

# Introductory Lecture

TO A COURSE ON THE

## PHYSIOLOGICAL PATHOLOGY OF THE BRAIN.\*

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### INTRODUCTORY REMARKS ON THE MODE OF PRODUCTION OF SYMPTOMS OF DISEASE OF THE BRAIN.

*Almost all our admitted notions as regards the physiology and physiological pathology of the brain are grounded on wrong data—How these erroneous data have been obtained—Symptoms of brain disease do not issue from the loss of function of the part affected, or by merely setting in action supposed special properties of that part—An intermediate element exists between symptomatic manifestations and the diseased part of the brain—Symptoms arise from a change in other parts of the nervous centres than the one where we find disease—Necessity of distinguishing between effects of an irritation and effects of a loss of function—The effects of irritation are exceedingly variable, while those of a loss of function are constantly the same—Excessive excitability of the brain—Immense variety of effects of irritation of the different parts of the brain—Alterations caused by a lesion of a part of the brain in the nutrition of other parts of that organ itself and of the medulla oblongata, the spinal cord, nerves, and other organs or tissues—Comparison of effects of irritation of peripheral parts of the nervous system and effects of irritation of the brain—Disease may exist almost anywhere in the brain without any symptomatic manifestation—A lesion located anywhere in the brain may produce any of the symptoms of cerebral disease—The admitted or recently proposed localisations of function in the brain are to be rejected—Views of the lecturer on localisation—Some of his views on the physiology of the brain—Mechanism of production of symptoms, according to his views.*

WHEN we witness a loss of function occurring in connexion with a lesion of a part of the brain, we are very naturally led to suppose that the lost function has its seat in the part we find altered. Naturally also, but in a less degree, we are disposed to admit that if some muscles contract involuntarily in a limb, in the face, round the eye, &c., when a part of the brain is irritated, it is that the part thus put in action contains either the centre or the conductors usually employed by the will to move those muscles.

All our knowledge, or, more exactly, our supposed knowledge, of the physiology and the physiological pathology of the nervous centres is unfortunately grounded upon conclusions drawn from facts belonging to the two classes just mentioned. Natural, as I have said, as that mode of reasoning is, I cannot but think that it has been most mischievous; and, as regards the brain at least, I will try to show that the conclusions it has led to are essentially wrong and must be given up. This is no small undertaking, as what I have to prove is nothing less than the necessity—first, of throwing aside as false almost all our notions concerning the cerebral physiology and physiological pathology; and, secondly, of building up almost entirely new doctrines with regard to the physiological and morbid actions of the various parts of the brain.

If the mode of reasoning I criticise is carried out to its fullest extent, its danger becomes at once evident. Everyone knows what effect resembling laughter is produced when the sole of the foot is tickled. Concluding as has

been done, when effects are observed after an irritation of the brain, we should have to admit that the central power of laughter resides in the sole of the foot. When a worm in the bowels causes insanity, we should have to accept as proved that the seat of intelligence is in the intestinal mucous membrane. If paralysis, convulsions, anæsthesia, amaurosis, deafness, &c., appear in consequence of some intestinal irritation, we should have to admit that the mucous membrane of the bowels is the seat of the centres for voluntary movements for the perception of tactile, visual, and auditory sensations, &c.

Everyone would say that such conclusions would be absolutely false. No doubt they are so; but there is no doubt also that we commit the same kind of mistake when we conclude that the left third frontal convolution is the seat of the faculty of expressing ideas by speech, because when it is diseased that faculty is often disordered or lost,—or that the upper parts of the two convolutions bordering on the scissure of Rolando on one side are the centres for the voluntary movements of the two limbs on the opposite side, because sometimes these limbs will be convulsed or paralysed when there is disease in those parts,—or that the internal capsule contains the nerve-fibres conveying the orders of the will to muscles in its front and lower part, and the nerve-fibres employed in the transmission of sensitive impressions in its back and upper part, because in many cases of paralysis disease has been found in its large front and lower part, and in several cases of anæsthesia a lesion existed in its small back and upper part. I could lengthen considerably this list of instances in which, from facts showing that sometimes a disease in some parts of the cerebral lobes, in the corpora striata, in the optic thalami, in the tubercula quadrigemina, in the cerebellum, &c., has coexisted with certain symptoms, it was concluded that each of these encephalic organs had a well-defined special function.

