends to destroy the power of the brain as the organ of the mind; and, moreover, greatly to impair the whole nervous force.

It seems needless to occupy time with discussing whether, during these formidable paroxysms of epilepsy, or, as it used to be called, of "falling sickness," the muscles or the nerves are primarily affected. All the obvious phenomena point to the nervous system as being chiefly engaged, and to the muscular convulsion as entirely consecutive to, and dependent on, the disturbed state of the nervous system. I would remark, however, that the enormous development of muscular force which must be produced during the convulsive paroxysm, must, in conformity with the discoveries of Matteucci, alluded to in my last lecture, have a most

powerful reaction upon the nervous system.

The first point, then, which should occupy our attention must be as to the part of the nervous centres which is mainly disturbed in this disease—whether the spinal cord or the encephalon; and, if the latter, whether the whole or part of it: for it cannot be admitted that the disease is one of the nerves only: the nerves, indeed, may convey irritation to the centre, and again reflect that irritation to the muscles; but it would be impossible for such an amount of irritation as would create the paroxysm of epilepsy to exist without giving rise to great disturbance of the centres.

I think it cannot admit of doubt that in epilepsy the spinal cord is by no means primarily affected. That it is to a certain extent disturbed during the complete paroxysm which is accompanied by muscular convulsions, is evident; for otherwise the exciting cause of the convulsions could not reach the muscles of the limbs: but then this disturbance is secondary.

The nature of the convulsions shows that the spinal cord is not primarily affected. In my former lectures I have shown that the character of the convulsion dependent on primary irritation of the spinal cord is *tetanic*, with more or less permanent rigidity of the muscles and opisthotonos; and this can be clearly demonstrated by experiment. But although the convulsions of epilepsy are often complicated with a good deal of tetanic spasm, they nevertheless always present features which distinguish them, in a very marked way, from the tetanic convulsions, by the alternate contraction and relaxation,—violent combined movements, resembling voluntary acts, succeeded by relaxations, to be again succeeded by violent contractions.

Furthermore, if the spinal cord were affected primarily, we could not explain the sequence of the phenomena in the complete paroxysm of epilepsy.

If the spinal cord were primarily disturbed in epilepsy, the sequence of the phenomena would be—first, muscular disturbance, in consequence of the state of disturbance of the cord; then impairment of consciousness, in consequence of the extension of the disturbed state of the cord to the brain.

Now the natural sequence of the phenomena is—first, loss of consciousness; secondly, muscular convulsions.

Moreover, the supposition of the primary existence of disturbance of the spinal cord leaves us altogether without the means of explaining the phenomena of that form of epilepsy which in its consequences to the patient is quite as formidable as that accompanied by convulsions. I mean that form which consists simply in loss of

consciousness, which lasts for a variable term, and then the patient recovers himself.

Lesions of the spinal cord, it is now well known, do not affect consciousness. You may have nearly the entire cord severed from the brain, and yet consciousness will be retained. A man falls from a height, and fractures his cervical vertebræ, smashing a considerable portion of the cervical region of the spinal cord: he is taken up paralysed, in both sensation and motion, at all points below the head, but *consciousness is undisturbed*.

In the convulsive diseases confessedly spinal in their origin,—namely, in tetanus and laryngismus,—consciousness is undisturbed, or but slightly affected, however violent the convulsions may be.

It seems, therefore, certain, not only that a highly disturbed state of the spinal cord will not impair consciousness, but also that that state of disturbance of the cord is not prone to extend itself to the brain, and to interrupt the functions of that organ.

And these arguments applied to the ordinary spinal cord of anatomists—the intraspinal nervous mass—are equally opposed to the location of the disease in the true spinal cord of Marshall Hall, or in the sensorium commune of Prochaska. The sensorium commune of Prochaska is the spinal cord *plus* its intra-cranial continuation as high up as the crura cerebri. But primary irritation of this latter part produces the same phenomena as that of the spinal cord, with the addition of more or less laryngismus: therefore it is not the part first affected in the epileptic fit.

It is plain, then, that the primary disturbance in the epileptic paroxysm must be located in some part of the brain.

Now this organ is divisible into certain parts, each of which, there can be no doubt, enjoys a separate proper function, notwithstanding that all the parts are intimately united to each other.

These parts are—

First. The hemispheric lobes, consisting of the convolutions and the large mass of fibrous matter connected with them.

Second. The corpora striata and the optic thalami, in intimate connection with

Third. The medulla oblongata.

Fourth. The corpora quadrigemina, in intimate connection with, and part and parcel of the mesocephale.

Fifth. The cerebellum.

Pursuing the method of exclusion, I hope to arrive at the determination of the part the disturbance of which gives rise to the phenomena of epilepsy, and of the epileptic paroxysm.

I shall first eliminate the medulla oblongata. An affection of this part, such as could provoke convulsions, would give rise

to *tetanic*, not to *clonic*, convulsions; and, in some cases, it would act so powerfully and so directly upon the muscles of respiration, that it would probably in many instances annihilate that process altogether. It is true, indeed, that a certain extent of laryngismus often complicates the epileptic convulsion; but primary disturbance of the medulla oblongata would render laryngismus a constant and necessary accompaniment of the epileptic fit, *which is not the case*.

Primary affection of the medulla oblongata does not explain the early and constant loss of consciousness which is pathognomonic of epilepsy, and which is often its *only* symptom.

Diseased states of the medulla oblongata show themselves in impaired deglutition, in vomiting, in altered rhythm of the respiratory movements, in an abnormal proneness to emotional excitement, but not in impaired intellectual action, nor in any affection of consciousness.

We cannot admit, then, that epilepsy is due to a primary disturbance of the medulla oblongata.

We come next to the Corpora Striata and Optic Thalami. These remarkable ganglia, although intimately connected with each other, are nevertheless very different in structure, and probably, also, very different in function. They are no doubt directly concerned in the development of voluntary motions and of sensation, and their intimate anatomical union is in harmony with the close connexion and interdependence of sensation and motion. They have nothing to do with intellectual operations, and therefore not with consciousness; their functions seem limited to the simple exercise of the will, or to that of the perception of some impression made on a sentient organ. If the corpus striatum or optic thalamus be diseased, you have paralysis of motion or of sensation, or both; but a sound state of intellectual power, and of the consciousness, is quite compatible with extensive disease of both of these organs, provided it does not extend beyond them. Mechanical irritation of these bodies does not produce convulsions, nor does any morbid state of them ever give rise to these disturbances of motion. It is clear, then, that these bodies can no more claim to be the seat of the primary disturbance in epilepsy, than the medulla oblongata.

Has the cerebellum any share in the production of epilepsy? I think we must answer this question in the negative likewise. The cerebellum has no influence on consciousness: the absence of the cerebellum is perfectly compatible with consciousness and a certain amount of intellectual power, and these powers remain intact even where there

is considerable disease of that organ. Moreover, mechanical irritations of the cerebellum do not give rise to convulsions.

There remain, then, only two parts of the brain in which we can localize the primary disturbance in the epileptic paroxysms—namely, the hemispheric lobes, and the mesocephale.

I will at once confess that my reflections upon this subject have led me to the conclusion that both these parts of the brain are greatly concerned in the production of the epileptic state, and in the development of the paroxysms. First and mainly, the hemispheric lobes; secondly and consecutively, the mesocephale.

