I can only run over these matters briefly and cannot touch all the points. The patient has something the matter with his lungs, and therefore it is often thought that the first thing to do is to give expectorants. What do we mean by expectorants, and what is their action? We mean either that expectorants assist the patient to cough up the secretions in their tubes—to "loosen" it, as they say—or that the expectorant produces a secretion in the tubes. Of course we do not want to produce any secretion in the tubes in pneumonia, and there is not anything except in the pneumonic parts to bring up. So expectorants are contraindicated, and clinically they are found not to do good.

The patient has a cough. What is the use of that cough to him? It is intended to help him to get rid of the little secretion in his tubes, and therefore it is a thing to encourage and not to stop. And where the cough is not very troublesome you need not worry about it. This raises another question: Shall you give anything like that very favourite remedy, Dover's powder? That contains opium. What will be the effect of giving opium? You check the patient's sensibility and he will not get rid of the stuff out of his tubes. Remember that opium is a drug which must be employed with very great care in pneumonia.

Sleeplessness is another most important symptom to treat in pneumonia. Many patients sleep brokenly, but still they sleep. Other patients cannot close their eyes, they long for sleep but cannot get it, and they lie awake day and night. If you do not procure them sleep somehow, they will die of sheer exhaustion. Another point is, that not only are the patients sometimes sleepless, but they are actually delirious and wild, and are constantly getting out of bed, or trying to. And drinkers get delirium tremens under these circumstances, and if they are allowed, they run all about the room, seeing things. These patients frequently die. But if there is no delirium tremens, no drink, but an ordinary case of pneumonia with nervous symptoms, in a patient who has been working the brain very actively, in such patients pneumonia takes on the nervous type; they are often found to be unable to obtain sleep, and they become actively delirious. The other day I went to see a gentleman who had had no sleep for at least forty-eight hours, and yet his temperature had fallen and apparently his pneumonia was resolving, or at any rate had come to an end. He was incessantly talking and fidgeting, and trying to get out of bed and jump about. What shall you do for such a case as that? You must get him to sleep, and at once. How shall you do it? You cannot give large doses of chloral, because of the action which that exerts on the heart. It is no good playing with sulphonal and paraldehyde and things of that sort, which may or may not affect him, but will take some hours to do so in any case. It is no good giving potassium bromide, and you cannot give chloroform in such a case, and so it comes to this, can you give a subcutaneous injection of morphine? You must. The only contraindication to giving morphine in any chest complaint is a large amount of secretion in the tubes, because it prevents the desire to cough so as to get rid of it. But in a number of these cases there is nothing obviously wrong in the other parts of the lungs—only in the part which is inflamed—and if one lung is free of disease you may give some opium. But you must be prepared for frights after you have given it. You may give an adult patient one-third of a grain of morphine, and if he does not sleep as a result of that, you may give him another. With that dose of two thirds of a grain he may go to sleep, and then you find after a time that you cannot awaken him, and you begin to think that you have poisoned him with morphine; but this is not the case. The fact is, you have got him to sleep at last, and he is by that time so exhausted that it is almost impossible to wake him up for a time. So remember it requires careful consideration very often whether you shall give morphine or not, and in many cases the practitioner does not care to give it, and likes to share the responsibility with somebody else, whom he calls in. Yet it is the only thing to do if your patient is likely to die of exhaustion.

But in relation to sleeplessness and delirium, there is one drug which I think I ought to caution you against, because it has been used a good deal, and that is hyosciamin. My experience of that drug has been unfavourable. You will find it advocated for delirium

and it does good in mania and similar mental states, but in my opinion its use is very risky in cases of pneumonia. What you may give to a healthy lunatic with success may quite well be harmful for pneumonia.

But what shall you do for these delirious cases, short of giving morphine? And we do not use morphine as a rule for delirium. There are several things to bear in mind. What is the cause of the delirium? The high temperature is very likely to be the cause, and so we have to consider the various means of reducing temperature. Sometimes if you put cold applications on the patient's head you help him to get to sleep, or you may put the patient's head over the edge of the bed and take a watering-can and give it a shower bath. That will often send him to sleep. Now, some patients do not get markedly delirious until the temperature is practically normal, and that is the delirium of asthenia or exhaustion, and if you give the patient a little alcohol and a little sedative, the delirium will subside. Delirium in convalescence is generally an indication for stimulation of some kind.