Long ago it should have been found out that between the primitive cause of symptomatic manifestations (that cause being located where there is an evident organic disease of the brain) and these manifestations themselves there exists a middle term, an intermediate element, which is the efficient or immediate, although secondary, cause of the symptomatic phenomena or effects. In other words, it should have been found that the symptoms of a lesion of the brain are not immediate or direct effects of that lesion, and proceed from an unseen, but perfectly discoverable, alteration in parts of the cerebro-spinal centres, more or less distant from the seat of the known lesion. Long ago it should have been ascertained that the cerebral lobes, the cerebellum, and the base of the brain, are very much more excitable than the nerve-fibres of the various mucous, serous, or fibrous membranes of the body, and than those of the trunk of the cranial or spinal nerves, and that the mechanism of production of symptoms of brain disease is the same when there is an organic disease of the brain as when those symptoms appear as reflex effects of peripheral nerve irritation. It should have been discovered that it is an irritation propagated to other parts of the nervous centres from the diseased spot in the brain, which causes paralysis, anæsthesia, aphasia, amaurosis, loss of consciousness, &c., and not a loss of function of that diseased part; and that it is also an irritation propagated to other parts of the nervous centres from the diseased cerebral tissue which gives rise to convulsive and other involuntary movements. In the two kinds of cases just indicated, although the symptoms are so completely different, there is this similarity, that it is only by an irritation propagated to other parts of the nervous centres that a diseased part of the brain or its neighbourhood produces these symptoms.

The necessity of distinguishing the effects of an irritation from those depending only on a loss of function is very much greater than it is known or supposed to be. The sciences of physiology and medicine cannot be studied without our finding at every moment that wrong conclusions have been drawn from facts, owing to the neglect of making that necessary distinction. I will only mention a few facts to show the importance of making such a distinction. Magendie, in a celebrated experiment, divides the trigeminal nerve, and finds that the senses of sight, hearing, and smell are lost as well as those of taste and of touch (in the face) on the side of the operation. Not thinking of that distinction, he concludes that the real nerve of these senses

\* I ought to say that many of the views which are to be demonstrated in this course of lectures have already been made public, some so long ago as 1861 in my Galstonian Lectures at the Royal College of Physicians, and the rest in my Course of Lectures at the Faculty of Medicine of Paris in 1869, and in lectures I have delivered in 1867, 1872, and 1873, at New York, Boston, Philadelphia, Baltimore, Brooklyn, and Washington, in the United States. Many of my views have also been published in THE LANCET, the Archives of Scientific and Practical Medicine, and the Archives de Physiologie Normale et Pathologique.

is the trigeminal. Serres and others find that in man also those senses are sometimes lost on the side of a lesion of that nerve. They did not see that the lesion of that great nerve irritated its fibres and produced an arrest of the activity of all the special sensorial nerves,\* so that the effects were due to an irritation and not to a cessation of function of the trigeminal. If, instead of paying attention only to the cases of lesion of the trigeminal nerve producing a cessation of action of the sensorial nerves, all the cases of alteration of that nerve had been studied, it would have been found that the effect discovered by Magendie is not constant. There lies, as I will show in a moment, the great difference between the effects of irritation and those of loss of function, the first being essentially variable and often or sometimes missing, the second being absolutely constant.

The physiological history of the cerebellum is crowded with mistaken suppositions that would never have encumbered science, and blocked its path, if the distinction between the effects of irritation and those of loss of function had always been present to the mind of the able physiologists who have propounded these erroneous views. It is enough to recall here the suppositions that the cerebellum is the centre for the equilibration of the body, for the muscular sense, for the genital organs, for the movements of the eye, &c. I wonder that it has not been maintained that that nervous centre is also the centre for sight and for the movements of the stomach, as its lesions so often give rise to amaurosis and to vomiting.