You will bear in recollection that the complete epileptic paroxysm exhibits two features essentially distinct from each other: first, the loss of consciousness; secondly, the convulsions. The loss of consciousness and the other mental phenomena are dependent upon a disturbed state of the hemispheric lobes; the convulsions upon a disturbance of the mesocephale, consecutive to, and derived from, the disturbance of the hemispheric lobes.

There is no point in physiology more clearly made out than that the organ which is immediately active in the manifestation of intellectual operations is the convoluted surface of the brain, and the fibrous mass connected with it—the hemispheric lobes. Every fact in comparative anatomy points to this conclusion. Experiment confirms it likewise. When the hemispheric lobes are removed from a pigeon, as in Flourens' celebrated experiment, which I have more than once repeated, the animal became a mere living machine, capable only of manifesting the physical phenomena of life, exhibiting no indication of mental operation nor of consciousness: it fell into the deep unconsciousness of epilepsy.

In all instances where the nutrition of the cerebral hemispheres is disturbed, the intellect suffers: you have delirium or a maniacal state; or if the disease be of a kind tending to check nutrient change, or to destroy it, you have more or less sopor.

So, also, inflammatory states of the arachnoid and pia mater covering the convolutions of the brain, disturb the intellectual operations, because they are so intimately connected with the hemispheric lobes, being as it were the carriers of nutrition to them, that the nutrition of these membranes cannot be disturbed without perverting that of the convolutions themselves.

Although it is perfectly true, that the brains of persons dead of epilepsy in its earlier periods will exhibit, as Foville remarks, "nothing, absolutely nothing, which differs from the normal state," unless they have

died in the attack, when the cerebral congestion which exists is, in the words of the same distinguished physician, "a feature, not of epilepsy, but of the state of asphyxia induced by it, and in which the patients have died;" still, in the more advanced stages of the disease, when the patients have experienced many fits, morbid appearances are met with, and these affect the hemispheres of the brain chiefly. You have among the most common, opacities and thickening of the membranes, shrinking of the convolutions, enlargement of the sulci between them, increased subarachnoid fluid, alterations of colour and consistence of the grey and white matter of the hemispheric lobes. These alterations must be looked upon as the accumulated effects produced by the various paroxysms. Each fit does some amount of damage to the brain: in the interval the brain recovers itself to a great degree, when a new fit comes on, and new mischief is done; and so the repetition of the paroxysms leaves the brain altered as I have described it.

But, observe, the alterations are not of the cerebellum, nor of the medulla oblongata, nor of the corpora striata or optic thalami,—but of the hemispheric lobes.

Bouchet and Cazaurielh, who have conducted an extensive series of researches into the clinical history of epilepsy, found these alterations of the hemispheric lobes so frequent in the chronic cases, that they attribute the disease to a chronic inflammation of the cerebral lobes, which determines epilepsy if it affect the white substance, insanity if it affect the grey.

I refer to these researches, not because I believe that the conclusion founded on them by their authors is correct, but because they strikingly indicate that the seat of the organic disturbance (which these authors admit to be *caused by* the fits) is in the hemispheric lobes.

It cannot, I think, under the weight of argument which may be adduced in reference to the office of the hemispheric lobes, be doubted that an affection of these parts is capable of inducing all the phenomena of epilepsy, as far as regards consciousness and sensibility.

But an important question remains: can a disturbed state of the hemispheric lobes induce or excite convulsive movements? The proper answer to this question appears to me to be this: under ordinary stimulation of the substance of the hemispheres, the fibres are incapable of exciting motion. It is not the office of these fibres to propagate the nervous force to muscles, but to other nervous centres. Their function is to establish communications between the great sheet of vesicular or grey matter which covers the convolutions of the brain, and the cor-

pora striata, optic thalami, and mesocephale, so that changes in any of these centres may be propagated from any one to any other, or to all the rest. Hence the sections and irritations by mechanical and galvanic means to which these fibres have been subjected in the hands of various experimenters, produce no disturbance of motion, so long as the irritation is strictly confined to them.

So far, then, anatomy and experiment denote to us that of themselves these fibres of the hemispheric lobes cannot excite motion, but that they may do so through their influence upon other ganglia of the brain; and such phenomena, as I referred to at the commencement of the lecture, as a convulsive affection of the whole of one side of the body, evidently brought on by the deposition of tubercular matter on the surface of the cerebral hemisphere on the opposite side, and its consequent irritation, show that convulsive movements may be excited by a superficial lesion of the hemispheric lobes.

Hence we must not deny to these lobes a certain power of exciting motion, either directly or indirectly, through their influence upon other ganglia of the brain. But it is important to remark that the influence of the hemispheres is most manifest for this purpose when the lesion is superficial; that is, when it affects the grey matter. A deposition of tubercle, such as took place in the case I described at the commencement of the lecture, would produce little or no disturbance if it took place in the white substance: it would interrupt the functions of some of the fibres; but when deposited in the vesicular matter, among the particles of the generating plate of the nervous battery, the development of the nervous force becomes seriously impaired.

From all these facts, then, I infer that a disturbed state of the hemispheric lobes may undoubtedly give rise to so much of the phenomena of the epileptic paroxysm as refers to the affection of consciousness and sensibility, and that it may, *in some degree* at least, contribute to the development of the convulsions.

We must not forget that, in forming a theory of the pathology of epilepsy, we have to explain not a continuous state of disturbed sensation and motion, but a malady, the grand feature of which is the *periodical* recurrence of the paroxysms, the patient being wholly or almost restored to his normal state in the intervals between the attacks.

Now, it is not a little remarkable that there is no organ in the body which exhibits the same kind of periodical activity and quiescence in the performance of its functions, as the hemispheric lobes. This periodicity is manifest in the phenomena of sleep: throughout life the tendency exists,

that for a certain period each day the state of sleep comes on—when intellectual acts cease, the will is not exerted, the perceptive powers remain quiescent, the channels of sensation are closed. This tendency is greatest in the early periods of life than at any other time—greatest of all in infancy, when sleep engrosses the largest portion of time: very marked in childhood, less imperious in adult life, but assuming a somewhat increased influence in second childhood—OLD AGE.

It is unnecessary for me to take up time in adducing arguments in support of the assertion, that the phenomenon of sleep belongs to the brain, and especially to the hemispheric lobes. I must content myself with the statement, that the periodical recurrence of sleep is a part of the train of phenomena which belong to the normal nutrition of the brain. The diurnal variations of our planetary system are not more surely influenced by the laws which regulate them, than is the brain by that law of its nutrition which enforces that the activity of that part of it which is immediately associated with the operations of the mind should be from time to time interrupted by sleep.

In hibernating animals, we have a remarkable example of the extraordinary influence of this law of the nutrition of the brain, and of the curious mastery which it exercises over all the other processes of life. When the winter sleep sets in, the breathing becomes slow, the circulation is languid, the body feeds upon the store of fat which had been laid up during a period of exertion, the chemical changes of the body are reduced in activity and complexity. It lasts a certain time, and the animal becomes gradually roused, and resumes its wonted place among the active denizens of the world.

We have, indeed, in the animal economy, phenomena in some degree analogous to this alternate activity and quiescence of the brain. The heart beats with a certain rhythm, and pauses after it has accomplished its systole and diastole.

Respiration exhibits a similar rhythm in its movements.

So, also, perhaps, the peristaltic action of the bowels.