Supposing you have your patient convalescent, I would emphasize again what I said earlier in the lecture—during the first ten days examine your patient carefully. As a warning which will be useful, I will give you a case which happened a little while ago. A patient who had had pneumonia in the hospital was perfectly well, as we thought. He sat up in bed to eat his dinner, and fell back dead. Apparently he was quite well at the time he sat up. It was, of course, a great shock to everybody. We found empyema on both sides, and the pericardium full of pus. It would have been impossible for that to have been missed if any examination had been made of the base of his lung, or heart either. I mention that to show how very insidious are some of these suppurations which occur in pneumonia. If we had examined him regularly we must have had our suspicions roused. If we had suspected pus we should have found it, let it out, and possibly saved the patient.

Lastly, let me once more impress upon your mind that when treating a case of pneumonia you are in for a big fight, and that you are most likely to win if you never lose heart, but fight on valiantly to the finish, however desperate the struggle may seem.

The Purvis Oration

ON THE

DIAGNOSIS OF ORGANIC FROM FUNCTIONAL AFFECTIONS OF THE NERVOUS SYSTEM.

DELIVERED BEFORE THE WEST KENT MEDICO-CHIRURGICAL SOCIETY,

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HAD I not been sensible of the honour that the invitation to deliver this oration carries with it, a cursory glance at the list of past orators would have sufficed to convince me that I have been paid a high compliment. Although I needed no such reference to the list of previous orators to make me appreciate the honour, I have been unable to prevent myself from pondering over and wondering how it would be possible for me to acquit myself in a manner that would justify your associating my name with those of such distinguished members of our profession, whose orations must have contained a wealth of experience and information far beyond anything that I can possibly pretend to have at my command—fitting memorials of Dr. Purvis, in whose honour we are assembled this evening.

The subject I have chosen may be treated in many different ways, but for the purposes of this address it appears to me that cases in which the problem to be decided is as to whether we are dealing with a case of organic disease of the nervous system, as opposed to one in which the symptoms are due to functional disturbance, fall conveniently into one of four classes:

1. Cases in which the nature of the affection is such that if organic disease exists we may expect to find certain physical signs which are more or less inseparable from the affection.

2. Patients suffering from an affection in which certain physical signs may be determined, but these indicate functional disturbance of the nervous system, and not organic disease.

3. Those in which the affection is such that, although organic, no physical signs, such as those that prove so helpful in the first class, are to be expected, as they do not form a necessary part of the clinical picture of such cases.

4. Others in which the symptoms are due to functional derangements of the nervous system, but in which no special signs can be determined, from the existence of which it can be argued that a functional, as opposed to an organic, affection is to be held responsible for the symptoms.

It will readily be recognized that the cases that belong to the first class and those that belong to the second are far easier to pronounce an opinion on than those which are included in the third and fourth classes, for, instead of being able to rely on the presence or absence of certain well-defined physical signs, in both of these groups of cases the general features of the malady, the behaviour of the patient, and a variety of other factors have to be taken into consideration in making a diagnosis, all of which require special knowledge and experience to guide the medical man and assist him in forming his opinion. In short, any one can be taught that the presence of this or that physical sign means that the condition is organic, while, if certain other physical signs be present, the affection is functional. But when the case does not belong to either of these classes, then the diagnosis can only be made from a thorough knowledge of the general features of the disease which are to be expected if it is organic, as opposed to the absence of certain important details in the symptomatology if functional disturbance of the nervous system is simulating the organic malady, whose features it thus only represents in part. In addition to this, the functional affection that is a mimicry of the real disease may have certain peculiarities of its own which are foreign to the organic malady; or the general behaviour of the patient, whether it be by exaggeration of the symptoms, want of control of the emotions, or otherwise, may reveal the true nature of the condition to the experienced observer.

It thus becomes evident that it must often be very difficult for any but those who are constantly dealing with such cases to form a correct estimate of the patient's condition with sufficient confidence to warrant a grave prognosis on the one hand, or to justify the adoption of rigid measures in treatment on the other. These difficulties in diagnosis must of necessity be the greater the earlier the case comes under observation and the less pronounced are its symptoms.