The efforts recently made to locate special centres in the auditory nerve, or in the semicircular canals, might also have been avoided had the distinction I insist upon as being so important been clearly present to the mind of the ingenious physiologists who have made those attempts.

Up to a time not very far off the alterations of nutrition which so often follow the injuries to a nerve, or to the spinal cord, in man or in animals, have been attributed to the loss of a supposed function of the nervous system, consisting in the maintenance of the normal state of nutrition in the various tissues and organs. Led by the distinction I am now speaking of, I found, in 1849, that the alterations of nutrition depend on irritation of nerves, or of the spinal cord, or upon peripheric causes of irritation, and not on a pretended loss of function. I will refer to the admirable work of my friend Professor Charcot† for a full discussion of this subject and a demonstration of the correctness of my view, which he has almost made his own in contributing so many facts to support them.

Blaming others, as I do, for having committed the mistakes I have mentioned, and for having in that way delayed the progress of science, it is my duty to say that I have also been guilty of the same fault. When I found that animals always died in a very short time (much shorter than after the extirpation of the kidneys) when I had taken away their supra-renal capsules, I looked upon those small organs as having a function of so great an importance that life could not persist when that supposed function was lost. The truth is that the phenomena which follow the ablation of these singular organs are effects of an irritation of their nerves, and not effects of a loss of their function or functions. The power of these nerves is fully demonstrated by these two facts—first, that, as I have found, their irritation by compression (when the hilus of the capsule is tied strongly), if limited to one supra-renal organ, will produce rotatory movements; and, secondly, that their reflex inhibitory influence on the heart and the respiratory centres is considerable.

All the conclusions recently drawn by a number of ingenious physiologists and physicians from the effects of irritation of parts of the cortical substance of the brain, by galvanism or by disease, in animals and in man, having led to the view that in those parts are located the psycho-motor centres for the movements of the upper or the lower limb, of the eyelids, the neck, the face, the tongue, &c., are as

many great mistakes that might have been avoided had the distinction between effects of irritation and effects of a loss of function been properly appreciated by these investigators.

The effects of irritation for the same lesion in the same place are characterised by their very great lack of constancy; they may not appear at all, and when appearing their variety may be extremely great. Indeed, that variety may be considered as infinite, and it seems to be so, not only for the intensity or degree, but also for the kind, the place of appearance, and the association of those effects. Not so for the effects of a lesion merely producing the loss of the special function of the part where it exists. Here we find absolute constancy of existence and invariability of kind. These effects of course consist in the cessation of the function or functions of the parts destroyed or altered, and of the results of that cessation. A section of a nerve or of the spinal cord, the destruction by disease or otherwise of the whole brain, are good illustrations of the effects of lesions producing a loss of function.

An irritation can produce two very distinct sets of effects, while a lesion destroying a part endowed with a special function can produce but one set of effects. In this last case there is only and always the loss of the special function belonging to the diseased part, and the direct results of that loss; in the other case there may be, from an influence exerted on other parts, a cessation of their action (loss of function), or the production of a secondary irritation, through which an immense variety of phenomena can appear. Under the influence of irritation, therefore, the activity of nerve-cells of more or less distant parts of the cerebro-spinal centres may be either inhibited (arrested) or morbidly increased and put in play.