Something like a physical explanation of these phenomena may be offered, but we know no physical cause for sleep; and, in the existing state of knowledge, we must rest content with regarding it as an ultimate fact, that it is to the peculiar economy of the brain, and to the laws which specially regulate its nutrition, that we owe the unspeakable advantage and enjoyment of

“Tired Nature’s sweet restorer, balmy sleep.”

When these laws are impaired, there is either too much sleep, or it is almost banished from the eyes, and nature at length sinks exhausted.

Dr. M. Hall, indeed, thinks that he can derive some physical explanation of the phenomena of sleep from the antagonism which he assumes to exist between what he calls the spinal system and the cerebral system. “As sleep approaches, (he says) the levator palpebræ, a muscle of voluntary motion, ceases to act; whilst the orbicularis, a muscle of true spinal action, contracts and closes the eyelids.”

“Imagine,” he adds, “an event similar in its nature to take place in the muscles of the neck: volition ceasing, spinal action being energetic, certain muscles contract and compress the veins, and a degree of fulness of the neck and face, with turgidity of the eyes, is the obvious result; nay, there is even a degree of laryngismus heard in the respiration. All this is most observed in the first deep sleep.”

Now, to pass over certain very decided sources of fallacy in this view, such as the assumed energetic condition of spinal action, the contraction of the orbicularis and other muscles, the distinction between a muscle of true spinal action and of cerebral action, it is plain that it involves a *petitio principii*, and this is expressed in the first three words of the passage I have quoted—“as sleep approaches;” and in two words of the succeeding paragraph—“volition ceasing.” The whole question is involved in these two phenomena—the approach of sleep, the abeyance of volition: if we can determine what influences the approach of sleep, to what its first beginnings are due, what determines the abeyance of volition, we need inquire no further. According to Dr. Hall’s views, the increased energy of the spinal system, to which is due the contraction of the orbicularis and of certain muscles of the neck, is the immediate consequence of the first approaches of sleep, of the beginning of the state of quiescence of the cerebral system. In short, the state of sleep has already begun ere the exciting cause of this contraction of the muscles of the neck is brought into operation.

But to return to the question which we have in hand.

I say, then, that the periodical character of the phenomena of the normal nutrition of the hemispheric lobes of the brain denotes strikingly that these organs have much to do with the primary disturbances which cause those periodical paroxysms which constitute epilepsy.

But there is another part of the brain to which I think we must assign some share in the production of epilepsy.

This is that part of which the corpora quadrigemina form a prominent portion—the upper and posterior part of the mesocephale, the nodus encephali, the connecting medium between the several parts of the encephalon, which is not only anatomically related to all the constitutional parts of that organ, but possesses inherent powers of its own of great and general influence upon the movements of the body.

I have already referred to this part in my last lecture, as being the centre of emotion: as such, it is much influenced by mental states; and we know that epilepsy is often brought on by the shock produced by seeing another fall into a fit.

Flourens states, that while superficial sections of the corpora quadrigemina produce no other effect than the impairment or loss of vision, deep sections produce general convulsions.

I have been desirous of ascertaining whether the parts within the cranium, if excited by the stimulus of galvanism, would give rise to any phenomena similar or analogous to those produced by galvanic stimulation of the spinal cord. Accordingly, I determined to subject them severally to the action of the magneto-electric rotation machine,—a most convenient instrument for physiological experiments.

The experiments were performed on rabbits. I took the spinal cord first: here we had the well-known tetanic effects to which I have already frequently referred.

Next I tried the medulla oblongata: the effects of the stimulation of this organ were much the same as those produced by irritating the cord.

I then tried the corpora quadrigemina and the mesocephale. Having passed fine bradawls into the cranium in such a direction as I had previously satisfied myself would lead to this organ, I subjected it to the influence of the machine: general convulsions were produced, of a character essentially different from those which resulted from stimulating the spinal cord or the medulla oblongata. They were combined movements of alternate contraction and relaxation, flexion and extension, affecting the muscles of all the limbs, of the trunk, and of the eyes, which rolled about just as in epilepsy.

On inserting the awls into the hemispheric lobes, still different effects were produced by the application of the machine. I could observe nothing like true convulsions; but slight convulsive twitchings of the muscles

of the face took place, which were no more than what would be caused by the stimulus of galvanism acting upon the nerves of the face.

These experiments, which I repeated several times, and each time with like results, seem to denote that convulsions are modified according to the part of the cerebro-spinal axis which is primarily excited: if it be the spinal cord, they are tetanic; if the medulla oblongata, they are tetanic likewise, other parts being involved; if the corpora quadrigemina and the mesocephale, they are epileptic; if the cerebral hemispheres, you scarcely have any convulsions, but slight twitchings of the muscles.

Weber, in his excellent essay on Muscular Motion, published in Wagner's "Handwörterbuch" of Physiology, refers briefly to similar experiments performed by himself on the brain of a frog, and leading to the same results; and he draws this conclusion, that "the tonic convulsions, as trismus and tetanus, are the effect of disturbance of the functions of the spinal cord; whilst the clonic convulsions are due to derangements of the functions of certain parts of the brain."

Thus, then, I come to this conclusion respecting the parts of the nervous system which are directly concerned in the production of the epileptic paroxysm.

The part of the encephalon primarily disturbed, is the hemispheric lobes: if the disturbance do not go beyond a certain point, the phenomena are limited to loss of consciousness and impaired intellectual action, with more or less of sopor. But if the disturbance be considerable, then the tubercula quadrigemina and mesocephale become involved, and *epileptic convulsions* are produced. If the disturbance of this centre be very great, the medulla oblongata and the medulla spinalis become much excited, and the convulsions are complicated with a good deal of the tetanic character.

We know that there is great variety in the intensity of the epileptic paroxysm, *i. e.* not only in the intensity and duration of the coma, but also in the violence of the muscular paroxysm: all this depends on the nature and force of the primary disturbance in the cerebral hemisphere, but in all instances the hemispheric lobes are first disturbed, next follow the corpora quadrigemina, and upon the intensity of their disturbance depends the extent to which the medulla oblongata and the spinal cord are engaged.

### LECTURE III.—*continued.*

A QUESTION not less important to the determination of a correct pathology of epilepsy than that which we have now discussed, must next occupy our attention. This is, as to the intrinsic nature of the disturbance of the brain in this malady, and the exact organic condition of it which is capable of exciting the epileptic phenomena.

Is it a state of inflammation? or one of congestion? or is it a condition the opposite of congestion—one of deficiency of blood? or, in fine, is it a perverted nutrition, due more to a depraved *quality* of blood, rather than to alterations in its *quantity*, but by no means independent of the latter?

It is scarcely worth while to occupy time by stating the arguments against the idea of the inflammatory nature of epilepsy. We have now sufficient knowledge of the diseased states of the brain to enable us to recognise that affection when it occurs. Moreover, inflammation does not occur without leaving its traces behind,—and it is impossible that epilepsy could be an inflammatory disease, and leave behind it so little marks of such a disturbance of nutrition, as would leave it in the power of so able and so experienced an observer as Esquirol, to say—“we freely confess that pathological anatomy has hitherto thrown but little light upon the immediate seat of epilepsy.” The records of cases of undoubted inflammation of the brain show that epilepsy is not one of its symptoms, whatever part of the brain may have been the seat of the inflammatory process.