1. ORGANIC DISEASE WITH PHYSICAL SIGNS.

Let me illustrate what I mean when I say that cases included in the first class may be comparatively simple to diagnose, owing to the fact that any one may be taught to recognize that this or that physical sign indicates the existence of organic disease of the nervous system. The student of neurology may be shown how to use the ophthalmoscope, and thus he may learn to recognize the changes in the optic nerves which constitute neuritis or atrophy; and he may be taught that the altered conditions of the optic nerve in either case are sure indications that an organic disease exists, and that under no circumstances could uncomplicated hysteria or neurasthenia produce either condition. Similarly he may be shown how to elicit the knee-jerk, and how to employ reinforcement when it is difficult to obtain any response when the patellar tendon is struck. He can be told that if the knee-jerk is truly absent, he should not permit himself to ascribe this to functional affection of the nervous system, as he would have every justification for the belief that organic disease must be present to account for an abnormality of the kind.

One more example must suffice. The student can be shown that if in a healthy person the sole of the foot is stroked so as to evoke a reflex movement, the toes flex, and that the great toe may participate in this movement of flexion; whereas in persons suffering from certain diseases of the nervous system, when the sole is similarly stimulated, the great toe is drawn back towards the dorsum of the foot in a very characteristic manner. He can be taught that this abnormal phenomenon is the result of organic disease which has affected some part of the pyramidal system, either in the brain, the mesen-

cephalon, or the spinal cord, and that, if he can be sure that his observation is free from fallacy—as, for instance, that a voluntary movement of the toes has not been substituted for a true reflex—he is justified in assuming that something more than hysteria or neurasthenia exists to account for such a change in the character of the plantar reflex, and that some organic change must be going on in the nervous system, no matter how strongly the clinical picture may otherwise suggest the probability of functional disturbance.

It is probable that in no class of case is the mistake more commonly made of regarding symptoms the result of organic disease of the nervous system as due to functional disturbance than where disseminate sclerosis is concerned. It frequently happens that neurasthenia or hysteria is supposed to account for a clinical picture occasioned by this disease; and in a large number of these cases the abnormal type of plantar reflex has been the chief physical sign by which it has been possible to establish the diagnosis of the organic affection, a diagnosis which subsequent developments have unfortunately proved to be fully justified.

Although a single physical sign may have to be relied on for the diagnosis of organic disease, it is impossible to deal with this part of the subject without a word of warning in regard to the possibility of misinterpreting physical signs and of establishing a diagnosis of some grave and progressive malady of the nervous system on insufficient data. The mistake of regarding as organic what is in reality only functional, in my experience more often results from the belief that exaggeration of the knee-jerks is of necessity a sign of organic disease than under any other circumstances. It repeatedly happens that patients are regarded as suffering from some form of spastic paraplegia, due to sclerosis of the lateral columns of the spinal cord, when the only justification that can be offered for the diagnosis is the fact that the knee-jerks are exaggerated. It cannot be too strongly insisted that hysteria and neurasthenia may both occasion the most pronounced exaggeration of the knee-jerks, and that accordingly, unless this condition is supported by a true ankle clonus, the extensor type of plantar reflex, or some other undoubted sign of organic disease, a diagnosis of the kind cannot be justified by exaggeration of the knee-jerks alone.

There are, however, other cases in which the diagnosis appears more justified, in view of the fact that some other abnormality exists in addition to the exaggeration of the knee-jerks. Here again an incorrect interpretation may be placed upon the abnormal phenomena that are present. Thus it has happened that a patient with pes cavus, but no other sign of any known disease of the nervous system, has been regarded as probably suffering from some form, of spastic paraplegia, because in addition to the deformity of the feet the knee-jerks were exaggerated. In another case in which the knee-jerks were unduly active in a healthy, athletic young man, in whom there was neither rigidity of the legs, ankle clonus, nor the abnormal type of plantar reflex, but who had long been known to have a form of nystagmus, in common with several other members of his family, a similar mistake was made. This otherwise healthy young man, in whom there was no real reason to suppose the existence of any organic disease of the nervous system, was rejected on account of his health, in a competitive examination for an appointment, there can be little doubt, because disseminate sclerosis was suspected by the medical man who examined him.