The best criterion to find out if an effect is really due to the loss of function of a diseased part of the brain is whether it is or not *constantly* observed in cases of destruction of that part. According to this rule, no special function can be admitted to belong to a part of the brain if there is even one single case only without a complete loss of the supposed function, when the part, if considered as a conductor, has been cut across, and, if looked upon as a central organ, has been entirely destroyed. In other words, the supposition that a special function belongs to a part of the brain receives a death blow if even one case only exists in which that function persists in the circumstances just mentioned for a conductor or a central organ. Not only such a fact is a decisive evidence against our admitting that the part possesses the supposed special function, but it is also a proof that when an organic alteration of that part causes a loss of function, it is through some change in other parts due to the propagation to them of an irritation, starting from the diseased part or its neighbourhood. Two striking instances of the value of this criterion are furnished by the physiology of the medulla oblongata. Long ago I found that the part considered by Flourens as the "focus of life," the only origin of the supposed "vital force" can, although rarely, be taken away without any greater harm than the slow apparition of a lasting epilepsy, and that the reason of the sudden death occurring in almost all instances when that small part (the grey matter at the nib of the calamus) is taken away consists in the effect produced by the irritation of the neighbouring parts on other parts of the nervous centres and on the heart. I also found long ago that a complete transversal section of the medulla oblongata below the level of the part considered as containing the respiratory centre, is not constantly followed by a cessation of respiration and death, showing conclusively that the respiratory centre extends into the spinal cord.\* If respiration stops when the medulla oblongata is divided, it is owing to an inhibitory influence exerted on the nerve-cells above and below the section, and not because of the separation of a part or of the whole of the respiratory centre from its bonds of union with the respiratory muscles. A mere prick can produce as great an effect as the crushing of the whole medulla oblongata.

Other parts of the brain, which have till our days been considered as inexcitable, possess, however, almost as much, if not more excitability than the medulla oblongata. I have already shown, in my lectures at the Royal College of

\* My publication of this experiment was made in 1853. It has not attracted attention, but now that a German has *rediscovered* the same fact it will soon be known all round the world.

\* I will try to show elsewhere that this arrest of activity of the nerve-cells and nerve-fibres of the sensorial nerves is an inhibition similar to that of the nerve-cells of the heart when the par vagum is irritated, and will then mention facts showing that an effect as extensive and as great as that of a lesion of the trunk of the trigeminal can take place from an irritation of a nerve-twig, or a branch of that nerve. I will show also that an irritation of the optic thalami will also sometimes produce, by a similar mechanism, the same effects.

† *Leçons sur les Maladies du Système Nerveux, faites à la Salpêtrière.* 2me Edit. Paris, 1875. Vol. i., pp. 1-152.

Physicians, in 1861, that symptoms of brain disease are mainly due to irritation, which of course implies the existence of excitability. Thousands of cases of disease of the brain show how great is the excitability of the various parts of that organ. The experiments of Fritsch and Hitzig, those of Dr. D. Ferrier, and especially those of an able young French physiologist, Dr. Bochefontaine,\* have shown how powerfully a galvanic irritation may act even on the convulsions of the brain. I have obtained also from another mode of irritation—i.e., heat applied either to the convulsions, to the white substance of the cerebral lobes, or to the great masses of grey matter at the base of the brain, a considerable variety of effects, showing how great is the excitability of all parts of the brain.

The clinical and the experimental facts showing how various the effects of an irritation of the brain can be, prove that that organ is not to be considered as different from the peripheric parts of the nervous system. As I have maintained for a long time, and as I expect to prove fully in the course of lectures, we are to look upon all the symptoms of brain disease, such as paralysis, anæsthesia, amaurosis, aphasia, insanity, convulsions, &c., as being produced by the same mechanism, whether they arise from an irritation in any part of the trunk or limbs, or from an irritation in any part of the meninges or of the brain itself. The excessive excitability of the tissues of the brain is a very important argument in the demonstration of that analogy of mechanism.