It may next be asked, is epilepsy caused by a state of congestion of the brain? This is a favourite notion with many, nor can one wonder that it should be so, when we consider how exclusively of late years the attention of pathologists has been directed to the state of the solids, and how often they have failed to find any indications of morbid change, save such as are due to a greater or less amount of blood in the blood-vessels. It has been the prevailing tendency to look for congestion of blood, and to rest content with it as the cause of the phenomena during life, the observer all the while overlooking the important inquiry, as to the period of the occurrence of the congestion; whether it might not have occurred in the mortal agony,—whether, indeed, it could fairly be regarded as having taken place during life,—or whether it might not be a post-mortem appearance.

But what does morbid anatomy teach us on this point? Why, that congestion is far from being a constant appearance in the brains of epileptics,—that its presence is mainly influenced by the mode of dying of the patient,—that in a large number of cases there is rather a deficiency than a superabundance of blood in the brain,—and that when congestion of the brain does occur in epilepsy, it may be looked upon rather as the consequence of the paroxysm than as its cause, the paroxysm being such as, by the mechanical interference which it creates with respiration and with circulation, greatly retards the return of the blood from the brain—as from all other parts—to the right side of the heart.

Furthermore, it may be now confidently affirmed, as pointed out by experience, that a state the opposite to that of congestion—namely, a state of *anæmia*—is favourable to the occurrence of epilepsy; and the convulsions of children and those of lying-in women are frequently brought on from anæmia due to imperfect nutrition in the former, and to excessive floodings in the latter.

The following case—and my experience has supplied me with several similar—is a striking instance of the extent of disturbance likely to arise from great loss of blood:—

A married woman, aged 30, was admitted into King's College Hospital, in January 1849. Two years ago she had a miscarriage after a pregnancy of three months. In August 1847, she was delivered by instruments of a dead child. In June 1848, she again became pregnant, and, after having gone two months, she miscarried, without excessive hæmorrhage. From this time her health began to fail—she became low and desponding—and made no blood; looked pale and thin.

On the 28th of last December, having been some time in a state of great weakness, she suffered from giddiness of head, and became slightly delirious. For these symptoms she was bled from the arm, and had leeches applied to her head, and a blister to the back of her neck. The next day, the symptoms remaining unaltered, six leeches more were applied to her head. While these were yet drawing, she gave a slight scream, and fell forwards in a fit of convulsions, during which the orifice in the vein burst open, and a good deal of blood was lost.

On the 30th of December, while six

leeches more were being applied to her head, she had a second fit, exactly resembling the first, and during this the orifice in the vein in the arm again opened, and more blood was lost.

On the 3rd of January she had a third fit, which lasted about one hour, and on the following day she had two fits. She now became maniacal, but controllable, and in this state, exhibiting all the signs of extreme anæmia, she came into the hospital.

Here, then, is a remarkable instance in which a state of extreme anæmia—the practice of general and of local depletion—offered no protection against the epileptic state: might we not say that an opposite mode of treatment, one adapted to improve the blood both in quantity and quality, would, in all likelihood, have saved the poor woman from the fits of epilepsy, and from a prolonged state of chronic mania under which she afterwards suffered?

Dr. Marshall Hall attributes much of the phenomena of epilepsy to vascular congestion. He has put forward the following theory of epilepsy. He says:—“1. Some source of irritation, acting in a reflex or direct manner, excites the spinal system. 2. Contraction of certain muscles of the neck, compression of the veins, and congestion of the cerebrum, with cerebral symptoms—cerebralepilepsy—are the consequences. 3. Then follow laryngismus, with every formidable convulsive symptom, spinal epilepsy, and congestion of the encephalon in a tenfold degree, with all its dire effects on the intellect and on the limbs.”

Admitting, as I do, the great ingenuity of this theory, I cannot accept it as a correct explanation of the phenomena of epilepsy, or as adequate to explain the various and complicated symptoms of that malady.

Without discussing any further the question whether congestion of the cerebral bloodvessels can excite the epileptic paroxysm (a question, I think, answered in the negative by all reason and experience), there are two points which need to be proved in the most unequivocal manner before this theory can be admitted as adequate to explain the phenomena of epilepsy.

The first is, that contraction of the platysma and of certain muscles of the neck can cause congestion of the veins of the neck, and consequently of the head. Upon this point I confess that I can come to no other conclusion than that the anatomical disposition of the platysma unfits it to act as a compressor of the veins of the neck. If by its contraction it exercises any influence upon the venous circulation, especially the deep-seated veins, it would rather tend to protect them from the pressure of the super-

incumbent tissues. When the platysma myoides is strongly contracted, the hollow of the neck is obliterated, the integuments are raised up, and they are rendered tense between the clavicle and the jaw. To effect such contractions of this muscle, which is not so completely under voluntary control as some others, requires considerable effort, and the simultaneous contraction of other muscles. This effort, by impeding respiration in some degree, tends more or less to congest the veins of the head and neck: but I have seen strong contraction of the platysma on both sides kept up for a considerable time without inducing any amount of congestion which would attract notice unless the attention was particularly called to it. In slight or partial contractions no such congestion occurs, and it is still less likely to take place in any contraction which might be caused by reflex action, in which there is no effort whatever on the part of the individual.

A second point, which needs proof, is the connection between *laryngismus* and convulsions. This connection, according to Dr. Hall's views, is so intimate, that the former is a necessary precursor of the latter. In other words, it may be said that convulsion of the laryngeal muscles must precede that of the other muscles. Surely such a view as this is untenable. There cannot be any necessary connection between the convulsion of the laryngeal muscles and that of the other muscles supplied by cerebro-spinal nerves, so that convulsions of the one should necessarily precede that of the other. All muscles are equally exposed to the cause which excites the paroxysm; if that cause excite only one side of the brain, the muscles on the opposite side of the body are thrown into convulsion. And although we sometimes find that one set of muscles will take precedence of another in convulsions, there is no constant rule to determine what muscle shall be first convulsed, and especially that precedence does not belong to the laryngeal muscles.

I have ascertained, by direct experiment, that general epileptic convulsions may take place when laryngismus, which is the chief cause of the impediment to the venous circulation, and of the subsequent cerebral congestion, cannot occur.

I looked for some poison which was capable of exciting epileptic convulsions. Although the *Conanthe crocata* is stated by Toxicologists to have this power to a remarkable degree, I was unable to produce the effect in dogs with either large doses of the expressed juice of the root or with large quantities of a strong decoction of the roots. I therefore had recourse to Hydrocyanic acid,

which develops the epileptic state in a very marked manner.

The first experiment was upon a large dog. A free opening was made in his trachea, so that inspiration and expiration were performed with the utmost freedom. The dog was then poisoned by Prussic acid. In a short time epileptic convulsions of the usual character, complicated with a good deal of spinal convulsion, ensued.

In the next experiment I proposed to divide the recurrent nerve on each side, and thereby to paralyse the constrictors of the glottis. Although the division of one of the nerves was not affected, the other was effectually cut across, and a large piece cut out of it. The voice of the animal was destroyed, and, as the muscles on one side were paralysed, it was obvious he was quite as unable to close the glottis completely as if the muscles were paralysed on both sides. Hydrocyanic acid was then administered, as in the first experiment, and very complete convulsions were very shortly developed, the eyes being very much affected, and all the muscles of the trunk and extremities.

The third experiment consisted in tracheotomizing a dog, and introducing a large tube into the trachea, which insured a greater freedom of inspiration and expiration than when there was only a simple opening. The administration of Hydrocyanic acid was followed by the same distinctly epileptic general convulsions as in the two former instances.