Do not let it be supposed that I wish to minimize the importance of nystagmus as a sign of organic disease of the nervous system, for it may be a most important indication of serious mischief. Nystagmus is, however, capable of different interpretations. Taken in conjunction with certain other signs of organic disease of the nervous system nystagmus is all-important in diagnosis, but as an isolated phenomenon, unsupported by any other undoubted sign of organic affection, it cannot be allowed the undue weight which was evidently given to it in the case to which I have referred.

It must be remembered that organic signs may be long delayed in a case in which, nevertheless, they ultimately appear. This being the case, a diagnosis of functional affection of the nervous system to account for the symptoms cannot be made without considerable anxiety in many instances. In no class of case do I have greater

misgivings than when a patient complains of what suggests sciatica as the cause of pain in a leg, but in which no positive signs of neuritis of the sciatic nerve can be determined. Time after time cases of the kind ultimately prove to be due to malignant disease of some kind in the pelvis, although all the methods of examination at our command may long fail to detect any such source of pressure upon the nerves of the lumbosacral plexus.

This leads me to remind you that even when positive signs of affection of the sciatic nerve appear we are not freed from anxiety, as the physical signs which indicate interference with the functions of the nerve may equally well be produced by pressure on it as by a neuritis, as the effects are more or less similar. The state of the ankle-jerk is very important in this connexion, for when there is only functional disturbance the jerk is commonly abnormally active; whereas when the sciatic nerve is damaged it is diminished or abolished. Reliance on this sign has proved very helpful in a particularly difficult case, in which a neurotic man has persistently complained of pain in one leg for many months. His antecedents were such that involvement of the lumbosacral plexus in chronic inflammatory material was not only possible, but very probable. Abdominal and rectal examinations failed to determine a mass of any kind in the pelvis that could be involving the plexus, or causing pressure on the nerve, while from a neurological standpoint it was legitimate to argue that it was improbable that in a case in which any serious mischief was involving the plexus or nerve the patient could continue ill so many months without revealing any alteration of the ankle-jerk, some patchy anaesthesia, loss of motor power, or muscular atrophy. Indeed, instead of any diminution of the ankle-jerk in this case, it has been abnormally active on both sides, as have been the knee-jerks, a state of things quite compatible with functional disturbance of the nervous system.

In such a case, however, it is impossible to feel quite happy, owing to the knowledge that organic signs may be long delayed in obscure cases, and may yet ultimately appear. This makes it most important that we should observe the greatest caution before expressing too confident an opinion in all such cases.

This is well illustrated by the case of a man who was sent to University College Hospital suffering from a train of symptoms which are usually ascribed to neurasthenia. I saw and examined him on several occasions over a period of many months, during which time his symptoms did not improve, in spite of all that was done to restore his nerve tone. Indeed, he became progressively worse, and yet up to the last time that I had an opportunity of examining him he was free from any of the physical signs on which we rely for the diagnosis of an organic condition, which must have been underlying his neurasthenia, and which ultimately revealed itself. Although I have not had an opportunity of examining the man since the organic signs appeared, my colleague, Dr. Grainger Stewart, has done so, and has determined that he now has undoubted evidences of subacute combined degeneration of the spinal cord, an affection which usually proves fatal within a few years, or even months.

The following is another example of how a clinical picture that appears to be due to neurasthenia may ultimately prove to be produced by organic disease. A man met with an accident, in which he received various severe injuries, but presented no immediate symptoms to suggest a gross injury to his spinal cord. When he first came under my observation he complained of the usual train of symptoms that are ascribed to traumatic neurasthenia. A very careful physical examination of him failed to arouse the slightest suspicion that any organic condition was present. I was thus able to reassure him and encourage him in the belief that under appropriate treatment he would ultimately get well, although it might take a long time before this happy result was obtained. Some months later I had another opportunity of examining him, when I found that he was worse instead of being better; but he still presented no sign of organic affection, or evidences of structural damage to his spinal cord. His knee-jerks had been exaggerated from the first, but his lower limbs were free from spasticity, there was no ankle clonus, and his plantar reflex was of the normal flexor type on both sides. It was only on a third occasion, when I examined him about a year after the accident, that I determined the existence

of ankle clonus on both sides, and found that the plantar reflex, which had hitherto been of the normal flexor type, had altered, and had become extensor. It was accordingly impossible to continue to support the hopeful prognosis on which I had ventured when I first examined him.