That symptoms of brain disease can arise from an irritation is amply shown by the facts above mentioned, but there are other proofs of the possibility of that origin of those symptoms. The now well-known power of disease of, or injury to, the brain of producing alterations of nutrition, either in that organ itself at a distance from the primarily altered part or in the cerebellum, the medulla oblongata, the spinal cord, its nerves and the organs they go to, throws a great deal of light on the mode of action of irritation in producing the ordinary symptoms of brain disease. A change of nutrition being possible at once in most parts of the nervous centres from a limited irritation in the cerebral lobes or the base of the brain, it is easy to understand that symptoms may arise from that change of nutrition. When we see, for instance, that immediately after a certain irritation of the brain surface in dogs—as I have found and shown to the Biological Society of Paris—all the symptoms of meningo-myelitis may appear at once in the dorso-lumbar part of the spinal nervous centre, is it not easy to understand that symptoms of brain disease may be due to a sudden change of nutrition in some parts of the cerebro-spinal axis in cases of organic disease of the brain? Does not the same conclusion naturally flow out also from alterations like those I have found in the lungs immediately after a prick or a cut of certain parts of the base of the brain? These alterations consist—first, in anæmic spots, absolutely white and bloodless, not a single globule remaining in the capillaries; secondly, in spots of œdema, in which, as seen by my friend, Professor Ranvier, there were only white corpuscles and serum in the capillaries; thirdly, in ecchymoses and sometimes large effusions of blood; fourthly, in more or less extended foci of congestion resembling red hepatitis. Does not also the extreme rapidity of the formation of bullæ and of gangrene (bedsore) on the nates on the opposite side to that of brain disease, as found by my friend Professor Charcot, lead to a conclusion like that I have mentioned?

There can be no doubt that changes of nutrition may take place almost anywhere in and out of the nervous centres from an irritation of some part of the brain. There can be no doubt also that symptoms may arise from these alterations of nutrition. But much more than this can be proved by the study of cases of brain disease, as it shows that symptoms arise neither from a loss of the special function of the part diseased nor from a direct and immediate effect of the manifestation of a special property or power of such a part (as my ingenious friend, Dr. J. Hughlings Jackson, believes), but from an influence exerted on other parts at a distance from

the seat of an organic lesion, and caused by an irritation of the diseased part or of its neighbourhood.

The proofs of the correctness of those views are superabundant.\* I will refer for the details about them to my future lectures, and will only give here the following propositions, each of which simply constitutes a summary of facts. 1st. A lesion in one half of the brain may produce symptoms either on the opposite or on the corresponding side. 2nd. A very small lesion, whatever be its seat, can produce most extensive and violent symptoms. 3rd. A lesion occupying the same extent on the two sides of the middle line of the brain may produce symptoms only or chiefly on one side of the body. 4th. Symptoms may appear suddenly from a slowly and gradually-developing lesion. 5th. Symptoms may appear slowly from a suddenly-produced lesion. 6th. The greatest variety of symptoms may proceed from a lesion in the same part of the brain. 7th. The most various parts of the brain can give rise to the same symptom. 8th. Permanent lesions may produce symptoms by attacks, just as they produce epileptiform seizures. 9th. Symptoms may cease suddenly or rapidly notwithstanding the persistence of the lesion. 10th. Symptoms of brain disease may appear from an irritation of visceral and other peripheric nerves. 11th. Considerable lesions anywhere may exist without the appearance of symptoms.

To sum up all those propositions, I will say that there is no necessary relation between the seat, the extent, the kind of a lesion, and the symptoms that may appear from its influence. According to the criterion above mentioned, symptoms being so inconstant and so variable from the same lesion in the same part (whatever it be) of the brain, must be considered as effects of irritation, and not as effects of loss of function. Besides, their variety, even when they proceed from a lesion in the same place, is too great for our considering that those which are clearly due to an irritation, such as convulsions, vomiting, &c., are mere manifestations of the special properties or powers of the part where there is a lesion.