Undoubtedly in all these experiments, especially in those in which an opening was made in the trachea, there was less impediment to breathing, and less venous congestion; but, notwithstanding this, the epileptic state was fully developed under the influence of the poison. A free opening in the trachea, and the prevention of the consequences of laryngismus, did not prevent convulsions from taking place.

Thirdly. We may ask, is epilepsy due to an anæmic state of the brain,—to an imperfect supply of blood to that organ?

Although I cannot admit that epilepsy is commonly due to an anæmic state of the brain, still I must say that there are more facts in favour of this view than of the congestive theory, and that an imperfect supply of blood to the brain is very often associated with the epileptic condition.

I have already referred to cases in which epilepsy had supervened upon an anæmic state. These might be multiplied considerably, did time permit. What is particularly deserving of attention is the class of cases where convulsions ensue upon excessive uterine hæmorrhage,—cases long recognised by the most experienced obstetrical physicians.

A cachectic state, in which the red particles

of the blood are only very imperfectly developed as to quantity, is very apt to accompany epilepsy. We see this frequently in hospital practice, in a very hard-working class of men—compositors in printing-offices, especially in those of newspapers. These men frequently work all night; and, to support them in their toil, they addict themselves to the frequent use of spirits or other fermented liquors. General nutrition fails: the red particles are imperfectly regenerated, and the patient becomes epileptic.

Patients labouring under chronic disease of the kidney are much more apt to become epileptic, if they are pale, and if their blood is deficient in its red particles.

House-painters, or others exposed to the contamination of lead, are apt after some time to fall into a fearfully cachectic state, of which a principal feature is the deficiency in the red particles of the blood. I have seen several persons, under these circumstances, become epileptic shortly before death, and, in fact, die in consequence of the violence of the epileptic paroxysms.

All these are striking instances to show how blood deficient in quantity,—deficient in one of its most important staminal principles (the others, perhaps, not being quite normal),—and, perhaps, contaminated by the presence of some foreign noxious principle,—is favourable to the production of the epileptic state.

Experimental physiology supplies us with very striking facts to show that an insufficient supply of blood to the brain is very apt to occasion epileptic convulsions.

Every one who has witnessed the slaughter of sheep, which is effected by dividing the great arteries in the neck, must have observed the strong convulsions which so frequently precede death in animals killed in this way. All animals killed by loss of blood exhibit the same phenomena precisely, and die with convulsions of a more or less violent kind.

Dr. Seeds many years ago performed a series of experiments to observe the phenomena accompanying the deaths of animals by loss of blood. In all the cases convulsions preceded death.

In Sir Astley Cooper's experiments, by the application of ligatures to the carotid and vertebral arteries, the functions of the encephalon seemed more influenced by occlusion of the latter than of the former arteries. A considerable portion of the blood of the carotids is distributed to the external parts, to the membranes, to the parietes of the cranium; and those branches which supply the brain anastomose very freely with the vertebral arteries.

When the vertebral arteries were tied or otherwise obstructed, the carotids having

been previously tied, very serious symptoms ensued: a state of insensibility was induced, which frequently was accompanied with convulsions, affecting the muscles of the eye-balls, as well as those of the extremities. In one experiment the carotids of a rabbit were tied, without any material effect. In five minutes the vertebral arteries were compressed by the thumbs, the trachea being completely 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.

On a second compression of the vertebrae, similar phenomena ensued.

Three times more the compression of the vertebrae was repeated, with like result. On the fifth occasion, says Sir Astley, the result was the same—namely, suspended respiration, convulsions, loss of motion and consciousness.

These, surely, are symptoms very closely resembling those of epilepsy.

It is much to be regretted that Sir Astley's experiments on tying the jugular veins were not more numerous. Only two are recorded. In one, after ligature of both jugular veins, the respiration was reduced in frequency to about half the usual number for a few hours, and the animal recovered without any other untoward symptom. In a second, the same reduction in the frequency of the breathing ensued upon the application of the ligatures: for five days the animal seemed dull, and on the seventh day it became convulsed, and frequently rolled over, and lost its voluntary power and its sensation in a great degree. But these symptoms, it was proved after death, were due to the extravasation of a clot of blood in the left ventricle of the brain.

The application of ligatures to the jugular veins, so as to impede the return of venous blood from the brain, is evidently, so far as these experiments go, productive of less serious consequences than the cutting off the supply of blood to the brain.

But we do not need experiments to prove what an enormous extent of congestion the brain can bear without inducing anything like an epileptic state. Look at cases of old asthma, with dilated right side of the heart, and large jugular veins, into which the blood freely regurgitates at every systole of the auricle: witness, also, the excessive congestion which accompanies severe suffocative catarrh, chronic bronchitis, and some of the complications of phthisis which are attended with great dyspnoea. If congestion of the brain were the common or even frequent cause of epilepsy, that malady ought frequently to accompany the diseases above named: yet patients labouring under these

complaints, although they often become comatose shortly before death, seldom exhibit anything like convulsions, nor anything of a genuine epileptic state.

I come, therefore, to this conclusion,—that while it is highly improbable that a state of congestion *per se* gives rise to the epileptic paroxysm, it is extremely probable that the opposite state, one of anæmia or of imperfect supply of blood, does frequently cause the complete epileptic fit, and that such a state is much more to be feared and guarded against by practitioners than the state of congestion, not only because its consequences are more serious and more persistent, but also because they are much less amenable to the remedies within their reach.

At the same time, this state of anæmia is not in itself the cause of epilepsy. We have too many instances of epileptics in whom no indication of an anæmic state exists, to allow the admission that the influence of anæmia operates in any other way than as one of the most potent disturbers of the nutrition of the brain; and we are therefore led to class it with other causes of disturbance which interfere with and derange the normal nutritive processes of the brain, giving rise to irregular actions of certain of its parts.

Having freely discussed the more obvious disturbances of the circulation in the brain which have been and are set down as productive of the epileptic state, it remains for me to develop a theory of epilepsy such as seems most in accordance with our knowledge of the physiology and of the pathology of the brain.

I would lay it down that epilepsy denotes a state of abnormal nutrition of the brain.

This abnormal nutrition shows itself in the unnatural development of the nervous force at particular times, in such a manner as to disturb the functions of the whole cerebro-spinal centre, but of the brain in particular.

These periodical evolutions of the nervous force which give rise to the complete epileptic paroxysm may be compared to the electrical phenomenon described by Faraday under the name of *disruptive discharge*.

We know that this phenomenon requires for its production, first, a highly-charged or polarized body, which, when it reaches a certain point of tension, may be made instantaneously, and with violence, to discharge its electricity to a conductor.

Thus the abnormal nutrient actions of the brain, in epilepsy, tend to produce a polar state of the particles of the Hemispheres, of the Tubercula Quadrigemina, and of the mesocephale, which, when it reaches a certain tension, discharges itself, and induces with great violence a rapid polarization of the

neighbouring particles, involving in the general disturbance the medulla oblongata, the cerebellum, and the spinal cord.