2. FUNCTIONAL AFFECTIONS WITH PHYSICAL SIGNS.

Many examples could be quoted in support of what I have said with regard to the ease with which the diagnosis of functional disturbance of the nervous system may be made in the presence of certain positive signs which we ascribe to functional, as opposed to organic, affections of the nervous system, but one or two illustrations must suffice.

In a patient suffering from hysterical paralysis, one of the peculiarities may be the way in which the antagonists are brought into action and hamper the muscles which ought to be performing a given movement. This may be well seen in a case of hysterical paraplegia in which the patient is capable of performing movements of the lower limbs while lying in bed, although it may be in a very imperfect manner. When a patient is suffering from organic paraplegia in which voluntary extension of the lower limbs at the knees is at all possible, if after flexing the limb the observer prevents the extension by resisting the movement, directly the resistance is removed the limb will fall in the extended position. This may be quite otherwise in a case of functional paralysis, for when the resistance is removed, instead of the limb being extended in the way that obtains in the organic case, it commonly is kept semiflexed at the knee, owing to the fact that the hamstrings are as much in action as the quadriceps extensor muscles during the effort that it made to extend the limb.

The following case illustrates another sign which is very characteristic and suggestive of functional as opposed to organic affection. A young girl, who was frightened because atropin was put in her eyes to allow of their being tested for glasses, complained of pain in the eyes and pain in the back. On attempting to rise from her seat she was unable to walk. But for the history of fright, the onset of so acute a paralysis in so young a girl naturally would have suggested poliomyelitis as possible, and the diagnosis would have seemed the more probable owing to the fact that one leg was more affected than the other, and the sphincters had escaped paralysis. On physical examination of the patient I observed that, although the limbs were affected in their entirety, neither the knee nor ankle jerk was altered on either side; both were readily obtained, and were as nearly as could be judged normal in degree, instead of either of them being abolished in the way that might reasonably have been expected in so severe a case of paraplegia, the result of a poliomyelitis. Functional, as opposed to organic, disease of the nervous system was suspected, and the diagnosis, which subsequently proved correct, was further justified by the fact that anaesthesia, pronounced in degree, was determined in the affected limbs, contrary to what was to be expected in a case of poliomyelitis. Although the existence of anaesthesia was against the diagnosis of poliomyelitis, it was of course compatible with that of an ordinary transverse myelitis, which was, however, made improbable by the absence of sphincter affection, and of spasticity or undue flaccidity in the limbs, in addition to which there was no ankle clonus, and the plantar reflexes were not of the extensor type. That the anaesthesia was functional in origin was made clear when, on being told to say "Yes" when she felt a touch, and "No" when she did not feel it, the little girl as readily said "No" when she was touched on the anaesthetic parts as she said "Yes" when the stimulus was applied to an aesthetic region.

I must not allow what I have had to say in connexion with this case to leave the impression that the knee-jerks must of necessity be absent in a case of poliomyelitis or of any other organic lesion of the lower motor neurons, because this would not be correct. It is only when the muscles directly concerned with the jerk are affected that the knee-jerk or ankle-jerk is abolished, the thigh muscles in the one case and those of the calf in the other. I emphasize this because recently I experienced a difficulty in satisfying one of my audience that the diagnosis of a lower motor neuron paralysis was justified in a patient who was unable to perform certain movements of the foot at the ankle, and whose knee-

jerks were abnormally active. In reality the patient's paralysis was the result of trauma, which was also responsible for shock to his nervous system, which caused the instability which accounted for the increase of his knee-jerks, and, inasmuch as the quadriceps extensor muscles were intact and the atrophic paralysis was limited to the anterior tibial and peroneal groups of muscles, it will be evident that there was no reason why the knee-jerk should be absent in a case of the kind, although it would have been had the quadriceps extensor muscles participated in the paralysis and muscular atrophy which involved the muscles below the knee.