If these propositions are right, as I will fully demonstrate in other lectures, it is clear that we must give up all or almost all our notions about the physiology and the pathological physiology of the brain. What of the notion that the left side of the brain is the seat of the will power that moves the right limbs and of the power of perception of sensations coming from those limbs, if we find that a lesion of that left half of the brain can produce paralysis or anæsthesia not only in the limbs on the opposite side, but also on those on the same side†, and if we find, besides, that a considerable lesion existing anywhere in that left half of the brain may cause neither paralysis nor anæsthesia? What of the notion that the corpus striatum on one side serves to the movements of the limbs on the opposite side, and that the optic thalamus serves to sensibility for the limbs also on the opposite side, if both the optic thalamus and the corpus striatum on one side can be destroyed without paralysis or anæsthesia on the opposite side? What of the idea that the crus cerebri and the anterior pyramid‡ on one side are the only channels for the transmission of the orders of the will to the limbs on the opposite side if they can be deeply altered without paralysis? What of the notion that there are motor centres in the convulsions of the brain producing convulsions in the limbs on the opposite side if we find that these pretended centres can be irritated by tumours or inflammation without generating convulsions; and if, also, we find that they can produce those spasmodic movements on the side of the lesion? What of the localisation of the faculty of expressing ideas by speech, either in the third frontal convolution (Broca's view), or in the island of Reil (Professor Sanders's and Meynert's view) in the left side of the brain, if we find that disease anywhere else in that side of the brain may produce aphasia, or that disease of the right side of the brain may also produce it, or also that disease may exist in those places without aphasia, or, again, that aphasia may be cured although the disease in those places that had given rise to it continues to exist?

\* In his important paper Dr. Bochefontaine shows that a galvanic irritation of the surface of the brain has a very great power on the general circulation of blood, on the biliary and pancreatic secretions, on respiration, on the stomach, the bowels, the spleen, the uterus, and the bladder.—See Archives de Physiol. Normale et Pathol., Paris, 1876, p. 140-172.

\* A part of the proofs that can be given will be found in my papers on the origin of brain symptoms in my American journal, the Archives of Scientific and Practical Medicine, February and March, 1873.

† See my Lecture on Paralysis on the Side of the Lesion in the Brain in THE LANCET of January last, pp. 1, 76, and 159.

‡ See the already quoted Lecture in THE LANCET, p. 159, vol. i, 1876.

When, in the subsequent lectures, I give details on these points, I will also give other decisive arguments against the views recently proposed about the special functions of many parts of the brain.

It may be said that a good deal of what I have stated against the admitted views has value only if there is no possibility that one side of the brain will sometimes assume the functions lost by the other side. It is yet to be proved that one half of the brain can come to the rescue and perform, besides its own functions, those of the other. The view I maintain—i. e., that one half of the brain has power enough for the performance of the functions of the whole brain—seems to imply my acceptance of that supposition. Not entirely so: the study of clinical facts clearly shows that although we have two brains instead of one, most people develop the powers of only one for certain actions and of the other for certain other actions; so that if, for instance, an affection destroyed in the left brain the supposed centres for volitional impulses to the right limbs, the same supposed centres in the right brain would be, from lack of development of power, unable to perform the special function of the left centres. But the theory that there will be no symptom or a cessation of symptoms, notwithstanding the destruction of pretended special centres in one side of the brain, because the healthy other side will perform the functions of the injured one, must be cast aside in presence of facts—and there are many such—in which the supposed centres for special functions are destroyed in the two halves of the brain, without any loss of these functions.

In another lecture I will show how the physiology of the brain is to be brought to harmonise with clinical facts. I will only give here a short general outline of some of my views.

1. As regards localisation of function, a great many facts lead to the view that the nerve-cells endowed with the same function, instead of forming a cluster, so as to be in the neighbourhood of each other, are scattered in the brain, so that any part of that organ can be destroyed without the cessation of their function. It makes no difference whatever whether the distance between nerve-cells employed in the same function is a small fraction of a millimetre or very much greater, as in either case their communications with each other must take place by conductors (nerve-fibres), the length of which is unable to interfere with the function.

2. Each half of the brain is a complete brain originally, and possesses the aptitude to be developed as a centre for the two sides of the body, in volitional movement, as well as in all the other cerebral functions. Still, very few people develop very much, and perhaps nobody quite fully, the powers of the two brains; and, on the contrary, in most persons only one of these two primitively similar organs acquires great power for certain actions, and the other for other actions.