In some instances the tension may be limited to the hemispheres of the brain,—then consciousness and intellectual action only are disturbed; either a comatose state is induced, which lasts for a certain time, or mania or delirium are produced. In such cases the state of tension may pass off slowly and gradually without any distinct discharge, and so the nervous matter may assume a state of equilibrium, without creating any serious disturbance of the other parts of the cerebro-spinal centre. In other cases the hemispheres and mesocephale pass quickly to the highest degree of tension, and a rapid discharge takes place, exciting the other parts of the brain and the spinal cord, with all the violence of the discharge from a highly charged Leyden jar, or the shock from the electrical organs of the torpedo or gymnotus.

The gradual and silent manner in which this state of tension is induced in the brain, and the absence of all appearances of mischief recognizable by our means of investigation in recent cases, the rapidity with which it succeeds fit in the acute cases, leaving likewise no trace of organic mischief, denote that the peculiar state of the brain in epilepsy is brought on not by any change in the quantity of the blood, but by the accumulation of some material in the blood, which, acting on the brain as a poison, excites this polar state, and this disruptive discharge, and so escapes from the system, leaving the brain free from disturbance until a fresh accumulation excites a new paroxysm. The same comparison may be used here as I have already employed in speaking of other convulsive diseases. The nervous battery is excited by an abnormal fluid,—a fluid which causes it to develop the nervous force in irregular quantity,—a fluid which, when fully charged with its particular morbid element, excites a high degree of tension of the cerebral nervous matter, which can recover itself only by a discharge, which produces more or less serious disturbance of the neighbouring parts of the brain.

Under this view the infinite variety in the character and violence of the epileptic fits would be accounted for by variety in the quantity of the morbid material which contaminates the blood,—the poison which disturbs the normal actions of the brain,—by the different intensity with which it affects one part more than the other,—the comatose symptoms, being the more developed as the cerebral hemispheres are affected,—the convulsive symptoms, when the chief disturbance is in the mesocephale,—and, perhaps, also by difference in the quality,—the virulence—of morbid element.

This humoral doctrine of epilepsy is supported by a host of striking facts. The influence of certain poisons—as Prussic Acid and *Ceanothe Crocata*—in the production of artificial epilepsy, the exact counterpart of the disease as it affects the human subject, is one of the most important of these. Saillant, a writer in the memoirs of the French Royal Society of Medicine (ann. 1782-83), concludes a paper detailing some experiments on the artificial production of epilepsy, with these remarks:—"We are content with concluding that it is easier to produce an artificial epileptic paroxysm through the blood than by irritation of the brain and nerves." Saillant's experiments consisted chiefly in injecting air into the veins.

A second class of facts, bearing most strongly on this view of the nature of epilepsy, is the frequent connexion between this disease and imperfect eliminatory action of the kidney, a connexion which I have no doubt ere many years we shall find to be very much more frequent than is even now suspected.

Nay, a temporary functional suppression or deficiency in the secretory action of the kidney, would allow the urea, and perhaps certain other elements of the urine, to accumulate in the blood to such an extent as to exert a poisonous influence on the brain, and to develop the epileptic paroxysm. The fact, first I believe brought to light by Dr. Lever, that the urine of women who have suffered from puerperal convulsions is sometimes albuminous, suggests that in some of the patients who suffer this form of epilepsy the disease may be referred to defective action of the kidney. This point, however, has yet to be worked out, and when physicians come more generally to look for the causes of morbid phenomena in something beyond mere excess or deficiency of blood, we shall probably obtain some satisfactory elucidation of it.

A third class of facts favourable to this view is derived from the diseases confessedly humoral, and the paroxysmal character which they are apt to assume. I would allude more particularly to the effects of the paludal poison, and to gout. Under the influence of the former, we have periodical paroxysms almost convulsive in their nature. A severe fit of ague in its cold stage is not far from an epileptic convulsion. The marsh poison, whatever be its dose, shows its effects in regular periodical paroxysms; the poison accumulating in conjunction with certain nutrient changes in the blood, reaches the quantity necessary to produce a paroxysm only at certain periods. The introduction of quinine checks this regenerating or cumulative power of the poison, and ultimately kills it. In gout we have an analogous

tendency to the accumulation of the poison so as to occasion paroxysms. The regular development of the gouty paroxysm at the spring and fall of the year in some individuals, and its frequent occurrence at short intervals for a certain time, as once a month or once in two months, are phenomena analogous to the periodical development of the epileptic paroxysm.

Lastly, we have the very important fact, that certain animal poisons, as that of small-pox, measles, scarlet fever, or even the poison of typhus, may, on their introduction into the system, produce epilepsy.

Of the *nature* of the morbid matter, which, by its accumulation in the blood, may produce the epileptic paroxysms, no conjecture can be formed in the present state of our knowledge. It may, perhaps, vary in different cases: in the renal cases, for instance, it may consist merely of certain retained elements of the urine. Of these, the experiments of Dumas and Prevost point chiefly to urea as the most highly poisonous; but I apprehend this point needs further investigation. In the ordinary cases we may assume that the morbid matter is some subtle agent, possessing properties allied to those of prussic acid, whose chemical nature may yet, in the progress of chemical analysis, be ascertained.

What are the sources of this morbid matter? I apprehend the most common source is the brain itself. The frequency with which epilepsy follows undue exhaustion of the nervous power, from anxiety, grief, long-continued thought, habits of intemperance—mental, as anger, rage, despondency,—physical, as the indulgence in ardent spirits or other fermented liquors. The influence, too, of mental shock, in the production of the epileptic state, must be regarded as denoting that direct disturbance of the nutrient actions of the brain under mental influence, may operate as a primary exciting cause of this malady.

Now all these causes tend directly to impair the nutrition of the cerebral matter. We have no difficulty in understanding that overwork or underwork (if I may use the word) of a muscle may impair its nutrition, and so weaken its powers. But we do not attribute this to an over supply of blood, or to an insufficient supply. The excessive or the imperfect exercise of the muscular force affects the tissue itself, impairs the nutrient changes which take place in it, disturbs the attraction between the blood and the tissue, and perhaps alters the nature of the material which is formed out of the effete particles, in what Dr. Prout calls the destructive assimilation of the tissues. Just in this way the nervous matter suffers in the first instance from excessive or perverted use, the nutrient changes are impaired, and the secondary

processes of assimilation generate a material which becomes a source of contamination of the blood.

No doubt, however, there may be other sources of contamination,—the digestive organs, the sexual organs, the great glands,—which may not only generate morbid matters capable of contaminating the blood, but may, by the imperfect performance of their functions, weaken or otherwise damage the nutrient changes of the nervous matter, so as to make it more susceptible of the influence of any noxious agent, or to cause it to generate a morbid matter.

Where there is hereditary taint, the inherited tendency is to the generation of what I might call the epileptic morbid matter under the influence of any cause which may disturb general nutrition, or impair the local nutrition of the encephalon.

Such is the theory of epilepsy which appears to me most consistent with the known facts of the clinical history of the disease, and with the general principles of physiology and pathology.

That it is sufficient to explain the ordinary cases where attack succeeds attack with more or less of periodical regularity, is obvious; this periodicity being explained partly by the periodicity of the normal actions of the brain, and partly by the re-accumulation after certain intervals of the morbid or poisonous matter. Nor is it inconsistent with this view that at the expiration of each of these periods the patient should experience not one, but a series of epileptic attacks in rapid succession, the number and violence of the attacks being determined by the quantity of the poison and its virulence, and, in some degree, perhaps, by the state of excitability of the nervous system.