3. ORGANIC DISEASE WITHOUT PHYSICAL SIGNS.

We now come to by far the most difficult part of our subject, namely, the classes of case in which there is either an organic affection on the one hand or a functional on the other, but in which no positive sign can be determined which will definitely establish the diagnosis in favour of the one as opposed to the other. I can offer no better example of an organic disease of the kind than is supplied by paralysis agitans, for although this is a malady the clinical picture of which is so well defined and so easy to recognize from its general features, there may be no physical sign to which attention can be directed as evidence of the existence of organic disease in the way that can be done in, for instance, a case of disseminate sclerosis. This being so, it will be readily understood that, even to one who is familiar with the general picture of the established disease, the malady may be difficult of recognition in its early stages, before the general features become at all pronounced. Nevertheless, much may depend on an early diagnosis in a case of the kind. I have seen more than one patient who was in the early stage of this affection regarded as suffering from neurasthenia, in whom there was not sufficient to permit of a definite demonstration of any special features of the malady, and yet in whom there was enough to permit an experienced observer to be confident that paralysis agitans rather than neurasthenia was to be held responsible for the patient's symptoms. Let me call your attention to one or two points that may help in this class of case. When in a clinical picture of what appears to be neurasthenia the loss of power is unilateral, or, it may be, confined more or less to one limb, this should lead to the suspicion that something of a more serious nature must be present. Any stiffness of the limbs or inequality of the tendon-jerks on the two sides should also place us on our guard. I have not as yet had opportunities of observing a sufficiently large number of cases to justify any generalization from the following observation, but I have been struck by the fact that although the knee-jerk is so often more active on the side on which paralysis agitans begins, the ankle-jerk on the same side may be diminished, or even absent, at a time when the knee-jerk is increased. This may prove to be a sign of importance in the diagnosis of the disease in its early stages, but it certainly does not obtain in all cases. There are other cases in which paralysis agitans is quite evident, so definite are the tremors and other features of the malady, but in which the patient is nevertheless supposed to be suffering from a functional tremor because she is young, or perhaps it may be because the tremor has been noticed to be in abeyance when the patient has not been aware that she was being observed. In such cases it may be very difficult to convince a sceptic that an organic rather than a functional affection exists, and yet both observations are quite compatible with a diagnosis of paralysis agitans.

In such cases it may happen that a rhythmical tremor can be determined in certain of the facial muscles, in the tongue or in the jaw, which may be highly characteristic of paralysis agitans, and unlike anything that is seen in a case of functional affection, which otherwise simulates this malady.

It may be equally difficult to satisfy others that a clinical picture has been produced by a functional disturbance which simulates paralysis agitans rather than that the real disease exists, although the observer is himself quite sure of his position in the matter. A good example of this is supplied by the case of a woman of advanced age who was sent to University College Hospital, supposed to be suffering from paralysis agitans. Her age was in favour

of the diagnosis, the tremor of her arm simulated that which is met with in the disease, and it was quite possible that in so early a stage of the affection there had not been time for any of the other features of the malady to appear. Nevertheless, there was something in the woman's appearance and manner, and even in the character of the tremor, which made me suspect functional disturbance rather than organic disease, although it would have been impossible for me to put into words why I held the opposite opinion to that which had been expressed by the medical man who sent the patient to the hospital. That my suspicions were justified was proved by the fact that by means of a faradic current, tempered with that degree of suggestion which is always so useful in the treatment of functional affections of the nervous system, it was possible to persuade the patient that the electricity would rapidly cause arrest of the tremor, a result that was obtained in a way that placed the diagnosis beyond all question.

In another case, in which I have had opportunities of watching the unfortunate man as he has passed through successive stages of his malady, over a period of several years, the patient first came under my observation with a diagnosis of writer's cramp. He was a clerk, and complained of difficulty in writing. There was nothing sufficiently obvious in the case to justify fault being found with the diagnosis of writer's cramp, and yet there was that which made me suspect paralysis agitans. So little was there to justify the diagnosis of paralysis agitans, that it would have been impossible for me to demonstrate in the patient my reasons for suspecting so grave a malady.

Another instructive case was that of a man with slowly progressive hemiplegia, whose condition was regarded as due to an intracranial tumour. In holding a contrary opinion I felt that my position must be regarded as very weak, in that all I could say to the patient's medical attendant in justification of my diagnosis of commencing paralysis agitans was that there was that in the man's general appearance and mode of progression which gave me the impression that I was in the presence of that disease. To define more clearly what I saw and what I meant was beyond me. Nevertheless, the subsequent history of this case proved beyond all question that the ill-defined picture which I was unable to describe was really being produced by paralysis agitans, for in time this disease presented itself in all its well-known features.