3. Communications between the body and the brain can be more or less fully accomplished by means of a very much smaller number of conductors than would be necessary according to any view like the well-known clavier theory. As we know that the will only gives an order, and as we know by clinical facts that any part of the medulla oblongata can be destroyed without paralysis, and that in some cases a very small portion of it has proved sufficient for the persistence of voluntary movements, it would seem that the order may be transmitted as well by one fibre as another, and that it is necessary to recognise the existence of faculties of a much higher order in the nerve-cells of the spinal cord than those which are admitted to exist there. Many facts and a similar reasoning tend also to show that the nerve-cells of the spinal cord possess, as regards sensibility, faculties of a higher order than those which are admitted.

This is all I will say now about the physiology of the nervous centres.\* As regards the physiological pathology of the brain, I will prove in this course of lectures that symptoms of local brain disease arise only from irritation. I will show, also, that irritation acts either in stopping an activity or putting in play or exaggerating morbidly an activity. If it stops an activity, it is, as I will show in another lecture, through an inhibitory influence exerted on the nerve-cells endowed with that activity and widely diffused in the nervous centres. If it causes a manifestation

of a normal activity, or produces a morbid activity, it acts in the same way as a peripheric irritation when it produces similar effects.

Two kinds of symptoms therefore exist, both being due to irritation: in one kind a cessation, in the other a manifestation, of an activity. To the first kind belong paralysis, anæsthesia, amaurosis, aphasia, loss of consciousness, &c. To the second belong delirium, epileptiform and other convulsions, spasmodic rigidity, trembling, choreic movements, vomiting, hiccough, rotatory or circus movements, &c.

When I treat of paralysis, as I shall do in the following lectures, I will give the arguments on which I ground the view that the first of the two above series of symptoms depend on an irritation starting from the seat of an organic lesion and acting on more or less distant nerve cells so as to produce by inhibition an arrest of their activity.

## ON GELSEMINUM SEMPERVIRENS.

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(Continued from vol. i., p. 734.)

### ACTION ON THE PUPILS.

STRANGE to say, the internal administration of gelseminum produces an effect opposite to that occasioned by its local application to the eye itself. When given by the mouth in doses sufficient to produce symptoms, the drug, in every instance but one, caused contraction of the pupil. Indeed, in the case in which the drug produced weakness of the legs with a strong double internal squint, the pupils were contracted to a pin's point. In the exceptional patient just referred to, on whom we made two observations, though on each occasion we produced great giddiness, haziness, susceptibility to light, and diplopia, the pupils did not contract, but manifested a very unsteady condition, varying greatly in size in rapid alternation without any apparent cause. In another patient, the pupils were strongly contracted when exposed to light, but they dilated freely in a darkened room; and when they recontracted on exposure to light they seemed in an unstable condition, oscillating a good deal. The contraction of the pupil does not cease on the disappearance of diplopia or dimness; indeed, when the dimness has passed away, the contraction of the pupil may increase.

In the cases of accidental poisoning it is recorded that during complete general paralysis the pupils are widely dilated, and therefore it might be inferred that the condition of the pupil depends on the dose—a moderate dose contracting, a very large one dilating, the pupils. This explanation is possibly in part true; but the dilatation might be due likewise to the asphyxia induced by large doses. Thus Dr. O. Berger finds, and our observations confirm his statements, that dilatation of the pupils in poisoned animals occurs only when asphyxia from paralysis of respiration has set in, and that artificial respiration at once causes the pupils to contract.

On the other hand, the topical application to the eye dilates the pupil. We employed, in the first instance, a tincture (1 in 10) and the American liquid extract. These preparations dilate the pupil slightly, but they excite great irritation and smarting. We then employed the alkaloid prepared by Mr. Gerrard—one grain in twenty minims of water, a solution which causes scarcely any smarting. Mr. T. Fox and Mr. Sydney Pearce have made for us nineteen observations. A few drops of the solution were put into one eye, and in each case this trial pupil became widely dilated, the dilatation usually beginning in about thirty minutes. Not only does the pupil dilate, but the muscle of accommodation becomes paralysed and the sight affected. In twenty-four hours vision again becomes nearly natural,

\* For other views of mine on this subject see the Archives of Scientific and Practical Medicine, Feb. 1873, p. 119 et seq.