Many instances occur where men have had but one epileptic attack during a long life. Are these cases explicable by this theory? Such cases may rank with the puerperal cases, or with the convulsions of children. They are due to some rapid change in general nutrition when the nervous system is in an excitable state. They are often caused by a debauch, excessive indulgence in the pleasures of the table, or of some other kind, which at once excites the nervous system and disturbs general and local nutrition.

An interesting question which I have found it convenient to postpone until now, is as to the validity of that distinction which I admit is useful for practical purposes, into *centric* or *eccentric* epilepsy.

Under the theory which I have just enunciated, all epileptic cases are strictly centric—that is, result from a cause acting directly upon the centre. But may not the cause

originate at the periphery, and act upon the centre through incident nerves, affecting the brain through the spinal cord? To this question I think I must answer in the negative. Evidence is wanting to prove that the peripheral irritation of nerves is capable of exciting the epileptic paroxysm. You remember the remarkable conclusion of Sallant, which I quoted not long ago, "that it is easier to produce an artificial epileptic paroxysm through the blood, than by irritation of the brain and nerves." Then I may be asked, what becomes of the cases so generally admitted, in which epilepsy is caused by worms in the intestinal canal? in which, if you expel the worms, you cure the epilepsy. I am not prepared to deny the occasional association of worms and epilepsy, nor their connexion as cause and effect; but I would affirm that worms cause epilepsy not by direct irritation of the intestinal nerves, the spinal cord, and the brain, but by the disturbance of nutrition which they keep up. The mere presence of worms in the intestinal canal is an indication of deranged secretions there, and of a disturbance of its nutrition: worms do not flourish in a perfectly normal state of the intestinal canal. Therefore we may lay it down that there has been a disturbance of nutrition prior to the appearance of the worms, which is kept up and increased by the presence of the worms.

Another question connected with this subject appears to me deserving of consideration. Can permanent unchanging chronic disease of the encephalon or of the cranial wall, be regarded as capable of causing the epileptic fits? Take, for example, such a case as the following:—A man has been epileptic for many years of his life, and his malady has resisted various remedies. He dies, however, of some other disease. On inspection, the brain is found healthy, but about the centre of the falx there is a tumor as large as a small walnut. Has the tumor been the cause of the epileptic attacks in this case? I incline to say No to this question, because such tumors are of very slow growth, and we know that the brain adapts itself wonderfully to the extension of such morbid productions upon the space in which it lies,—because such tumors have existed without epilepsy,—because a cause in constant operation is not likely to produce an occasional effect. At the same time I think it must be admitted that a man with such a tumor is more liable to be epileptic under states of general or cerebral disturbance, than a man whose brain and its membranes are healthy, because the presence of such a tumor scarcely admits of the nutrition of the brain being perfectly normal.

Epileptiform attacks are apt to be produced by meningeal irritation, as I have already pointed out. Under frequency of

repetition, and the consequent increased cerebral disturbance, these attacks may become epileptic; and this is the more apt to occur if there be any constitutional malady,—if the local mischief originate in any constitutional taint. Thus scrofulous deposits in the membranes will readily give rise to fits, at first epileptiform, and finally epileptic. And cases of the following kind are not uncommon:—A man contracts syphilis; some time afterwards, with or without other secondary symptoms, he gets periostitis of the tibia, or of some other long bone; at length he has an epileptiform attack, or it may be an epileptic fit; and now it is discovered that some part of the pericranium is painful, and there is good reason to believe that the dura mater is affected: there is periostitis within the cranium. Many of these cases do well under the judicious combination of iodide of potassium and mercury, or under the use of the former remedy only.

So, also, injuries of the head, proving after the lapse of a longer or shorter time injurious to some part of the osseous wall of the cranium, seem to cause epilepsy, and are more especially likely to do so if any derangement of general nutrition occur,—gout or struma, or the syphilitic taint. Perhaps in some of the cases epilepsy would have shown itself, even though no injury had taken place. How many hundreds of instances are there where injuries of the head, even of an extensive kind, have occurred, without a sign of epilepsy!

The terms *centric* and *eccentric* are so far useful as directing attention to the *source* of the depraved nutrition of the brain; but epilepsy is always the result of such depraved nutrition, and it is highly improbable that it can ever be looked upon as produced by reflex action—that is, by a peripheral irritation propagated to a centre, and exciting great irritation there, involving spinal cord, and motor and sentient nerves.

I shall conclude with a few remarks upon the treatment of epilepsy.

If much obscurity hangs over the pathology of this malady, we cannot wonder that our attempts to treat it have received so little encouragement.

One point of peculiar difficulty as regards the treatment of epilepsy arises from the uncertainty of the effects of remedies. Many of the anti-epileptic remedies produce no sensible effect on any of the great functions of the body: they do not purge, nor sweat, nor excite the kidneys. Under the use of the remedy the patient's fits become less frequent, less violent, and his general state improves. But it often happens that without any remedy fits become less frequent, less violent, and general health im-

proves. Have we any means of distinguishing between what may be regarded as genuine effects of a remedy, and mere coincidences,—changes which our knowledge of the clinical history of the malady tells us may take place independently of the influence of any remedy?

In many instances I must confess I know no means of making this distinction. It is in cases where the fits are very frequent that we may most readily, and with greatest probability of truth, draw conclusions respecting the effects of remedies. If a patient have had for some time one or more fits daily in a confirmed manner; and if, shortly after commencing the use of a remedy, the fits diminish considerably in frequency and violence, or cease entirely,—this we may reasonably ascribe to the influence of the remedy. And this conclusion might very usefully be tested by omitting the remedy for a time, and resuming it: the test would be highly confirmatory of the conclusion, if, during the cessation of its use, unfavourable symptoms showed themselves, which, after its resumption, disappeared.

A good deal of reliance may be placed on the convictions of some patients themselves as to the good or bad effects of particular remedies. If the patient expresses a strong opinion respecting a particular remedy, the practitioner ought to be influenced by it, and more especially if he adheres to this opinion after the use of the remedy has been long continued, or if he expresses a strong desire to return to the remedy after it had been discontinued.

Hitherto the search has been for some remedy which would cure epilepsy as quinine cures ague, or even as iodide of potassium cures syphilitic periostitis;—something which, when introduced into the system, would kill or neutralise the abnormal matter which is ever being formed, or would stop its formation. This search, which has now been continued for so many years in vain, we must, nevertheless, not abandon. It may be that a remedy of this kind may yet be vouchsafed to us, nor can one conceive a more inestimable blessing for mankind. Among the vast stores of the vegetable kingdom, there may yet be discovered some subtle agent—perhaps some alkaloid—readily assimilable by the brain, which may exercise the beneficial influence so earnestly to be desired, and diminish or remove this dreadful scourge.

Hitherto, I say, our search for a specific against epilepsy has been in vain; for who will venture to say that, out of the long list of anti-epileptic remedies with which our materia medica supplies us, any one really deserves the name? Many of the mineral and vegetable tonics do good for a time: valerian has acquired a great name, and

many authors of repute relate cases apparently cured under its influence; in the present day we are able to administer *valerianates*, in which valerianic acid (probably the active principles of valerian) is in combination with zinc, or iron, or quinine.