The class of case with which we are now dealing may be still more difficult of diagnosis, owing to the fact that the symptoms arise under circumstances that are commonly responsible for functional disturbance of the nervous system.

Thus, a man met with an accident, in which he received a shock, on an electric railway. His symptoms were compatible with those of traumatic neurasthenia, and none of the organic signs on which reliance has to be placed for the diagnosis of structural damage to the brain or spinal cord could be determined. Nevertheless, there was that in the character of his tremor which aroused my suspicion, and which prevented me from falling into the error of diagnosing traumatic neurasthenia when paralysis agitans was really responsible for the symptoms, as was proved by the subsequent course of the case. The association of paralysis agitans with trauma is, of course, well recognized, but the point I wish to emphasize here is that in the case to which I have referred there was nothing in the man's complaints or appearance to suggest paralysis agitans, nor was the tremor characteristic of this malady. All that can be said is that the tremor was rather coarser than that which is usually seen in neurasthenia, and that although increased when he was asked to extend his hands in front of him, some tremor could be determined from time to time when his limbs were in repose. I may, perhaps, be permitted to remind you that it commonly happens that in paralysis agitans the tremor is present when the limbs are at rest, and may temporarily cease when a voluntary movement is made. This, however, is not an invariable rule, as the tremor is sometimes brought out or increased by volition.

4. FUNCTIONAL AFFECTION WITHOUT PHYSICAL SIGNS.

A like difficulty has to be contended with in the class of case in which the symptoms are due to a functional

affection of the nervous system, and in which there are no special signs, such as may obtain in some functional cases, to establish the diagnosis. The following is a good illustration of what is meant:

A woman was seen in the out-patient room at the Queen Square Hospital, with a history from her doctor that there had been two attacks of pneumonia, one of which had been complicated by pericarditis, and that subsequently to this she had developed certain choreiform movements, with inability to speak. The patient was unable to stand or walk, and was apparently so weak that she could not even sit up. Severe spasmodic movements affected all of her limbs, together with her neck and trunk. This was clearly not the kind of case in which I could look for assistance from one or other of the signs of organic disease to which I have called your attention, and physical examination did not reveal any particular signs that may exist in hysteria. My suspicion as to the real nature of the case was, however, aroused when I tried to induce the woman to speak, for, instead of the articulatory difficulty so characteristic of chorea, with the rolling about of the tongue and other unsuccessful efforts at articulation, the patient made a grimace that did not recall to my mind anything that I expected in chorea, and spoke in a whisper without any of the articulatory defects for which I was prepared. With this clue to the real nature of her condition, I was next able to persuade her to control the movements of one or other limb, so as to enable me to test her tendon jerks and plantar reflexes, and with isolation and strong faradism, combined with suggestion, she rapidly recovered in a way that would have been quite impossible had true chorea accounted for the clinical picture.

The following is another case in which the diagnosis of functional disturbance had to be made without any one positive sign which could be regarded as pathognomonic of hysteria:

A young man was sent to University College Hospital because his illness had proved obscure and had not yielded to treatment in a provincial hospital. The history of his illness did not strongly suggest any known organic affection of the nervous system, but there was just that in it which made it possible that disseminated sclerosis might be found to account for the symptoms. I accordingly examined him in detail, having clearly in mind the possibility that this organic disease might be present, and, beginning with the cranial nerves, I reviewed the whole of his nervous system, with negative results, until the knee-jerks were tested and found to be exaggerated. This did not, however, prove that there was organic disease, as you will recognize from what has already been said on the subject. I next examined him for ankle clonus, but the result was negative. I was, nevertheless, still prepared to find evidence of organic disease, for when his lower limbs were uncovered my attention was attracted by the position of his great toes, notably by the left one, which was drawn back towards the dorsum of the foot in a manner that suggested the possibility that the extensor type of plantar reflex might be present. This did not, however, prove to be the case, for on testing them both plantar reflexes were found to be flexor in type, and not even on the left side was there any suspicion of the abnormal extensor type of response. The complete absence of any sign of organic disease of the nervous system made the diagnosis of hysteria certain, although there were none of what I may perhaps be permitted to call the positive signs of hysteria. On making the patient get out of bed to attempt to walk, the awkward character of his station and locomotion was such as one has never seen in any case of organic disease of the nervous system, and was so bizarre as to baffle accurate description. Moreover, on making him attempt to stand with his feet together and his eyes closed, the way in which he swayed and recovered himself, first in one direction and then in another, without opening his eyes or taking hold of any of the objects around him, made the diagnosis still more certain, although it cannot be said that it was ever in doubt after the physical examination in bed had been completed.

In this patient, as in the woman with the general choreiform movements, the assurance that the faradic current would cure his malady enabled his treatment to be conducted with complete success in a very short time, so that he also left the hospital able not only to walk, but to run.

In conclusion, let me ask you to accept two apologies, one for the general imperfections of the lecture, of which I am painfully conscious, and the other for my failure to present the subject to you without appearing to be egotistic, a state of things that is particularly distasteful to me, but which circumstances have made it difficult to avoid.

A Clinical Lecture

ON

SOME OF THE COMMONER SYMPTOMS OF CEREBELLAR ABSCESS.

DELIVERED AT UNIVERSITY COLLEGE HOSPITAL,

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In the last few weeks I have had under my care in the hospital two cases of cerebellar abscess which illustrated some of the clinical aspects of that disease well enough, perhaps, to justify me in saying something about them now. I propose first to tell you, avoiding as well as I can tedious detail, about the more instructive of the two cases, then rapidly to remind you of some of the symptoms of the disease, and finally to give you a short summary of the second case.

CASE I.

A. W., male, aged 25, was admitted July 25th, 1907. He had had a discharge from the left ear for three months. A month before admission a polypus had been removed from the ear. For the last week he had had severe earache and headache; the discharge continued.

The left meatus was filled by a large polypoid mass; there was tenderness over the mastoid but no swelling. There was a profuse discharge of offensive pus from the ear, no facial paresis, and the optic discs were normal. No abnormal nervous physical signs. During the day the temperature rose to 104° F.

The case was manifestly one of chronic middle-ear disease complicated by acute mastoidal suppuration. There was no evidence to suggest any intracranial complication. The same evening the radical mastoid operation was done in the usual way. The tympanum, the antrum, and numerous mastoid cells were found to be full of granulation tissue bathed in foul pus. The operation afforded practically no relief. The temperature continued to range between 100° and 103° F.; the patient felt ill and weak, and from time to time suffered much from headache. On the second and third days after the operation he vomited, but not again. The pulse from the first had been rather slow in relation to the temperature, varying between 65 and 95 per minute.

It was obvious, therefore, that some infective process was still going on. There was, however, nothing to indicate, with a degree of precision great enough to justify action, where the source of the symptoms lay. Moreover, it is not uncommon for quite severe symptoms to persist for some days after the mastoid operation has been done for acute disease, and then to subside spontaneously. There was no course open to us, therefore, but to wait, and to watch with the greatest care for the development of any sign which would localize the seat of the lesion.

The condition above described persisted unchanged for nine days, and then an ominous development took place. The temperature, which had scarcely been below 100° since the operation, fell abruptly from 104° to normal, with no accompanying improvement in the headache or the feeling of illness. Still no nervous physical signs were present. After a slight irregularity for a couple of days the temperature settled down to the normal line, and remained steadily there. This disappearance of the fever was in no sense an encouraging change, for the headache was still severe at times, the patient still felt very weak and ill, complained of giddiness if he sat up, and was definitely though very slightly drowsy.

The third stage of the slow unfolding of the symptoms began on the sixth day after the temperature had reached the normal—that is, on the fifteenth day after the mastoid operation. The first evidence of the change was that early in the morning the patient vomited. About 10 a.m. he was drowsy, and complained of occipital headache; the drowsiness did not seem more marked than on the day before. A note was made that the eye movements were good. Two hours later conjugate movements of the eyes to the left were noticed to be a little impaired, and were accompanied by slight nystagmus.

At 6 p.m. lateral movements of the eyes showed distinct nystagmus, and conjugate movement to the left was distinctly weak.

The optic discs were normal. The right knee-jerk and the