It is not my intention to enter into any comparison of the value of the countless number of remedies which have been at various times suggested for the relief or cure of this malady. I shall content myself with remarking that it is very important for the practitioner to provide himself with a good list of remedies, that he may not appear to his patient to be wanting in resources; and that, by changing the remedy from time to time, he may keep up the hopes of his patient, and secure his confidence. As Dr. M. Hall remarks, with great justice, “the confidence of the patient is a security against many baneful emotions, and time is gained for Nature to cure the disease.” I would add this remark by way of precaution, that, in the employment of the various reputed anti-epileptic remedies, the practitioner should employ those chiefly which he knows will do no harm, and that he should abstain from, or use with great caution, such remedies as exercise a known effect on other functions, and which may perchance affect them injuriously.

Very lately, my friend Mr. Salter, of Poole, whose sound judgment and great experience give great weight to his opinions, has directed the attention of the profession to the virtues of the *Cotyledon Umbilicus* as an anti-epileptic. He has narrated one very striking case as having apparently yielded to the influence of the remedy. The best form for its administration is no doubt the expressed juice of the fresh plant, which may be given in ounce or larger doses two or three times a day; or in the form of inspissated juice, as now prepared by Mr. Hooper, of which Mr. Salter states that half a drachm is equivalent to an ounce of the fresh juice. I hope this remedy will be extensively tried: it is one of those which, if it do not cure, at least is not likely to exercise an injurious influence on the constitution of the patient.

I am not aware that any of the means proposed to check a fit, once it has begun, have been attended with success. With our present experience it seems to me clear that the best practice in the fit is to abstain from interference, every precaution having first been taken to protect the patient from injury. Bleeding in or after the fit is certainly fraught with great dangers, and the plan of pressing the carotids, proposed long ago by Dr. Parry, can clearly have but little influence as long as the vertebrae remain free, nor is it by any means certain that the stoppage of the circulation through them

may not in many cases tend rather to increase the evil.

In the general treatment of epilepsy, the practitioner must ever keep in view the three following objects: first, to remove sources of irritation, of disturbance of general nutrition, and consequently of contamination of the blood; secondly, by hygienic and moral means, to promote a healthy frame of body and mind; and, lastly, to find, if possible, some means of preventing the access of the fits, with a view to breaking the habit of their frequent periodical recurrence.

I need not extend this already too long lecture by enlarging upon the various means by which peripheral sources of irritation or of contamination may be removed. It will be the duty of the practitioner to investigate with great diligence all the functions—digestive, sexual, that of the circulation, the skin, the kidneys,—and direct his special attention to such as may indicate a deranged state.

To enforce a good system of hygienic and moral rules, it is absolutely necessary that the practitioner should demand and obtain a complete control over his patient. He should regulate with the utmost precision the ingesta, both as to quality and quantity—the times of rising and of retiring to rest—the hours of sleep—the time of meals—the amount and period of exercise—the amusements—the nature and amount of study; and the aim of his hygienic system should be to invigorate the nervous system, and to promote by every means a healthy and vigorous nutrition: while that of his moral management should be to tranquillize the mind, to encourage hope, and to promote cheerfulness. It is from such a system, faithfully carried out and diligently enforced, that the greatest good may be anticipated for the epileptic patient.

To find some means of checking or of postponing the fit, is an object of great importance. In general, the nervous system and the mental functions suffer most when the fits are most frequent. If, then, the fits can be considerably reduced in number,—if, as it were, they can be kept at bay,—surely the patient cannot fail to be a great gainer.

Shortly after the anæsthetic powers of sulphuric ether became known in this country, and after I had satisfied myself, by the experiments which I referred to in a former lecture, of its great power in depolarizing the nervous centres, or preventing them from assuming a highly polar state, I determined to ascertain what influence its inhalation in the same manner as for surgical operations might have in checking the access of the epileptic paroxysm, or in diminishing the excitability of the nervous system. Although I have since learnt that about the same time it was tried in France, I was not

aware of these trials at the time, nor have I since seen any satisfactory account of the results of them.

The first case which presented itself was one of epilepsy, in which the fits were preceded and followed by mania. The patient was a young girl, aged 17: the fits were of frequent occurrence,—twice a week, and she was subject to them for seven years. Within the last year the fits were accompanied by the maniacal state. This girl had been in a maniacal state for six days prior to her admission into the hospital: she was very violent, attempting to bite every one who came near her. She had even bitten herself.

In this case I administered ether on a sponge, holding it lightly over the mouth. The effect, to my great astonishment, and that of the bystanders, was instantaneous. Within a minute, noted by the watch, the patient fell off into a profound and placid sleep, which lasted eight minutes. She then awoke, and seemed disposed to be violent as before. It was necessary to administer the ether five times, after which prolonged sleep was produced. She slept well that night, and from this time became quite manageable and controllable: she continued for three or four weeks in the hospital, and although she had two or three fits there was no return of the maniacal state.

I have since had many opportunities of administering ether, and subsequently chloroform, in cases of epilepsy. The cases which I have selected have been those in which, as in the case just related, the paroxysms have been accompanied by mania, or in which the paroxysms have been of frequent occurrence—daily, several times in a day, once or oftener in a week. In these cases I have administered chloroform daily, often twice, sometimes three times a day, and always producing the full effect. I have not met with any instance of untoward result in some twenty-five cases; and the general conclusion at which I have arrived is this, that the frequent administration of the chloroform does no harm to the brain, and that it undoubtedly diminishes the frequency of the fits, and exercises a very marked control over the maniacal state.

I have given it in cases in which there could be no doubt of the existence of organic disease within the cranium, either meningeal or cerebral, and with decided benefit. One old man, who had all the symptoms of diseased cerebral arteries, with, perhaps, some amount of white softening, and severe epileptic fits, was distinctly the better for the inhalations, and became more intelligent, and his fits less frequent.

In puerperal convulsions, chloroform may be administered with advantage. I know of several cases which occurred in the prac-

tice of others, in which the fit yielded to the influence of the chloroform. My friend Dr. Simpson, of Edinburgh, informs me that, among upwards of two hundred women whom he had delivered under chloroform, he has had no case of convulsions. In the epileptic convulsions of children also it may be used. Shortly after I began to use ether in epilepsy, my friend Mr. Ceeley, of Aylesbury, informed me that he had tried it with decided success in a case of infantile convulsions; and Dr. Simpson tells me that Dr. Robertson, President of the College of Physicians in Edinburgh, has administered chloroform in infantile convulsions with great success.

The administration of chloroform in epilepsy requires further clinical research. Upon this subject I am at present engaged, and hope, at some future period, to lay my results in a detailed form before my professional brethren. Perhaps some other anæsthetic may be found better suited to epilepsy than chloroform. I am not without hope that some anti-epileptic agents—valerianic acid or valerianates—may be more efficaciously administered through the inhalation

of chloroform than by the mouth. Mr. Savory, of Bond Street, has sent me some specimens of valerianates held in solution by chloroform; but whether these substances will volatilise with the chloroform so as to admit of their introduction into the system in sufficient quantity, I have yet to learn.

I need scarcely remark, and yet I feel that I ought not to conclude the lecture without doing so, that chloroform must not be used without some careful precautions. One cardinal point is, that in its administration atmospheric air should be at the same time allowed to be freely inhaled: another point is, that the administrator should not be eager to obtain a rapid effect, although he should aim at obtaining a full effect; a third, that the recipient should always be placed, during the administration of the vapour, in the horizontal posture; and, lastly, the practitioner should abstain from administering it in feeble cases, where the heart is diseased or feeble, and the circulation languid or otherwise disturbed: and in all cases he should bear in mind that chloroform is a very depressing agent, and he should provide accordingly.





